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

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Residual Disturbances in the Higher Functions of the C.N.S. Induced by Oxygen at High Pressure.

Young albino rats were subjected to high O_2 pressure for 16 exposures and then trained on the Lashley Maze III, a control group being treated comparably but without high O_2 pressure exposure. No significant difference in learning ability was demonstrated, although the retention of the maze was adversely affected in a marked manner by exposures to increased O_2 pressure. This result is interpreted to mean that the higher C.N.S. functions of memory are inhibited by intermittent exposures to increased O_2 pressure, which has a cumulative effect.

T. G. ANDREWS (Psychol. Abstr.).

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Convulsive States and Coma in Cases of Islet Cell Adenoma of the Pancreas.

This report is an analysis of neurological and psychiatric manifestations observed in 27 patients suffering from verified adenoma of the islet cell apparatus of the pancreas. In addition relevant laboratory data, mainly those related to the glucose metabolism and in the last 11 patients also the EEG findings are discussed.

The clinical data were arranged in four groups: (1) autonomic-visceral, (2) somatic-neurological, (3) " psychomotor " manifestations, and (4) seizures and seizure fragments.

These four groups of data are then discussed in terms of anatomic-physiological levels using concepts established by Himwich. The authors' findings differ in several important aspects from those found in therapeutic insulin shock. An attempt is made to understand the reasons for this divergence.

Glucose tolerance curves in a majority of the cases are diabetic in pattern and often also in level. " Flat " curves are the exception in the authors' experience.

The EEG findings are those to be expected in patients whose cerebral metabolism is depressed. In addition suggestive " convulsive " patterns such as spike-and-wave bursts are found in most of their patients, even those who had no clinical convulsive seizures. (Authors' abstr.)

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Psychopharmacologic Study of Schizophrenia and Depressions.

Patients with schizophrenia are characterized by reacting to the intravenous administration of subnarcotic doses of sodium amytal and amphetamine sulfate with (*a*) psychologic responses which, in the present study, were clinically measurable in approximately 50 per cent. of the group, and (*b*) the rapid development of tolerance in terms of type and duration of the psychologic responses. In contrast, patients with pathologic depressions are characterized by reacting to the same drugs with (*a*) psychologic responses which, in the present study, were clinically measurable in approximately 90 per cent. of the group, and (*b*) absence of rapid development of tolerance in terms of type and duration of the psychologic responses.

(Authors' abstr.)

Action of Acetylcholine on Motor Cortex.

The electrical discharges of the coretx produced by acetylcholine are similar to seizure discharges. These discharges can be correlated with the motor components of seizures. Variations of cortical stimulability, neuronal transmission of discharges, sensory precipitation and sensory inhibition have been correlated with acetylcholine discharges and epileptic discharges. Because of these observations and the normal presence of acetylcholine in the cortex, the view is taken that acetylcholine plays an essential role in the physiologic genesis of epilepsy.

(Author's abstr.)

Histopathologic Effect of Anoxia on the Central Nervous System.

Twenty-five dogs were exposed daily to atmospheres of low oxygen concentration at the pressure of sea-level and 10 monkeys were similarly exposed in a decompression chamber. The oxygen content of the arterial blood was measured in the dogs. Histologic studies were made on the central nervous systems of all the animals and on the adrenal glands of the dogs.

The degree and duration of anoxia were important.

It was found that a single, sudden exposure to a simulated altitude of 32,000 feet (10,000 meters) for 25 minutes was capable of producing extensive laminar necrosis in the cortex of the monkey.

With repeated exposures to mild hypoxia, it was observed that the first histologic changes occurred in the cell bodies of the cortical gray matter. This took place at a level of about 12 or 13 volumes per cent. of oxygen in the blood if the exposures were long enough and were repeated often enough.

When the percentage of oxygen was reduced still lower, to about 10 volumes per cent., and the number of exposures was increased, the white matter also became involved and presented a pattern of demyelination in the corpus callosum, the

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centrum semiovale and the adjacent fingers of subcortical white matter which, in the cases of more severe anoxia, suggested a resemblance to Schilder's disease.

Aside from the lesion of the white matter, frank necrosis was usually found to occur only after episodes of anoxia sufficiently severe to produce cessation of respiration.

The frontal lobe was most often involved and the temporal lobe least often. The cerebellum was more frequently affected than the basal ganglia, and the spinal cord and medulla were unaffected by any degree of anoxia compatible with life. An oxygen level of 4 or 4·5 volumes per cent. was about as low as a dog could tolerate. Respirations quickly ceased below that level.

The adrenal glands showed increased cortical activity. (Author's abstr.)

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Philadelphia Neurological Society.

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Histologic Changes in the Brain in Cases of Fatal Injury to the Head.

As a result of craniocerebral trauma, certain changes may result in the structure of the cortical nerve cells of the cerebrum and cerebellum. These changes may be general, as a result of shock of the original traumatizing force (chromatolysis), or consequent to the generalized edema (vacuolation) which so often follows such injuries in civilian life. More noteworthy, because of their variety and degree, are those alterations which are the direct result of focal injuries of the cortex. Among the various local cellular reactions may be named preservation necrosis (retention of normal structure by dead cells); pigmentary infiltration (which is found in the vicinity of traumatic cortical hemorrhages); pyknotic, ischemic or sclerotic change and lipoidal degeneration (all resulting from local circulatory disturbances); a modified edematous change (incident to local edema following contusion or laceration); acute traumatic change (which may occur in the form of acute traumatic chromatolysis); acute total disintegration (ghost cells) or acute progressive

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necrosis; multinucleation of nerve cells (evidence, presumably, of an abortive attempt at regeneration); and, finally, ferrugination (wrongly designated by some as "calcification"), a process by which a dead cell may remain among the living as a mummified relic for many months or years.

The occurrence of definite and widespread chromatolytic changes in the nerve cells, which may persist for some time before reversible change takes place, furnishes a possible basis for the persistent psychic residual disturbances which so often follow craniocerebral injury. Local changes in nerve cells observed at the margin of regional traumatic injuries of the cerebral cortex not only furnish an explanation of the deficit pictures which result from loss of functioning areas, but probably are responsible for residual convulsive seizures. The possible physical counterparts of abnormal discharge of electric current responsible for such seizures could not be determined in this study, concerned as it was largely with cases of craniocerebral injury with relatively short periods of survival. (Authors' abstr.)

Disturbances in Sleep Mechanism: A Clinicopathologic Study. III. Lesions at the Diencephalic Level (Hypothalamus).

Of 17 cases of pathologic sleep, there was direct involvement of the hypothalamus in 14 cases. In three cases there was compression of the hypothalamus with pathologic changes in its nerve cells. In 16 cases the caudal part of the lateral hypothalamic area showed changes bilaterally. There were, however, only 7 cases with pure hypothalamic involvement. In the remaining 10 cases there was also invasion, compression or edema of either the thalamus or the basal ganglia or of both, with implication of the thalamohypothalamic and striohypothalamic pathways.

Increased intracranial pressure was present in 13 cases and absent in 4. A high incidence of increased intracranial pressure in this series should be expected, as the neoplasm encroached on the ventricular system.

Ocular manifestations, in the form of diplopia, ptosis, defective conjugate deviation, paralysis or paresis of ocular muscles, or nystagmus, were present in 14 cases. These symptoms were undoubtedly the result of compression or edema of the nuclei of the ocular nerve or their pathways.

Endocrine disturbances were present in four cases. In all these cases the tumor was situated in the pituitary region, or there was compression of the hypophysial duct or gland.

Deviations in temperature were present in the form of hypothermia in five cases and of hyperthermia in four cases.

From this series of cases of diencephalic lesions, it may be concluded that damage to the hypothalamus, especially the posterior part of the lateral hypothalamic area and of its various pathways bilaterally, causes somnolence. The influence of the thalamus or the basal ganglia on this disturbance cannot be completely ruled out. (Authors' abstr.)

Disturbances in Sleep Mechanism: A Clinicopathologic Study. IV. Lesions at the Mesencephalo-metencephalic Level.

In eight cases of pathologic sleep there was involvement of the mesencephalon and metencephalon. The hypothalamus was normal in all cases. In all the cases there was implication of the descending hypothalamic pathways and the nerve cells of the vegetative nervous system of the brain stem.

Increased intracranial pressure was present in all cases. The high incidence of increased intracranial pressure in this group should be expected, as in all cases the neoplasm encroached on the fourth ventricle or the aqueduct of Sylvius.

Ocular manifestations, consisting of diplopia, paresis of ocular muscles and nystagmus, were present in all instances; in two cases only nystagmus was shown. This phenomenon should be expected because of the location of the nuclei of the oculomotor nerves in this part of the central nervous system. The lack of sleep disturbances in other cases with ocular manifestations would seem to indicate that the various components of the ocular mechanism are not an indispensable part of the sleep mechanism.

Slight hypothermia was present in two cases and hyperthermia in one case.

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From this series of cases it can be assumed that lesions of the ascending and descending hypothalamic pathways of the mesencephalon (mammillary peduncle and mamillotegmental tract) and some of the vegetative nervous centers in the brain stem result in disturbances of the sleep mechanism. (Authors' abstr.)

MARCH.

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Importance of Neural Fibroblasts in the Regeneration of Nerve.

1. The perineurium and endoneurium of peripheral nerve are formed of specialized connective-tissue cells of two types. One is a large flat cell of mesothelial type, seen typically in the perineurial lamellas; the other is a specialized fibroblast.

2. The Schwann cells play only a subsidiary part in the regeneration of nerve. The neural fibroblast is immediately activated by injury and then proliferates. It invariably accompanies regenerating nerve fibers, often preceding them, and ensheaths newly formed nerve fibers and bundles. The large flat mesothelial cells provide an outer perineurium.

3. The uncontrolled migration of these mesoblastic cells is responsible for the traumatic neuroma. Dispersal can be prevented by the provision of an intact perineural sheath.

4. The fibrosis of suture lines and grafts is associated with previous activity of the neural fibroblast. Factors of importance in the production of collagen by these cells when once activated are ischaemia, tension and their ageing without provision of nerve fibers.

5. The perineurium can be utilized for efficient repair of defects in nerve.

(Author's abstr.)

Vasoparalysis and Vasothrombosis of the Brain in Infancy and in Early Childhood.

In three illustrative cases of a rapidly fatal disease in early childhood, attended by symptoms of acute encephalitis, necropsy revealed circulatory disturbances characteristic of vasoparalysis and vasothrombosis.

In all three cases there were no signs of inflammation. The most striking observations were different phases of vascular alteration.

The earliest manifestations of the pathologic process consisted in focal areas of perivascular transudation of serous fluid and concomitant liquefaction of the adjacent nerve parenchyma. Since no lesions could be seen in the walls of the vessels, the pathologic process was interpreted as due to increased permeability of the vessel for serous fluid.

In Case 2 a more advanced stage of vasoparalysis was displayed with increased permeability of the vessel wall for red blood cells, resulting in perivascular hemorrhages in the white matter.

The changes in Case 3 were characterized by thrombotic occlusion of the smaller veins, and were interpreted as late sequelae of a prolonged state of vasoparalysis.

The pathologic changes in the three cases appear to represent three phases of

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the same morbid process. The difference in their morphologic features can probably best be explained by the difference in the duration and severity of the circulatory disturbance.

Regional peculiarities of vascularization appear to be responsible for the greater vulnerability of the white substance and the relative preservation of the cortex.

(Author's abstr.)

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Narcolepsy. II. Theory of Pathogenesis of the Narcolepsy-Cataplexy Syndrome.

On the basis of analysis of a case of narcolepsy in a combat soldier, a theory of the pathogenesis of narcolepsy, which is essentially an elaboration of the formulation of Wilson, is advanced.

1. The brain of the narcoleptic patient is regarded as having an abnormal susceptibility to inhibition, and it is held that this is the fundamental pathophysiologic cause of the disorder.

2. The nature of the process of inhibition in the central nervous system is unknown, and the cause of the abnormal susceptibility of the narcoleptic patient to this process is equally obscure.

3. It is held that Pavlov's law of the limit of the intensity of stimulation is applicable to the narcoleptic patient. This law states that when a single stimulus or the summated effect of repeated stimuli becomes too strong for the capacity of cortical cells the ultraparadoxic phase of cortical activity supervenes, in which excitatory stimuli become inhibitory.

4. In conformity with this law it is held that the phenomenon of cataplexy results from a single excessive ultra-maximal stimulus, the magnitude of which is such that it produces the ultraparadoxic phase and consequent sudden internal inhibition in motor cortical cells which were destined for excitation. This sudden inhibition in these cells causes loss of tone and falling to the ground. In cataplexy it is held that there is no spread of internal inhibition to cortical fields involving consciousness.

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5. The case of the combat soldier described here is used to illustrate the fact that it is not the effective quality but, rather, the magnitude of so-called emotional stimuli which provokes the phenomenon.

6. The trancelike cataleptic states seen in narcoleptic patients are held to be due to the summation of repeated stimuli provoking the ultraparadoxic phase, with partial spread of internal inhibition to the motor cortical area, producing a partial alteration of movement and plastic tone, or catatonia, without spread to the cortical fields involving consciousness in the usual case.

7. The phenomenon of sleep paralysis sometimes seen in narcoleptic attacks is held to be similar to that of cataplexy, but the patient does not fall because he is in the sleeping position at the time. It is held that in sleep paralysis there is partial spread of generalized inhibition, together with sudden isolated inhibition in motor cortical cells.

8. The narcoleptic attack itself is held to be due to the effect of the summation of repeated stimuli, which produce the ultraparadoxic phase, and consequent massive internal inhibition, which spreads widely over the cortex and subcortical centers as well, leading to both the motor and the psychic phenomena of sleep.

9. The failure of autopsy material to shed light on this disorder is explained on the basis that abnormal susceptibility to inhibition is probably a chemical disturbance without demonstrable structural change. (Author's abstr.)

Disturbance in Sleep Mechanism. A Clinicopathologic Study: V. Anatomic and Neurophysiologic Considerations.

From the analysis of the clinicopathologic material presented, the cases reported in the literature and animal experiments, it is possible to reconstruct the centers and pathways concerned with the sleep mechanism.

The cases of cortical lesions indicate that fibers for the control of sleep may originate in the cerebral cortex, especially the hippocampal, cingular, frontal, premotor and temporal convolutions. To a certain extent, therefore, the hypothalamus is under cortical control. These impulses are mediated by voluntary and involuntary pathways. The main afferent and efferent pathways connecting the hypothalamus and the cortex are: (1) the medial forebrain bundle, which runs between the ventromedial olfactory correlation areas of the cortex and the preoptic and hypothalamic areas, and (2) the corticohypothalamic pathways, which are essentially the fornix and the inferior thalamic peduncle. The connection furnished by the latter fibers between the cortex and the hypothalamus is best illustrated by the cases of corticodiencephalic lesions. Other, less well established, corticohypothalamic pathways are the frontotuberal tract and the neocorticoseptal tract. There are experimental suggestions that in normal sleep the cortex becomes deafferented. Sleep, therefore, is impossible without the cortex. Forced wakefulness and diurnal sleep are cortical functions.

The evidence in the clinicopathologic cases of the diencephalic group and the results of other anatomophysiologic investigations indicate that the hypothalamus is the main center regulating sleep. The hypothalamus is in intimate connection with the thalamus, the striopallidum and the hypophysis, and its main afferent and efferent pathways are as follows:

1. Thalamohypothalamic and hypothalamothalamic pathways: They consist essentially of fibers from the medial and midline thalamic nuclei to the hypothalamic nuclei.

2. Thalamomammillary fibers: These pathways set up relays of somatic, visceral and sensory impulses from the neopallidum to the hypothalamus. The impulses from the hypothalamus to the thalamus are mediated via the mammillothalamic tract.

3. Mammillotegmental tract: This tract consists of fibers from the mammillary bodies terminating in the tegmentum.

4. Stria terminalis: This structure, which also contains preoptic and hypothalamic components, conveys fibers from the amygdaloid nucleus to the hypothalamus.

5. Supraoptic commissure.

6. Striopallidohypothalamic and subthalamohypothalamic pathways: The existence of such connections, reported by many observers, has not been fully accepted.

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7. Hypothalamohypophysial pathways: These fibers run from the supraoptic, paraventricular and tuber nuclei to the neurohypophysis.

8. Interhypothalamic pathways: These fibers connect the various hypothalamic nuclei.

Lesions interrupting these pathways may lead to sleep disturbances. Some of our clinical material and the results of animal experimentation indicate that bilateral damage to the posterior part of the lateral hypothalamic area produces somnolence. When the waking center, the hypothalamus, is disturbed, somnolence ensues. The secondary involvement of the thalamic nuclei, striatum and pallidum in many of the clinico-pathologic cases and the edema of these structures in the experimental animal suggest that these areas may also be concerned with regulation of sleep. Their influence, however, is mostly the result of involvement of the pathways which are in intimate association with the hypothalamus.

There is some evidence, largely clinical, that somnolence or other disturbances in the sleep mechanism may result from lesions at the mesencephalometencephalic level. These lesions were usually in the region of the periaqueductal gray matter. The known hypothalamic and mesencephalometencephalic connections are via (1) the mammillary peduncle, probably an ascending system of mesencephalic origin ending mostly in the lateral mammillary nucleus, and (2) mammillotegmental tract, an efferent pathway arising most likely from the dorsal part of the medial mammillary nucleus and terminating in the dorsal tegmental nucleus of the midbrain.

The opinion that somnolence and lethargy are related to lesions in the nuclei of the ocular nerves cannot be accepted, for these phenomena were essentially observed in the cases of the mesencephalometencephalic group and in some of the cases of the diencephalic group. The absence of such dysfunction in the other groups and the lack of sleep disturbances in other cases with ocular manifestations would seem to indicate that the various components of the ocular mechanism are not an indispensable part of the sleep mechanism.

Psychologic consideration of psychogenic disturbances and of some organic disorders with psychoneurotic symptoms indicates that the pathways and centers aforementioned, especially the hypothalamus, are important in the regulation of sleep. In most of the cases of psychogenic disorders sleep is a retreat in order to avoid the unpleasant features of reality, the powerful and perverted instinctual drives. In some cases of somnolence the retreat is resorted to in order to obtain gratification—a wish fulfilment of the distorted instinctual drives. The repressed drives lead to emotional tensions, which, in turn, result in dysfunction of the vegetative nervous system, of which sleep forms a part. (Authors' abstr.)

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The Development of the Human Lateral Geniculate Body.

(1) The human lateral geniculate body (pars dorsalis) differentiates in the lateral thalamic nucleus, while the pars ventralis is subthalamic in origin.

(2) The pars dorsalis is recognizable in the embryo of 22 mm., and arises as the optic tract reaches this region of the thalamus. It is the first nuclear mass to be distinguished in the thalamic portion of the diencephalon. The pars ventralis is first seen at 35 mm.

(3) No lamination is evident until the 6th month of gestation.

(4) From the outset there are six U- and V-shaped laminae which present their closed convex aspects ventro-laterally and receive the fibres of the optic tract. From the dorso-medially directed concavities issue the optic radiations.

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(5) The four larger outermost laminae consist of small cells, while the two smaller innermost laminae contain large cells.

(6) There is no such thing as " eversion " of the laminae in the human or monkey lateral geniculate body as originally described and figured by Clark and his associates. The laminae are curved to a lesser degree in the lemurs and to a greater degree in man and the monkeys, but all are curved in the same direction, the convexity always receiving the optic tract. (Author's abstr.)

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The Interrelation of the Strio-Pallidum and the Thalamus in the Macaque Monkey.

Experimental lesions of the strio-pallidum were made in nine monkeys and the resulting degeneration was studied with the Marchi technique. Area 4 was found to project into the putamen, area 6 into the pallidum. The caudate nucleus and the putamen send their fibres mainly into the pallidum. The internal division of the rostral part of the pallidum forms the ansa lenticularis, which sends fibres into the ventral anterior nucleus and the ventro-medial nucleus of the hypothalamus. Some fibres of the internal divisions pass directly into the ventral anterior nucleus. External and internal division together form the fasciculus lenticularis and pallido-subthalamic fibres. The fasciculus lenticularis distributes numerous pallidal efferents into the lateral thalamic nucleus via Forel's field and the fasciculus thalamicus. Some fibres from Forel's field terminate in the mammillary body and hypothalamus while others end in the region between mammillary body and subthalamic nucleus as far as the level of the interpeduncular nucleus. The fibre tracts described above indicate a close interrelationship between the strio-pallidum, the thalamus and hypothalamus. (Author's abstr.)

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Discrimination Between Normal and Psychotic Subjects by Revised Examination M.

Comparison of scores on the Revised Examination M (an intelligence test used by the Canadian armed forces) made by psychotics and by a normal army group reveals the existence of a psychotic profile. With statistical weighting of subtest scores, normal males average about 700 ± 220 points, and mentally ill males average less than 300 points. F. W. FINGER (Psychol. Abstr.)

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Iron Encrustation of Nerve Cells in the Vicinity of Old Traumatic Lesions of the Cerebral Cortex.

1. A change in nerve cells in which these elements become transformed into a deeply staining, bluish-purple mass has been recognized for almost a century, being first seen by Virchow at the margin of an old brain wound.

2. Because of the deep color assumed by these cells in the presence of hematoxylin, it was assumed for many years that the material with which they were encrusted was of the nature of calcareous salts. It has since been determined that these granules are composed instead of some ferrous salt.

3. This change is found in a great variety of conditions aside from traumatic lesions—in old encephalitic lesions, ischemic foci, areas of arteriosclerotic softening, at the margins of cerebral tumors, in juvenile paresis, and in degenerative lesions of the cerebrum and cerebellum.

4. This particular type of cell change may be divided into two phases: (1) a pre-encrustation stage which is characterized by its shrunken irregular contour and double coloration of its cytoplasm, and (2) the encrustation stage in which granules of some ferrous salt are first deposited on the exterior of the cell and then within its substance. This process continues if uninterrupted by death until the cell is a more or less solid mass of iron.

5. This alteration seems to occur at the margin of the scar in areas where the regional circulation has been partially preserved—sufficiently so as to permit survival of the interstitial elements but insufficient to maintain the life of the nerve cells. Thus the areas in which these elements are found have a " fibrous " appearance, which stands out, differing from the anisomorphic scar which surrounds them.

6. Ferrugination seems to be a continuation of pyknotic change beyond the point where reversal is possible with functional restoration of the cell. The cell goes on to cell death but is preserved by some means in its (pre-encrustation) shrunken state until iron salts are deposited in its structure (encrustation stage), by which process its identity may be preserved for many years.

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7. The iron is apparently derived from some soluble ferrous salt in the blood plasma. It is precipitated on and within the dead nerve cell in some form which is demonstrable with hematoxylin stain. At first these granules are discrete, but ultimately become confluent to transform the cell into a minute pyramid of iron which remains for months and years at the margin of the scar.

(Authors' abstr.)

Intracranial Complications of Infections of the Nasal Air Passages and Accessory Sinuses.

1. This study is concerned with the intracranial complications of diseases of the nasal air passages and accessory nasal sinuses as found in a consecutive series of 30,000 autopsies performed at the Los Angeles County Hospital. This survey is therefore an extension of a study made several years ago of similar findings in the first 15,000 autopsies.

2. There were 160 cases of intracranial complications associated with infectious or malignant disease of the nasal cavities, but in 42 cases there was an associated otitis media, and in these cases it was often very difficult or impossible to know just which of the two infectious foci was responsible for the intracranial lesion.

3. The extracranial, cranial, and intracranial complications of suppurative processes within the frontal sinuses present the most kaleidoscopic group of lesions. Aside from a localized osteitis, or a spreading osteomyelitis as cranial lesions, there may be found pachymeningitis externa or extradural abscess, pachymeningitis interna or subdural abscess, thrombosis of the superior longitudinal sinus; and as cerebral lesions, foci of red softening, small cortical or subcortical or large central abscesses, the latter being found most commonly in the frontal lobes, but when secondary to subdural abscess occurring in any portion of the affected hemisphere.

4. Ethmoiditis as an isolated lesion is rarely responsible for an intracranial infective lesion, but in some acute and fulminating cases septic meningitis may follow incident to focal erosion of the cribriform plate or extension along the spaces about the filaments of the olfactory nerve. Rarely, thrombosis of the cavernous sinus develops. The occurrence of frontal lobe abscess under such circumstances has been postulated, but is as yet an unproved assumption.

5. Sphenoiditis is likewise rarely primarily responsible for intracranial lesions. But when this is the case septic meningitis is also the most frequent complication, although thrombosis of the cavernous sinus may also occur. Regional (perihypophyseal) subdural abscess may also occur, but abscess of the brain is of questionable occurrence as a direct complication.

6. Infections in the maxillary sinus do not often produce intracranial complications, but when these do occur, they are found most often in dentigerous osteomyelitis of this bone in childhood. At times, however, osteomyelitis arising from a primary sinusitis may occur, and spread via the greater wing of the sphenoid to the tip of the middle fossa to produce a local subdural abscess, local meningitis, and abscess of the tip of the temporal lobe.

7. In a goodly number of this group of cases, an association of rhinal and otitic infections is found in the presence of some intracranial infectious complication. It is then difficult or impossible to determine which lesion is responsible. The association of otitis media and sphenoiditis in the presence of septic meningitis has become a well-known pathologic complex in otorhinologic circles.

8. Next to infectious lesions, neoplasms constitute the second most interesting group of nasal lesions responsible for secondary intracranial ones. These complications may be the result of direct invasion of meninges, nerve roots, or brain, or may by erosion provide an atrium for infection.

9. As a pathologic curiosity, there sometimes is found a granulomatous infection which erodes the skull in the vicinity of the sinuses, and either extends its process into the cranial cage or provides an avenue for the entrance of pyogenic organisms. Some of these rare examples have been cited in this contribution.

10. It is to be expected, with the introduction of the sulfonamide drugs and penicillin and other antibiotic substances, that the next 15,000 necropsies from this institution will show a sharp drop in the incidence of these lesions. Rare though they will likely be, they are not to be forgotten, for such lesions will continue to occur in the ignorant, the prejudiced, and the predisposed.

(Authors' abstr.)

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

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Preference for Sex Symbols and their Personality Correlates.

To investigate the relationship between attitudes toward sex and personality structure, preference for male or female sex symbols was correlated with responses on a personality questionnaire. Pairs of pictures each showing one male and one female symbol were presented to 119 female undergraduates, who indicated their " aesthetic " preference in each pair. Scores were assigned to the female symbols.

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The group was divided into a low and a high half. Relationships between responses to each of the questionnaire items and both the score groups were tested by the Chi Square method.

Sixteen questionnaire items proved significant on the 5 per cent. level or better. Considering all of these differences together, it was concluded that girls preferring male symbols were more mature, i.e. accepted their role as women and accepted men as their counterpart, while girls preferring female symbols were less mature.

(Author's abstr.)

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The Human Pyramidal Tract. XIV. A Study of the Representation of the Cortico-spinal Components in the Spinal Cord.

1. Many fibers remain in the pyramidal tract area of the spinal cord following unilateral hemispherectomy which causes practically complete degeneration of descending fibers within the pyramids.

2. Relatively more fibers are left intact in the cord area in the crossed or lateral than in the direct or ventral cortico-spinal component.

3. It is believed that, on a numerical basis, the ventral cortico-spinal tract may be important.

4. Lesions in the pyramidal tract area of the spinal cord must affect fibers other than those arising from the cerebral cortex of one side.

(Authors' abstr.)

The Cerebellar Nuclear Gray in the Sparrow (Passer domesticus).

1. The cerebellar nuclei in the sparrow constitute on either side a mass of greatly convoluted gray. The degree of convolution has some relation to the

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degree of development of the inferior olive in this particular bird, and probably to the degree of activity of which this bird is capable.

2. Such relatively narrow bands of cerebellar gray consisting of various cytologic fields indicate probably the presence of specific functional areas.

(Author's abstr.)

The Ponto-cerebellar Projection in the Rabbit and Cat. Experimental Investigations.

By means of a modified Gudden method (Brodal, 1939, 1940) the retrograde changes in the pontine nuclei (griseum pontis) following circumscribed lesions of the cerebellum are studied in cats and rabbits.

After a survey of the normal anatomy of the pontine nuclei in the species employed in this investigation the principal features of the cellular changes are analyzed. The findings in the individual experiments are presented diagrammatically.

The existing discrepancies between some of the earlier authors and our own results on certain points are analyzed and discussed. The more important conclusions reached on the basis of the present study are the following:

1. All parts of the cerebellar cortex, probably excepting only the flocculo-nodular lobe, receive pontine fibers.

2. The vermis receives a good many ponto-cerebellar fibers, although the relative amount of fibers to the vermis is by far not so abundant as to the ansoparamedian lobule.

3. A considerable mass of the ponto-cerebellar fibers pass to the paraflocculus, especially in the rabbit. The paraflocculus has a comparatively well circumscribed area of projection in the pontine gray.

4. The ponto-cerebellar fibers connect the nuclei of the griseum pontis partly with the homolateral half of the cerebellum, partly with the contralateral half. The homolateral connections are proportionately more prominent in the projection on the vermis.

5. The major cerebellar subdivisions receive the bulk of their fibers from distinct, but rather diffusely delimited areas of the pontine nuclei.

6. The dorsal and rostral parts of the nucleus reticularis tegmenti have no cerebellopetal connections.

7. The concept of neo- and paleocerebellum (Edinger) is discussed in the light of the demonstrated pontine connections with the vermis and the paraflocculus. It appears most practical to reserve the term neocerebellum for those parts of the cerebellum which are characterized not only positively by receiving "pontine fibers predominantly" (Larsell), but also negatively by the absence of spino-cerebellar and vestibulo-cerebellar fibers as well. The neocerebellum thus will comprise Ingvar's lobus medius, the lateral parts of the anterior lobe and the paraflocculus.

8. The significance of the presence of pontine fibers also to paleocerebellar division is discussed. No doubt their presence may give hints as regards the understanding of cerebellar function, demonstrating as it does that also the older parts of the cerebellum may be influenced from the cerebral cortex.

(Authors' abstr.)

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https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

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https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

Effects of Anoxia on Performance at Several Simulated Altitudes.

Critical flicker frequency and perimetry can be used to detect changes in performance of small groups of Ss under anoxic conditions (10,000 ft. and above). Body sway can be employed to demonstrate differences in performance at 14,000 ft. and above. The magnitudes of most of these changes are such that individual scores are within the normal distribution of values at sea level, and thus cannot be used as criteria, in a clinical sense, of the extent of anoxia in the individual.

Body sway increased markedly in Ss about to collapse, and suggests a possible use of the test as an indicator of acute anoxia. Perimetry and critical flicker frequency were not significantly altered in these Ss at this time as compared with the performance of other Ss under comparable anoxic conditions. Apparently the visual functions do not reflect factors determining imminent collapse of an S due to anoxia, but rather, appear to follow the blood oxygen tensions.

The lack of correlation among performance decrements in the tests suggests considerable variation in the underlying physiological adjustments to anoxia. Hence, it would seem desirable to use groups of Ss and several measures of performance requiring various levels of activity when attempting to relate personnel efficiency to anoxic stress. These procedures are indicated because of the intra-individual variability and the lack of high inter-correlation of performance decrements. (Authors' abstr.)

An Empirical Test of a Derived Measure of Changes in Skin Resistance.

The usual measure of the galvanic skin response (GSR) is in terms of a change in skin resistance, measured in ohms. The change of resistance, however, is directly related to the level of skin resistance just before the change. If the GSR scores (in ohms resistance) are grouped according to the general level of skin resistance at the time the measure is taken, it is found that both the means and the sigmas of these distributions increase as the general level of skin resistance increases. With this type of relation, it is impossible to make statements concerning the relative size of one GSR score as compared with another GSR score falling at another resistance level.

From the direct measurement of the change in ohms resistance a measure of the GSR may be derived which is independent of the general level of skin resistance. This derived measure is—

$$\frac{\log \text{GSR} + \text{k}}{\text{level of skin resistance}} \times 100.$$

If these derived measures are grouped according to the general level of skin resistance, both the means and the sigmas of the distributions are independent of the general level of resistance. Furthermore, the sigmas are independent of the means. This derived measure gives a double distribution of scores: one of these distributions approximates the normal distribution of scores, while the other seems to be a distribution of zero scores (i.e. small random fluctuations).

In practical application of this refinement, a conversion table is constructed which is used to convert the ohms resistance scores into the derived measure. The table is entered with the general level of skin resistance and the ohmic change in skin resistance, and the derived (or corrected) GSR measure is obtained directly. (Authors' abstr.)

An Attempt to Correlate the Occipital Alpha Frequency of the Electroencephalogram with Performance on a Mental Ability Test.

1. In 1,100 adult subjects no significant correlation was found between the occipital alpha frequency of the EEG and the score on a group test of mental ability (the R.C.A.F. Classification Test).

2. It was suggested that, if the positive correlations found by other investigators for eight-year-old normal children and mental deficients are representative of a true relationship, the developmental change in alpha rhythm may be associated with a change in mental age only to a critical mental age level, presumably between eight and twelve years. (Author's abstr.)

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

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Jung's Psychological Types; their Frequency of Occurrence. *Gray, H., and Wheelwright, J. B.* 3
*A Quantitative Approach to the Study of Responses of Psychotics in the Completion of Figures Involving Visual and Motor Responses. *Yacorzynski, G. K., and Neymann, C. A.* 19
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A Quantitative Approach to the Study of Responses of Psychotics in the Completion of Figures Involving Visual and Motor Responses.

1. A quantitative analysis of the responses made by 40 controls, 30 manic-depressives, and 30 schizophrenics in completing eight figures, involving visual and motor components, was performed in order to determine the perceptual principles which govern the atypical responses of the psychotics.

2. All of the differences which the manic-depressives displayed from the controls and the schizophrenics in completing some of the figures can be accounted for by an increase in motor activity. The schizophrenics completed some of the figures in such fashion as to indicate a decrease in motor activity.

3. The methods used by the schizophrenics to complete some of the figures also showed that the perceptual process was affected. The principle underlying this process appears to be due to the "patterning of stimuli," i.e. the relationship and position of the responses to each other. These atypical patterns, although differing from the patterns used by the controls and the manic-depressives, are used with a great deal of consistency by each schizophrenic patient.

4. It is pointed out that the use of tests which require motor manipulation to study perception can be interpreted only if the effect of the motor component in the test situation is known, and that should such motor elements be eliminated from the test situation, the possibility exists that manic-depressives would show characteristic differences in perception. (Authors' abstr.)

Respiration and Blood Pressure in Sensory Motor Conflict.

The following inferences are tentative, subject to verification on a larger group of subjects, and should be considered as hypothetical trends:

1. The relations between basal time and error scores, and their respective increments under conflict, may involve such personality factors as frustrationality, impulsiveness and prudence.

2. The relation between time and error scores in this experiment may prove to be an indicator of orientive-emotional balance. For example, if a mounting error score fails greatly to affect the time score under conflict, the subject may be considered as well-adjusted to the task, i.e., efficiently reorienting his visuo-motor mechanisms without emotional disruption.

3. The Woodworth I-fraction at rest and the error score with direct vision may be indicators of frustrationality and emotionality, i.e., susceptibility to frustration and potential emotional activity for the conditions of this experiment.

4. The circulatory and respiratory indices each reflect a respective category of psychological change: the former predominantly emotive, and the latter orientive. In the case of respiration, the amplitude and I-fraction seem to be disparate.

5. Some subjects apparently exhibit *less* emotional activity directly after conflict than during the pre-conflict rest period.

Empirical evidence is presented with reference to the continuous curvilinear relationship presumed, by Jenkins and the author, to exist between emotion and orientation. It is suggested that further study of a theoretical and normative nature be directed toward the elucidation of the limen of orientive breakdown. The concept of "orientive breakdown limen" is proposed as an index of frustrationality and emotionality. (Author's abstr.)

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

J. NERV. MENT. DIS.

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Studies on Neuromuscular Dysfunction.

(1) Neostigmine has been administered to 11 patients who had developed hemiplegia 1 to 31 years before. Definite improvement was observed in all of these cases, and in a few of them the improvement was marked. Improvement was retained after therapy was discontinued.

(2) Eight patients who had suffered from hemiplegia for a period of six weeks to five months were treated with neostigmine. Acceleration of recovery of motor function was observed in a number of these cases.

(3) Six patients with the spastic type of cerebral palsy were given neostigmine. Three children all showed significant improvement, while of three adults, aged 23 to 33 years, only one showed definite slight improvement from this therapy.

(4) Three patients with the athetoid type of cerebral palsy (adults aged 20 to 31) all showed significant improvement from neostigmine therapy.

(5) Two children with mild ataxia, one of whom was a case of cerebral palsy with severe stuttering and the other of whom had had a subarachnoid hemorrhage at the age of 1½ years, improved remarkably on neostigmine.

(6) Definite improvement from neostigmine occurred in ataxia and facial paralysis in a patient who had had an acoustic neuroma removed more than three years before. Moderate improvement was observed in a disability of the upper extremity in a patient who had had a prolapsed cervical disc removed four months previously. One patient with pseudohypertrophic muscular dystrophy derived no benefit from neostigmine.

(7) Three patients with lesions of the spinal cord were treated with neostigmine with slight or no improvement.

(8) Neostigmine has been found to decrease spasticity, thereby decreasing resistance to passive motion, increasing range of joint motion, relieving muscular pain and decreasing deformity. This drug facilitates voluntary motion, resulting in restoration of motions which had long been absent, increasing strength, improving co-ordination and diminishing fatigue. In addition, neostigmine has resulted in definite improvement in athetosis, ataxia, dysphagia, dysarthria, facial paralysis, motor aphasia (one case), and in conscious proprioception from a variety of types of lesions of the brain. Improvement has been retained after therapy was discontinued.

(9) For maximal functional recovery, neostigmine therapy should be accompanied by exercise and muscle training.

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(10) The mechanism of the therapeutic action of neostigmine in patients with chronic brain lesions is discussed. Since the lesion itself is irreversible because of the lack of capacity of the brain for regeneration, the drug can only facilitate the taking over of lost functions by alternate remaining brain pathways. Evidence is presented that the locus of the therapeutic action of neostigmine in these cases is on the central nervous system. (Authors' abstr.)

Electronarcosis : Its Application and Therapeutic Effect in Schizophrenia.

(1) Electronarcosis, previously shown to be a clinically feasible procedure, was applied to 47 patients suffering from schizophrenia, in order to determine whether this procedure would provide a simple and effective method to treat this condition.

(2) The clinical procedure for applying electronarcosis is described in detail.

(3) The patients treated were classified according to type and duration of the illness.

(4) One month after termination of treatment there were 19 complete recoveries, 16 social recoveries and 7 failures. Of special interest were 13 patients in which the disease had a gradual onset ; of these 6 recovered and 5 were social recoveries. Those patients whose illness was of more than two years' duration contributed 4 of the 7 failures. In the entire group a check-up after six months revealed that of the 35 satisfactory results 6 had relapsed.

(5) Though over 1,400 treatments were given, no important complications were encountered. (Authors' abstr.)

Histamine Therapy in Multiple Sclerosis.

The use of histamine intravenously in the treatment of 20 cases of multiple sclerosis is reported.. Seventeen of these cases failed to show any improvement. Three cases showed only temporary improvement of less than six months and then relapsed. Certain relationships between clinical allergies and multiple sclerosis were discussed. Despite the histopathological similarities between these conditions, the clinical approach to the two conditions seems quite distinct.

(Author's abstr.)

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Aphasia, Apraxia and Agnosia.

A reclassification of aphasias, apraxias and agnosias is presented, with three levels of psychic integration above those of sensation and the muscle contraction. The first level, the syntactic, is that of the relation between spatially and temporally adjacent sense data on the sensory side and between simultaneous muscle contractions on the motor side. The second level, the semantic, is that of the relation of sense patterns to memories on the sensory side, and of the relation of memories and ideas to motor patterns on the motor side. The sensory process at this level is one of recognition, recall or labelling of sense patterns, while the motor process is one of the choice of reaction patterns as determined by the verbal or skeletal action plan previously formulated. The third level, the pragmatic, is that of the relations between memories and ideas on both the sensory and motor sides, and is the level of the comprehension of the sensory situation and of the comprehension of and development of a plan of action for the motor situation.

Further terminological simplification is proposed in a plan to limit the term " aphasia " to defects at the pragmatic level, whether verbal or non-verbal, with

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the application of the term " agnosia " to all syntactic and semantic sensory disorders, and assignment of the term " apraxia " to all syntactic and semantic motor disorders. (Author's abstr.)

APRIL.

The Brain in a Case of Human Cyclopia. *Marburg, O., and Warner, F. J.* . 319
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Thalamic Degeneration following Bilateral Premotor Frontal Lobe Atrophy of the Strumpell Type.

In the brain of an epileptic imbecile atrophy of the cerebral cortex involved the major part of both frontal lobes exclusive of the precentral gyri. Clinically there was no gross locomotor defect. The right side of the brain was examined in detail. It was found that the Betz-cell containing Area 4 and the greater part of the agranular frontal cortex (Area 6) were intact. In the thalamus severe retrograde degeneration implicated the whole of the dorso-medial nucleus, and also the medial third of the anterior part of the ventro-lateral nucleus.

Comparison with the experimental findings in monkey and ape suggests that with the expansion of the frontal lobes associated with rise in the evolutionary scale, so the projection field of the anterior ventro-lateral nucleus shifts progressively in a frontal direction. (Author's abstr.)

Rhythmic Slow Discharges in the Electroencephalogram.

1,316 cases have been scrutinized for—

(1) Rhythmic slow activity (upper limit 4/sec.).
(2) Spike-and-wave complexes of all kinds.
(3) Blocking by eye-opening, or augmentation by eye-closing of all rhythms slower than 8/sec.

The factors involved in the segregation of these groups are discussed.

Apart from one case all reactions to visual stimuli (28) occurred in " rhythmic delta " cases, of which there were 71, composed of 26 idiopathic epileptics, 20 tumours and one abscess, 17 head injuries, and 7 unclassified.

In all groups there was clear correlation with youth.

Wide variation in spike-and-wave activity makes it impossible to draw any line between it and simple delta rhythms. Some " spike-and-wave " appears to be random and some more or less focal, but it is commonly bilaterally synchronous and usually frontal. In two epileptics a delta rhythm which responded to visual stimuli was definitely occipital.

In " organic " cases the delta rhythms are either unilateral and correspond with the site of the lesion, or bilaterally synchronous and occipital. Among the latter there is correlation, definite in tumours and probable in the remainder, with lesions of the epithalamic region. No correlation is found with raised intracranial pressure or internal hydrocephalus.

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

Response to visual stimuli may occur in unilateral or bilateral rhythms; in the former it is almost confined to the left side.

Despite the similarity of the "organic" delta rhythms to those occurring in epilepsy, no correlation whatever is found in the tumour group between epilepsy and rhythmic delta activity; nor is this correlation apparent in the head injury group, but incomplete follow-up renders this less definite.

Correlation with a short interval between injury and recording is almost complete.

Distinction between "organic" and epileptic delta rhythms is not possible in borderline cases, but the epileptic outburst tends to be less continuous and more sharply paroxysmal, while the spike-and-wave complex is rarely associated with organic lesions. (Author's abstr.)

The Effect of Age, Head Resistance and Other Physical Factors on the Stimulus Threshold of Electrically Induced Convulsions.

1. Using a constant stimulating voltage the threshold duration of stimulus for induced convulsions was determined in 70 psychotic subjects.

2. Convulsion threshold was found not to be correlated directly with height, weight, or size of head (inter-electrode distance).

3. A positive correlation was found between threshold and head resistance, and between threshold and age, but not between age and head resistance. There is no reason to suspect that the relation between threshold and age is not linear.

4. Convulsion threshold is determined by more factors than age and head resistance, but the data permit no speculation about the nature, number, or importance of these other factors.

5. A parallel is drawn between the rise in the electrical convulsion threshold of the C.N.S. during the third and fourth decades of life and increasing stability of behaviour over the same period. (Author's abstr.)

J. NEUROPATH. EX. NEUROL.

VOL. V. JANUARY, 1946.

On Re-innervation of Paretic Muscles by the Use of their Residual Nerve Supply. *Billig, H. E., et al.* 1

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*Cortical Cerebellar Atrophy without Ataxia. *Lichtenstein, B. W., and Levinson, S. A.* 29

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*The Pyramidal Tract. *Lassek, A. N.* 72

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The Effects of Local Applications of Acetylcholine to the Acoustic Cortex.

Application of acetylcholine to the acoustic cortex of cats results in a period of depression of electrical activity with decreased response to auditory stimuli, followed by a period of spontaneous ACh discharges and a heightened response to auditory stimuli. Distant responses to auditory stimulation can be obtained. ACh discharges can be inhibited by repeated auditory stimulation. Correlations between the acetylcholine treated cortex and the cortex of epileptics are discussed. (Authors' abstr.)

Cortical Cerebellar Atrophy without Ataxia.

Parenchymatous cortical cerebellar degeneration may be a primary disorder or it may be secondary to degeneration elsewhere in the nervous system.

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

The degeneration may affect only one type of cell exclusively or predominantly, such as the granule cell or the Purkinjë cell for example, or it may affect all types of parenchymatous elements.

Parenchymatous cortical cerebellar degeneration is to be looked upon as a histopathological state and not as a disease entity, for it may be found in a variety of disorders.

Sharply circumscribed areas of primary cortical cerebellar atrophy characterized by degeneration of the parenchymatous elements and secondary gliosis probably represent the end state of a degenerative process. The pathogenesis is unknown, and many factors, particularly pressure, may play an important role in its causation.

This disorder may be asymptomatic and be discovered accidentally at necropsy.

(Authors' abstr.)

Terminal Degeneration Within the Central Nervous System as Studied by a New Silver Method.

The reported silver method differs from the Bielschowsky method in two main respects: Different concentration of silver nitrate is employed and a preliminary treatment of the sections with the ammonia-alcohol solution is introduced. This procedure dissolves the greater part of the myelin and thus provides the basis for a much more evenly stained section. Other advantages of the method are the perfect staining of the myelinated and non-myelinated fibers and the light brown colour of the nerve cells. The latter allows the more intimate study of the relation between the surrounding nerve fibers and the nerve cell protoplasm.

The Gros modification of the Bielschowsky method uses an ammonia silver nitrate solution as reducer, and therefore tends to stain the nerve cells completely black. Such a simple agent as alcohol, however, prevents the reduction when added to the ammonia-silver nitrate solution, and reduction only takes place in the final 10 per cent. formalin solution. As the result of this the otherwise spotty appearance of the section is prevented and the staining process is considerably stabilized.

For the study of the finer plexus of the corpus striatum the above-described silver stain was the only one which revealed the finest axonal arborizations, whereas all the other commonly used methods gave only a poor impregnation. When studying the fiber degeneration of fine non-medullated fibers, it is of the greatest importance to have at one's disposal a method which permits a clear distinction between degenerated and normal fibers. Some methods, such as the Cajal technique, tend to stain normal fibers in a granular fashion, making it exceedingly difficult to decide whether a fine fiber is normal or degenerated. This difficulty of discrimination, however, is avoided in the method described above.

As has been pointed out in the introduction, the term "terminal degeneration" is used to cover the degeneration of the boutonal type ending and the free ending. The bouton type of degeneration within the spinal cord is, on the whole, very conspicuous, due to the abundance of terminal rings in the normal state. Numerous examinations of the cat's spinal cord have convinced us that the most common morphological expression of the synapse in establishing contact with anterior horn cells or internuncial neurons is the ring-like end formation. This view has been arrived at not only by means of the technique reported above, but also by other variations of the silver impregnation methods.

The picture of the cerebral cortex, however, differs greatly from that of the spinal cord. Even with careful examination of the normal cerebral cortex it is difficult to demonstrate ring-like endings. Their possible existence cannot, however, be denied, and a special study devoted to this problem is at present in progress. It seems that the synapse within the cortex is mainly represented by free terminals of the pericellular plexus. However, recent experiments have proved that the absence of the terminal rings is in itself not an impediment in the search for terminal degeneration.

Experiments show that pericellular degenerations can also be detected around Betz cells after interference with intracortical connections. Because of the intricate nature of the morphological aspect of the synapse, much more work is required for an understanding of the cortical synapse. This necessity of studying the cortical synapse in particular has been stressed recently by O'Leary (1944) in his contribution to publications on the precentral motor cortex. (Author's abstr.)

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

Effect of Extirpation of Parastriate Cortex on Learned Visual Discrimination in Monkeys.

Monkeys have been trained to discriminate between pairs of visual stimuli on the basis of size, shape and color. Monkeys so trained lose the discrimination habit if areas 18 and 19 are destroyed bilaterally in one stage. The discrimination can then be relearned post-operatively at approximately the same rate as originally. If the cortical destruction is carried out in two stages with an interval between (in this case 18 days) during which testing is continued, the habit is lost after neither the first nor second operation. (Author's abstr.)

The Pyramidal Tract. The Representation of the Lateral Corticospinal Component in the Spinal Cord of the Cat.

1. Very few, if any, fibers of the crossed pyramidal tract extend throughout the entire length of the spinal cord in the cat.

2. The tract terminates largely in the upper two-thirds of the spinal cord.

3. There is evidence that many of its fibers terminate high in the cervical region of the cord.

4. Many fibers are normally present in the pyramidal area of the cord which do not belong to any part of the pyramidal system, including the so-called homolateral corticospinal component.

5. Degeneration of axons of the pyramidal tracts in the cat was detected as early as three days by the protargol method of staining.

6. Cellular activity of the supporting elements continues for nine months or more in the pyramids following massive, cortical ablation.

7. In its phylogenetic development the pyramidal tract in the cat appears to occupy a position intermediate between the lower mammals and man.

(Author's abstr.)

APRIL.

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Central Nervous System Resistance.

A survey of the literature revealed that although several investigators have reported effects of cerebral anemia, they were not able to produce a complete anemia and their results were variable. It was also noted that other types of deficiency in substances normally brought to the brain, such as hypoglycemia, vitamin B deficiency, inanition, CO poisoning, etc., often produce lesions in the brain which, in many instances, resemble those of the present investigation.

Animals were subjected to periods of complete arrest of the cerebral circulation of two, four, six, eight and ten minutes.

Susceptibility differences are marked in different areas of the brain, and a certain degree of local selectivity or specificity of lesions is evident.

There appears to be a gradient of resistance, which from lowest to highest degree of resistance is as follows: Cerebral cortex (*isocortex*), cerebellar cortex (Purkinjĕ cells), sensory and integrative centers of brain stem, pons and medulla oblongata, cerebral cortex (*allocortex*), and finally, large motor cells of brain stem, pons, medulla oblongata, and spinal cord.

Some possible significant bases of the cell and blood-vessel changes are discussed. It is felt that, since the lesions appear in many cases restricted to anatomic units (nuclei), these groups may be not only morphological, but also metabolic units.

It is pointed out that the extreme sensitivity of the brain to ischemia and anemia is of great clinical importance, and that lesions observed clinically are often similar to those reported. It is, however, important to consider the technique used, as a

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

tool for the study of electrical and metabolic changes of the brain under conditions of increasing severity of anemia and blood stasis. Such studies, it is hoped, will lead not only to information of clinical value, but also to data clarifying the basic relationships between abnormalities of structure and function. (Author's abstr.)

Cerebral Cortex in Very Old Human Brain.

The overwhelming majority of very old brains showed at least some degree of cortical atrophy, diffused or regional in distribution. The degree of cortical atrophy was not necessarily proportionate to the age of the patient. The cyto-architecture was surprisingly well preserved in the very old brains, the three types of pyramidization, granularization and spindlization being as obvious as in juvenile and adult ones. Cell changes were consistently present in the very old brains. The cell changes, however, were not proportionate to the age of the patient. Neither the degree of cortical atrophy nor the extent of cell destruction was proportionate to the duration of the clinical history.

There was no constant vulnerability of one of the three cell types, although in some instances the pyramidal cells seemed more vulnerable than the granular and the spindle cells.

Satellitosis was not a constant feature in the very old brain. There was no parallelism between the degree of cell destruction and satellitosis. The latter showed a definite predilection for the anterior parts of the brain. All the anatomical features described occurred regardless of the type of the clinical picture.

(Author's abstr.)

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*Rapid Changes in Cerebral Oxygen Tension Induced by Altering the Oxygenation and Circulation of the Blood. *Roseman, E., et al.* . . . 33
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*Recovery of Motor Function after Two-stage Extirpation of Area 4 in Monkeys (*Macaca mulatta*). *Ades, H. W., and Raab, D. H.* . . . 55

Reflex Pupillodilator Mechanisms.

Parasympathetic denervation of the eye results in marked enlargement of the pupil, but not in its maximal dilatation. Maximal pupillary dilatation probably requires active contraction of the radial muscle of the iris, which is innervated through sympathetic nerves.

Peripheral pain producing stimulation elicits moderate reflex dilatation of the pupil. This reaction is not altered by sympathetic denervation of the eye, but is abolished by section of the oculomotor nerve or extirpation of the ciliary ganglion; consequently, it is mediated through the parasympathetic nerves.

Strong stimulation of the hind foot or the exposed sciatic nerve, following transection of the spinal cord in the lower cervical region, resulted in slight enlargement of the pupil in some animals under nembutal anesthesia, but not in unanesthetized animals. This response in the anesthetized animals apparently indicates increased excitability of the ciliospinal center due to the anesthetic agent.

Following dilatation of the pupil due to atropine depression of the cholinergic innervation of the iris, moderate stimulation of the oculomotor nerve elicits further pupillary dilatation. The reflex pupillodilator response elicited by peripheral stimulation is greatly reduced following incomplete depression of the adrenergic nerves by the intravenous administration of ergotoxine. These results support the assumption that the pupillodilator reaction mediated through the mesencephalic parasympathetic center is actively integrated and controlled. The inhibition of the circular muscle of the iris is brought about, not by inhibition of the parasympathetic center, but by activation of this center which results in the discharge of efferent nerve impulses through adrenergic fibers arising in the ciliary ganglion.

(Authors' abstr.)

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

Effects of Inhibitors of Choline Esterase on the Nerve Action Potential.

The effect of choline esterase inhibitors on the nerve action potential has been studied. The preparations used were the giant axon and the fin nerve of the squid.

1. Eserine alters and, in sufficiently high concentration, abolishes within 15 minutes the action potential of both the giant axon and the fin nerve. This is consistent with the concept that ACh is the depolarizing agent released during the passage of the impulse. In that case the physiological role of choline esterase should be the rapid removal of the ester, and consequently, inhibition of the enzyme should result in enduring depolarization and therefore abolition of conductivity.

2. The effect described is easily reversible. This had to be postulated if it is the inhibition of the enzyme which is responsible, since the inhibition of choline esterase by eserine is easily reversible *in vitro*.

3. In contrast to eserine, prostigmine has no effect on the nerve action potential, although both compounds have the same powerful inhibitory effect on choline esterase *in vitro*. Eserine is a tertiary amine which, in its undissociated form, can pass through the lipoid membrane. Prostigmine is a quarternary ammonium compound and cannot penetrate a lipoid membrane. This has been shown experimentally.

4. Strychnine, another chemical with a high affinity for choline esterase, also alters and then abolishes the nerve action potential reversibly.

5. Cocaine and procaine first depress the action potential and then, as is well known clinically, abolish conductivity reversibly. They act in concentrations so low as to make it improbable that the effect is due to inhibition of choline esterase.

6. The observations explain why ACh, a quaternary ammonium compound, does not act if applied to the axon externally but does at the nerve ending; there the absence of a myelin sheath makes penetration possible. They are consistent with the concept that the physico-chemical mechanism of conduction of the impulse along the axon does not differ fundamentally from its transmission across synapses.

(Authors' abstr.)

The Mechanism of the Electrophonic Effect.

1. Monaural beats may arise when an electrical and a mechanical stimulus are applied to the same ear.

2. The psychological attributes ascribed to the sensations produced by the interaction of two stimuli with varying degrees of difference in their frequencies are essentially similar whether two mechanical stimuli or a mechanical and an electrical stimulus is employed.

3. When a mechanical and an electrical stimulus of identical frequency are applied to the ear it is possible to cancel the one by adjusting the phase and intensity of the other.

These three facts are considered evidence that the two types of stimuli, mechanical and electrical, activate the same cochlear element and that the activating force is mechanical in both instances.

4. When two distinct tones differing in frequency by a few cycles are simultaneously presented to the same ear, and the phase of one of them abruptly reversed, an interruption in the continuity of both stimuli is observed.

This is taken as evidence that the basilar membrane is critically damped.

(Author's abstr.)

Rapid Changes in Cerebral Oxygen Tension Induced by Altering the Oxygenation and Circulation of the Blood.

With a platinum electrode, salt bridge and suitably adjusted voltage it is possible to record the oxygen tension of tissue *in vivo* as a function of the amperage. By this method studies were carried out on the cortices of cats anaesthetized with dial and immobilized with Beta erythroidine. A decrease in cortical oxygen tension invariably followed erythroidinization; this was believed to be due to a decrease in cerebral blood flow secondary to a decrease in systemic blood pressure. When adrenalin or ephedrine were administered, an increase in cerebral oxygen tension occurred; this was believed to be due to an increase in cerebral blood flow secondary to a rise in systemic blood pressure. Amyl nitrite caused a fall

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

in oxygen tension, probably secondary to the fall in systemic blood pressure. Ergotamine tartrate and nicotinic acid had insignificant effects. Inhalation of nitrogen produced a precipitous fall in cortical oxygen tension to a low level, which was considered asphyxial. With the readmission of air, the cerebral oxygen tension rose rapidly to a level above that observed before asphyxiation. Breathing O_2 raised the cerebral oxygen tension. (Authors' abstr.).

Carbonic Anhydrase in the Nervous System.

Thiophene-2-sulfonamide in concentrations of 27 mgm. per cent. or higher has no effect on several properties and functions of the axone, the spinal cord or the cortex. Since the carbonic anhydrase of the nervous system is inhibited more than 99·99 per cent. by these concentrations of the drug, it is concluded that the catalyzed hydration and dehydration of carbon dioxide are not essential reactions in the nervous system. (Author's abstr.).

The Effect of Cellular Hydration on Experimental Electroshock Convulsions.

1. Cellular hydration produced in rats by depletion of 40 per cent. of extracellular electrolyte without change in total body water lowers the electroshock seizure threshold by an average of 56 per cent.

2. Cellular hydration produced to an equal degree by orally administered water lowers the electroshock seizure threshold to an equal extent.

3. Combination of these two methods of cellular hydration produces a synergistic reduction in threshold for electroshock convulsions, and spontaneous seizures may occur.

4. The threshold for metrazol convulsions is reduced in hydrated rats to the same extent as the threshold for electroshock seizures.

5. Rapid electrolyte replacement increases the electroshock threshold to normal and above. An equivalent amount of electrolyte given to normal rats raises the normal seizure threshold.

6. An increase in extracellular fluid volume without alteration of cell volume or electrolyte concentration does not lower the convulsive threshold.

7. It is concluded that cellular hydration decreases and cellular dehydration increases the seizure level independently of changes in volume of extracellular fluid.

8. Not all anti-epileptic drugs can be shown to raise the normal electroshock convulsive threshold. This is particularly true of diphenylhydantoin. In contrast, diphenylhydantoin as well as phenobarbital and tridione significantly elevate the electroshock threshold lowered by cellular hydration.

9. A method for the laboratory assay of anti-epileptic drugs is suggested which is superior to that employing the normal electroshock seizure threshold.

(Authors' abstr.).

Recovery of Motor Function after Two-stage Extirpation of Area 4 in Monkeys (Macaca mulatta).

A phenomenon of bilateral compensation of the effects of unilateral lesions of area 4 is reported, such that if area 4 of the remaining hemisphere is removed after a suitable delay (3 to 4 months), none of the usual signs of pyramidal injury appear. Such compensation occurs as well in the absence of callosal connections, but is prevented if areas 3, 1 and 2 are also removed. A possible participation of the premotor cortex in the process of compensation is also discussed.

(Authors' abstr.).

MARCH.

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*Synaptic Potentials of Motoneurones. *Eccles, J. C.* 87
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*Pyruvic Acid Exchange of the Brain. *Himwich, W. A., and H. E.* . . . 133

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Effect on the Electroencephalogram of Localized Pressure on the Brain.

1. A new method for the experimental production of seizure patterns has been introduced.

2. Slowly increasing localized cortical pressure produced the following abnormalities in the EEG pattern: (i) seizure patterns; (ii) associated seizure patterns consisting of fast and slow waves, as well as spikes; (iii) delta or slow waves, below 5 cycles per second; (iv) combination wave patterns consisting of fast waves superimposed on delta waves; (v) flat waves wherein the voltage was reduced to below 20 microvolts.

3. The seizure, and associated seizure patterns, showed involvement of all three electrodes in 47·5 per cent.

4. The seizure, and associated seizure patterns, were definitely localized to the pressure electrode in 42·5 per cent.

5. Ninety-five per cent. of the seizure, and associated seizure patterns, involved the pressure electrode.

6. During a paroxysm single or multiple patterns were observed.

7. The incidence of seizure patterns occurred more frequently during compression than during release.

8. The delta waves, flat waves and combination waves were primarily confined to the pressure electrode.

9. Delta voltage increased during compression until approximately the fourth turn; then the voltage decreased. During release the delta voltage slowly increased.

10. The incidence of flat waves increased in direct relation to the degree of compression.

11. The incidence of combination waves slowly increased until the sixth turn of compression, then decreased. Combination waves were absent during the release stage. (Authors' abstr.)

Eye Movements following Strychninization of the Superior Colliculus of Cats.

1. The path of reflex attraction of the eyes toward a light in the peripheral visual field is activated in anesthetized cats by strychninizing a small area of the superior colliculus and flashing a light into either eye.

2. Each point on the superior colliculus is found to regulate movement of the eyes to a particular part of the visual field, medial points causing upward lateral movements, lateral points causing downward lateral movements, each colliculus always causing movements to the contralateral visual field.

3. The superior colliculus regulates reflex conjugate eye movements by causing relaxation of antagonist muscles as well as contraction of agonist muscles of the eyes.

4. The superior colliculus charted for control of localized eye movement compares well with the superior colliculus charted for the projection of the visual field on the colliculus. (Author's abstr.)

Synaptic Potentials of Motoneurones.

Motoneurones of the cat's or frog's spinal cord have been subjected to direct synaptic excitation by a single synchronous volley of impulses, and the resulting potential changes have been recorded from the ventral root fibres as they emerge from the spinal cord.

When synaptic transmission of impulses is blocked by a sufficiently deep anaesthesia, a negative potential change is still set up in the motoneurones and propagated electrotonically along the ventral root fibres. This catelectrotonic potential (called the synaptic potential) is shown to be homologous with the endplate potential of striated muscle and the synaptic potential of sympathetic ganglia. The mean durations of latent period, the rising phase, and the half-time of the exponential decay are respectively 0·7, 2·5 and 5·2 msec. (cat), and 1·5, 5·0 and 29 msec. (frog). The synaptic potential is decrementally transmitted along the ventral root, halving approximately in every 4·5 mm. (cat) and becoming slower in time course.

There is summation of synaptic potentials set up by two volleys, or by repetitive stimulation. Even at the highest frequency (200 per second—frog, 400 per second —cat) cessation of stimulation has been followed by a prompt delay, there being no sign of any enduring depolarizing action, such as occurs in sympathetic ganglia.

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In the unanaesthetized preparation the synaptic potential begins at least 0·2 to 0·3 msec. before the discharge of impulses by the motoneurones.

When synaptic transmission of single volleys is just blocked by anaesthesia, a discharge is still produced by a second volley at a short interval—up to 15 msec. in the cat. The size and latent period of this facilitated response show the variation with stimulus interval that would be expected if facilitation were due to summation of the synaptic potentials. Owing, apparently, to an accommodation-like process, the threshold is set by the slope of the summed potential, as well as its absolute value.

Similarly, synaptic transmission to a single nerve volley occurs as if the synaptic potential were the causal factor in setting up the discharge of impulses from motoneurones.

There is no evidence of any synaptic excitatory mechanism other than the synaptic potential. The sole factors governing synaptic transmission would appear to be the synaptic potential (acting simply as a catelectrotonus) and the stability of the motoneurone's surface membrane. The anaesthetic nembutal, blocks transmission largely by increasing the stability of the membrane, but also partly by diminishing the production of synaptic potential. It has no appreciable action on the time course of the synaptic potential

Analysis of the synaptic potential by means of the local potential theory gives a short duration for the synaptic transmitter action, especially when allowance is made for electrotonic distortion of the synaptic potentials during their intramedullary transmission. The average values for total duration are 2 msec. (cat) and 4 msec. (frog). No appreciable lengthening of this duration is observed after rapid repetitive stimulation.

Curarine has a strychnine-like action on the spinal cord.

Eserine, even in large doses, has no effect on single or repetitive synaptic potentials. This finding is considered in relationship with the evidence so far adduced that acetylcholine is the synaptic transmitter, and it is concluded that acetylcholine probably plays no significant role in synaptic transmission in the spinal cord. The evidence presented in this paper accords with the hypothesis that synaptic transmission in the spinal cord is due to the action currents of the presynaptic impulses. (Author's abstr.)

The Pyramidal Tract. Effect of Maximal Injury on Acid Phosphatase Content in Neurons of Cats.

1. This study demonstrates that the acid phosphatase technic is a delicate method for determining the integrity, and course of subsequent degeneration, of the axis-cylinders following maximal injury to the cells of origin.

2. The enzyme disappears from the axons of the pyramidal tract of the cat between the second and third day following cortical removal. The largest axis-cylinders are more sensitive, and thus disappear faster than the medium- or small-sized fibers.

3. Once disappeared the acid phosphatase does not reform or reappear throughout the prolonged process of secondary degeneration. This has been followed for as long as one year post-operative.

4. The occurrence of the enzyme within the axon is discussed in relation to certain other recent studies on nerve cells, as well as indicating the possible significance of the axis-cylinder in the maintenance of the myelin sheath.

(Authors' abstr.)

A Cortico-bulbo-reticular Pathway from Area 4-s.

By strychninizing the cortex and recording the electrical activity of the brain stem of the monkey, a pathway has been found to arise from area 4-s and diverge from the cortico-spinal tract in the pons to reach the bulbar reticular formation. From its known physiological properties this cortico-bulbo-reticular pathway is an extrapyramidal system mediating relaxation. (Authors' abstr.)

Pyruvic Acid Exchange of the Brain.

A study of the pyruvic acid exchange of the brain of 41 patients shows that small, but significant amounts of pyruvic acid, averaging 0·22 mgm. per cent. are

https://doi.org/10.1192/bjp.92.388.605 Published online by Cambridge University Press

added to the cerebral venous blood in the quietly resting human subject in the post-absorptive state. These data support the conclusion that the brain constantly produces some energy without the equivalent utilization of oxygen. The carbohydrate balance of the brain not only includes that portion which is oxidized, but also the part split to lactic and pyruvic acids. (Authors' abstr.)

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MARCH.

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Histologic Studies of the Brain following Head Trauma. II. Post-traumatic Petechial and Massive Intracerebral Hemorrhage.

1. Three cases of post-traumatic petechial and massive intracerebral hemorrhages are described in detail clinically and histologically.

2. Brief notes are presented of five other cases demonstrating the presence, in addition to superficial cortical lacerations, of petechial and massive hemorrhages within the deeper structures of the brain.

3. The view is expressed that the deeper forms of hemorrhage result, in part at least, from vasoparalytic phenomena caused by transmission of force from the site of impact to the deeper structures of the brain.

4. The changes described in this paper represent a more severe stage of brain damage than cerebral swelling and edema, described in the first paper of this series. (Authors' abstr.)

Water Content of the Brain after Concussion and its Non-contributory Relation to the Histopathology of Concussion.

Experiments were performed in guinea-pigs to determine whether previously described structural changes and loss of nerve cells in the brain after concussion might be secondary to pressure occasioned by an increase in water content of the brain. An accurate method for quantitative determination of water in brain tissue was developed. Eight and 17 hours after concussion slight though significant increases in the water content of the brains were observed. The maximum increase amounted to only 19 mgm. It was also shown that concussion was not followed by an increase in the rate of formation or the pressure of the cerebrospinal fluid.

In other experiments a highly significant increase in water content of the brain was brought about by administering excessive quantities of water by stomach

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tube. A group of animals so treated was permitted to live for a period of time equal to that after which the greatest post-concussional cytological changes were encountered. In controlled histological studies it was demonstrated that no alterations had been produced. It may be concluded that brain edema after simple concussion in the guinea-pig is slight, and does not produce the degenerative changes in neurons that are characteristic of post-concussion.

(Authors' abstr.)

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DECEMBER.

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*Studies in Insulin and Metrazol Therapy. *Schnack, G. F., Shakow, D., and Lively, M. L.* 125

The Intelligence-Vocabulary Ratio as a Measure of Temperament.

1. The present investigation provides experimental evidence that differential performance on a conceptual and on a vocabulary test of intelligence is related to temperament.

2. The scores of 1,821 male and of 987 female neurotic patients at Mill Hill Emergency Hospital on the Progressive Matrices and on a vocabulary test of the synonym-selection type were used. Difference scores were calculated for each subject, and the case records of patients with a high intelligence-vocabulary ratio compared with those with a low intelligence-vocabulary ratio.

3. Full information was available on 250 men and 200 women with a high intelligence-vocabulary ratio and on 290 men and 140 women with a low intelligence-vocabulary ratio. The former groups were found to contain significantly more patients with a hysterical make-up and symptomatology, and the latter significantly more patients of an anxious or depressed make-up, suffering from anxiety states and/or depression.

4. The groups differed also with respect to age, education, employment and earnings. The low intelligence-vocabulary ratio groups were on the whole younger and of a poorer educational level.

5. The data were reanalyzed by separately equating the groups for age and for education. Certain differences between the older and younger groups and between the elementary-school groups and the secondary-school groups emerged, but these applied equally to the high and to the low intelligence-vocabulary ratio groups.

6. The separation of patients into the hysterical type on the one hand and the anxious and/or depressed on the other was therefore not due to differences in age or educational background, but was based on their differential performance on two cognitive tests.

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7. If the score obtained on a vocabulary test be interpreted as a measure of the application of innate intellectual ability, the anxious and depressed patients utilize their intellectual ability significantly more than the hysterics. Since introversion has been held to be correlated with anxiety states and depression and extraversion with hysteria, the investigation points to a possible correlation between introversion and a low intelligence-vocabulary ratio and extraversion and a high intelligence-vocabulary ratio. (Author's abstr.)

Studies in Insulin and Metrazol Therapy. I. The Differential Prognostic Value of Some Psychological Functions.

A battery of psychological tests was given to 70 male schizophrenic patients two weeks before insulin or metrazol shock therapy was instituted. Test scores on the 1916 Stanford-Binet, the Kent-Rosanoff Word Association Test, and a test of aspiration were compared with the relative clinical condition of the patient at various times following treatment until April, 1942, which constituted a period of one to five years after the first examination. In a search for measures which might be prognostic of long-run clinical improvement, a set of 13 signs was found useful in predicting improvement by insulin therapy. The best results were obtained when the individual subject satisfied four or more of these signs. Another set of 11 signs was found useful in predicting improvement by metrazol therapy, the same general degree of dependability being achieved when the individual satisfied six or more of these signs. The most significant index for predicting improvement from insulin treatment was the number of insulin signs satisfied which exceeded the number of metrazol signs satisfied.

Although the insulin and metrazol signs made use of the same measures, the ranges of scores did not overlap. In each case the signs for improvement with insulin therapy are the higher, more nearly normal, whereas the metrazol signs tend in the opposite direction. It would seem that metrazol therapy is most valuable for persons who, at the time of initiation of treatment, appear from the psychological tests to be the more profoundly affected by the psychosis.
(Authors' abstr.)

Studies in Insulin and Metrazol Therapy. II. Differential Effects of Some Psychological Functions.

A battery consisting of the Stanford-Binet, the Kent-Rosanoff Word Association Test, and an aspiration level test was given to 70 male schizophrenic patients before and after treatment with insulin or metrazol. The means of the test scores before and after treatment were compared to determine the effects of treatment on intellectual functions. Considerable changes in the direction of improvement were noted in most of the measures.

To test the significance of the changes comparison was made with individually matched control patients who had had neither form of therapy, but who had been given two tests while under routine hospital care. The results indicated that approximately two-thirds of the improvement in test scores may be attributed to the ordinary hospital regime and familiarity with the test situation. The data further indicated that those with mental ages below 12–0 on the first test gained more than those with higher mental ages. There appears to be a differential effect between metrazol and insulin in this respect. Metrazol seems more efficacious than insulin with the lower intelligence group, but seems deleterious with the higher group. Insulin, on the other hand, though helpful in both groups, seems more effective in the case of the higher intelligence group when the changes occurring beyond those shown by control patients under routine care are taken as the criterion.
(Authors' abstr.)

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The Pavlovian Theory of Generalization.

The neo-Pavlovian system of explanatory principles is built upon two fundamental postulates: (1) that in primary conditioning all stimuli which act during excitation of an unconditioned reaction tend to be associated with that reaction; (2) that effects of training with one stimulus irradiate to produce association with similar stimuli, with a strength of association proportional to the degree of similarity. Explanations of stimulus equivalence, of generalization, of "afferent neural interaction," and of perceptual organization or "patterning" are based upon these two postulates. Both postulates are contrary to fact.

(Authors' abstr.)

Emotion in Man and Animal.

The main conclusions of this paper can be summarized as follows:

(1) The recognition of a full, characteristic expression of emotion is the classification of a deviation of behavior from an habitual base line. It is not a discrimination of the momentary behavior itself, but of the direction of the deviation, so that both present and past behavior affect the observer's judgment.

(2) The recognition of emotion otherwise is a discrimination of a state of changed responsiveness detected from "associated signs"; acts which would not have a definite emotional significance in themselves, but which have been observed as the accompaniment of more openly emotional behavior.

(3) The emotions thus detected are inferred special states which facilitate or actually produce the primary emotional behavior of (1), although little is known of these states or of a satisfactory classification for them.

(4) The recognition of emotion in man and animal is not fundamentally different. Conceptually, the states of changed responsiveness discriminated are the same in both cases and have the same relationship to overt behavior.

(5) Even when the subject himself does the recognizing, the ultimate criteria of the various emotions are found in distinctions of overt behavior. In subjective recognition cues are available which a second observer cannot utilize, but these cues (of imagery and so on) must be essentially of the nature of associated signs which the growing child learns gradually to interpret after first learning the meaning of emotional terms in relation to actual overt behavior.

(6) Finally, it may be concluded that the failure to obtain a reliable recognition of emotions in the laboratory experiments of the last thirty years was the result of a particular experimental procedure (the use of too short a period of observation), and does not show that emotion cannot be recognized socially. Also, the conclusion of some writers that emotions are nothing but figments of the imagination stems directly from the apparent unreliability of recognition; if recognition is reliable socially and does not depend mainly on knowledge of the stimulus, this conclusion is unjustified.

A strong argument in favor of the hypothesis presented is that it removes the contradictions inherent in current discussions of emotion and emotional recognition. A stronger argument perhaps is found in the original datum of this paper: the fact that psychologically sophisticated and unsophisticated alike experience an overwhelming tendency to name the chimpanzee's emotions, with a human terminology. Yet the details of emotional expression are quite different in the two species. Recognition of emotion from the chimpanzee's facial expression is even worse than with man's, and most of the *incidental* signs of chimpanzee excitement have a significance that must be learned by prolonged observation. Yerkes makes it clear that close familiarity with the chimpanzee is necessary for the discrimination of many emotional states, and that the individual components of emotional behavior do not have the same significance in man and chimpanzee. It is only in the higher-order units that the identity of behavior in the two species becomes evident. Consequently the tendency of any observer to recognize and name chimpanzee emotions is not due to a mere superficial similarity. From my own observations I should say that the recognition of identity really begins only after several weeks of observation, and that the strength of one's conviction that the identity is real, increases thereafter for months.

Such a fact might be evidence only of suggestibility, but this cannot be the whole explanation. The behavior of the dog is complex enough, but life-long familiarity with this animal does not produce the degree of "anthropomorphism"

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in psychologically sophisticated persons that six months of exposure to the chimpanzee will produce. The tendency to identify human emotional patterns in the chimpanzee is the more convincing since, as I have shown, the identification is not indiscriminate. The naming of attitude or emotion is much less frequent than the reader might suppose, and occurs only when the behavior in its long-term significance and intercorrelations is something already nameable in man. When the elements of behavior have a relation that is not known in man, no name is given to the underlying emotion. The naming, therefore. is not an irresponsible form of animism, but a classification of deviations of behavior. The facts of behavior must constitute the ultimate reference of emotional terms. (Author's abstr.)

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*The Stress Tolerance Test. *Harrower, M. R., and Grinker, R. R.* . . 3
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The Stress Tolerance Test. Preliminary Experiments with a New Projective Technique.

It has seemed from this study that certain clearly demonstrable differences exist between the performance of patients with operational fatigue and the control subjects in the Stress Tolerance Test.

These differences can be expressed qualitatively and quantitatively, and the extent to which certain characteristic answers appear in a record seems to bear a direct relationship to the severity of the case. While no weighting system has yet been devised which will give a specific numerical point at which a patient should be considered severely, or mildly, disturbed, we feel that utilization of the criteria listed will be of considerable help in evaluating records.

These detectable differences, moreover, are understandable theoretically when viewed in the light of the patient's attempt to cope with a situation which precipitates him suddenly into reliving painful experiences. His ability to handle this experience objectively rather than retreat from it or relive it too closely indicates his progress along the return path to stability.

This test proved valuable when used as an objective measure of the degree of the patient's improvement. (Authors' abstr.)

A Study of Conditioned Vasomotor Responses in Ten Human Subjects.

1. The vasomotor system of 4 to 10 subjects was conditioned rapidly to a light stimulus. A faradic current was used as the unconditioned stimulus and the vascular reactions were measured by means of the photo-electric plethysmograph. Once obtained this conditioned response appeared to be relatively stable.

2. Conditioning and extinction curves of this type of conditioning did not always follow the typical curves of learning and forgetting. The irregularity of the curves may be attributed to:

(*a*) The instability of the unconditioned responses in some subjects.
(*b*) Changes in attitude and emotional set of the subject.
(*c*) Other factors not studied in this experiment.

3. Three subjects who manifested signs and symptoms of autonomic nervous system imbalance were more easily conditioned than subjects who did not.

4. Subjects who were relatively easily conditioned showed more rapid and complete elimination of incidental vascular reflexes to the light stimulus, and showed a tendency toward more stable conditioned responses during extinction than subjects who were not easily conditioned.

5. The intensity of sensations as reported by a subject was not a valid indication of the magnitude of the physiological vascular reactions concomitant with these sensations. (Author's abstr.)

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A Study of Personality of Normal Young Men Maintained on Restricted Intakes of Vitamins of the B Complex.

Various aspects of personality were studied in eight normal young men maintained 161 days on a partially restricted intake of B-complex vitamins, which was followed by 23 days of acute deficiency, and 10 days of thiamine supplementation.

Self-ratings and man-by-man ratings gave no evidence of change in the status of well-being and adjustment during the partial restriction, but indicate consistent and striking deterioration during the acute deficiency. Supplementation of the diet by thiamine alone produced rapid recovery. The Minnesota Multiphasic Personality Inventory also gave no evidence of change in the partial restriction. During the acute deficiency significant changes were obtained in the scores on the three psychoneurotic scales—depression, hysteria and hypochondriasis.

In the Rorschach test records made at the end of the partial restriction, slight deteriorative changes were indicated in three out of the four experimental subjects. These changes increased in magnitude in the pair of subjects placed subsequently on the acutely deficient diet. The nature of this deterioration was loss of spontaneity with an increase in tension. The Rorschach findings suggested that individuals with " better " initial personality were better able to resist the dietary stress.

Cattell's Cursive Minature Situations Test during the partial restriction indicated a very slight and statistically *not* significant increase in the number of lines erroneously crossed (" emotionality " score). During the acute deficiency there were evidences of further increased " emotionality " and " timidity." On the other hand, those indices which have distinguished psychotics from normals showed no change in our deficient subjects. The personality changes were among the earliest symptoms of the experimentally produced borderline and acute deficiencies.

(Authors' abstr.)

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1. Biochemistry, Physiology, Pathology, &c.

Effect of Insulin on Metabolism of Muscles and Brain. Merezhinskii, M. F., and Cherkasova, L. S. [*Biochem. J.* (*Ukraine*), **17**, 341–56 (*in Russian*) (*English summary*, 356–7) (1941).]

The effect of insulin shock, in guinea-pigs (I) 60–80 units, rabbits (II) 20–30 units, and dogs (III) 4–5 units per kgm. of body weight, began to show in the second 0·5 hour in general weakness, inability to stand up, lack of response, followed by sleepiness, salivation, twitching, and convulsions in about 2 hours; subcutaneous injection of 10–15 gm. of a 20–40 per cent. glucose solution to (II) usually resulted in quick recovery and acceptance of food; refusal to eat was generally followed by a more severe recurrent shock. The Qo_2 of the total brain tissue for (I) was 5·08–7·05 before the shock and down to 2·12–4·35 at the height of the shock effect; gray matter of (II) from 5·74–6·87 to 2·71–4·05, and white matter from 3·11–4·96 to 2·07–3·60; gray of (III) 5·96–7·14 to 3·81–4·35, and white from 3·11–4·01 down to 2·51–3·71. The Qo_2 in the animals killed on the following day (after the shock) was 5·32–8·38 in the whole brain of (I); gray matter of (II) 6·38–8·71, and white 3·04–4·34. The lowering of respiration intensity of brain matter was general for the three classes of animals; at the same time there were cases where this reduction was not observed. Femoral artery, femoral vein and cerebral (sagittal sinus) vein blood in 10 dogs on an empty stomach before the shock showed for O content (volumes per cent.) a range of 16·21–25·14, 6·43–14·48, and 6·18–14·48; for glucose (mgm. per cent.) 73–132, 61–109, 81–128, respectively; 30 minutes after the shock O was 15·35–23·91, 6·56–14·21, 7·49–12·37; glucose, 65–105, 62–97, 50–94. After 60 minutes, O, 15·08–24·62, 8·64–13·98, 7·96–19·18; glucose, 48–96, 41–75 and 42–74 respectively. After 180 minutes, O, 18·02–24·68, 11·24–18·61 and 14·16–20·40; glucose, 31–51, 29–57 and 32–59. On the following day, O, 17·13–24·68, 6·17–13·01 and 6·49–11·89; glucose 96–124, 85–112 and 81–113, in the arterial venous and sinus blood respectively. Instances were observed where the tissue

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lost the ability to fix and hold its sugar, and even gave it up, as then the glucose in the arterial blood was equal to or lower than in the venous. An increase in the uptake of sugar by the muscles and partially by the brain in comparison with the O demand indicated that the sugar fixation was not necessarily accompanied by an increased consumption of O. It is also probable that under the influence of insulin some organs gave up their sugar; this caused the fluctuation in the sugar level. That the muscle tissue adsorbs and holds the sugar more intensely than the brain does was shown in those instances in which the consumption of O did not decrease despite the reduction in sugar consumption. Introduction of glucose improved the consumption of O, with a rise in the arteriovenous O difference; this was not observed before the shock. In guinea-pigs killed on the following day, the Qo_2 of the total brain tissue was 5·03–8·38; (II), gray matter, 6·38–8·71, white 3·04–4·34. The fluctuating values were probably due to variations of respiration intensity during some stages while the hormone was still in effect. A series of (I) and (II) were given repeated injections of insulin in doses sufficient to cause convulsions; the Qo_2 for the whole brain of (I) (each value for a different animal) was, after 8 injections, 6·98; after 15, 7·74; after 25, 8·01; 17, 8·59; 30, 4·39; 34, 3·35; 27, 6·65; 6, 7·95; 35, 4·38; and one after 41 injections, 3·45; the Qo_2 for (II), gray and white matter respectively was, after 6 injections, 6·95 and 4·05; after 35, 7·38 and 4·08; 58, 5·37 and 3·52; 64, 3·85 and 2·92; 59, 4·35 and 3·01; and one after 60 injections, 3·92 and 2·92. The injections to individual animals varied from 6 to 63; some survived many, while others died very quickly after only a few injections. All lost weight at once; the animals were killed 2–3 days after the last injection. B. GUTOFF (Chem. Abstr.).

Absence of Alteration in the Electroencephalogram with Stilbestrol and Progesterone. Cress, Charles H., Jr., and Greenblatt, Milton (Harvard Med. School). [*Proc. Soc. Exptl. Biol. Med.*, **60**, 139 (1945).]

Large doses of stilbestrol and progesterone given alone and together to non-pregnant, non-menstruating women had no effect on the cortical encephalogram. L. E. GILSON (Chem. Abstr.).

Effect of Glucose Injections on Fasting Ketonemia after Low-protein Diets (in Rats). Tidwell, Herbert C., and Treadwell, C. R. (Southwestern Med. Coll., Dallas, Texas). [*Proc. Soc. Exptl. Biol. Med.*, **60**, 101 (1945).]

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

Experimental Alcoholism and Alcoholic Polyneuritis in Pigeons. Lecoq, Raoul (Hôpital de Saint-Germain-en-Lage, Paris). [*Bull. soc. chim. biol.*, **24**, 320–4 (1942) (*see C.A.*, **39**, 2809[8]).]

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

Phenylpyruvic Oligophrenia: A Clinical and Biochemical Study. Lepow, Harold. [*Monatsschr. Psychiat. Neurol.*, **110**, 161–92 (1945).]

The effect of various diets and compounds on the urinary excretion of phenylpyruvic acid (I) was studied in a patient with phenylpyruvic oligophrenia. The total amount of (I) excreted in each of from 4 to 14 successive 24-hour urines was determined by the method of Penrose and Quastel (*C.A.*, **31**, 4391[8]). The (I) excretion increased on a high and decreased on a low protein diet. The average excretion increased after feeding phenylalanine (II) or (I) along with the low protein diet, but the variation from day to day was large and the extra excretion represented only a small part of the amounts given. Neither tyrosine (III) nor vitamins B_1, B_2, B_6, or C had any effect on the excretion of (I). There was a small increase in the (II) concentration of the blood after ingestion of (I) and (II), but not after (III). Lepow discusses in detail the metabolism of (II) and (III) on the basis of findings in phenylpyruvic oligophrenia, alcaptonuria and tyrosinosis.

WARREN M. SPERRY (Chem. Abstr.).

Research Work on Degenerative Disease. Kountz, Wm. B. (St. Louis City Infirmary and Washington Univ. School of Med.). [*Biol. Symposia*, **11**, 74–8 (1945).]

In 20 patients (diseases not specified), aged 50 to 90, parallelism was observed between I.Q. and motor functions (strength of grip of the hand, pedal movement

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of the legs, vital capacity), and between I.Q. and physiological functions (urea clearance, concentration diuresis, circulation time of the blood, glucose-tolerance, and cardiac activity), but not between motor functions and blood sugar, urine chemistry, electrocardiogram, basal metabolic rate, or chest radiography findings. The closest parallel with declining physical activity of the body was shown by glucose-tolerance and loss of ability of the body to concentrate and hold water. Conclusion: To demonstrate degeneration from a clinical standpoint, the functional ability of the body rather than its state at any particular time should be studied.
MARION HORN PESKIN (Chem. Abstr.).

Arteriosclerosis and Lipide Metabolism. Page, Irvine H. (Lilly Lab. for Clin. Research, Indianapolis City Hosp.). [Biol. Symposia, **11**, 43–73 (1945).]

A critical review with bibliography. Evidence is presented that: Atherosclerosis is not invariably associated with the loss of vascular elasticity accompanying ageing; the development of atherosclerosis is favored by mechanical strain (hypertension), by hyperlipemia, hypercholesterolemia, and a factor acting on the artery cells themselves which is inhibited by (I), KSCN, and possibly lipocaic and choline; hyperlipemia and hypercholesterolemia cannot be prevented by withdrawal of fats and cholesterol, respectively, from the diet (with the possible exception of cholesterol in a diet with greatly increased phosphatide content); atherosclerosis can develop in the absence of hyperlipemia; old age does not cause hyperlipemia; lipides other than cholesterol are concerned in atherosclerosis, and are deposited in the arterial wall in approximately the same proportion as they exist in the lipide mixture of plasma. Theories of Virchow and Aschoff, of Klotz and Leary, of Winternitz, and of Hueper are discussed. Possible control of factors predisposing to atherosclerosis involves (1) prevention of hyperlipemia (as by treatment of diseases causing it, e.g. diabetes mellitus), (2) reduction of mechanical strain (as by reduction of elevated arterial pressure), and (3) prevention of that state of the tissues in the arterial walls which increases the receptivity to plasma lipides or prevention of that state of the blood which leads to abnormal precipitability of its lipides (this has been accomplished in experimental animals only, e.g. by iodides and thiocyanates). MARION HORN PESKIN (Chem. Abstr.).

The Relation Between Etiology and Morphology in Degenerative and Sclerosing Vascular Diseases. Hueper, W. C. (Warner Inst. for Therapeutic Research, New York, N.Y.). [Biol. Symposia, **11**, 1–42 (1945).]

An attempt is made to establish the relationship between etiology and morphology of nonsenile arteriosclerosis by systematizing and integrating biological, pathological and experimental data from the literature and from unpublished studies. Hueper believes that the various chemical and physical causes of experimental and spontaneous arteriosclerosis act by interfering with the oxidative metabolism and nutrition of the vascular wall. The anoxemic causative mechanisms are: (*a*) Changes in vascular tonus, e.g. by histamine, acetylcholine, nitrates, nitrites, cyanides, CO, barbiturates, As, Hg, Mn, reduced atmospheric O pressure, traumatic shock (hypotonic agents), and by adrenaline, sympathomimetics, angiotonin, tyrosine, tyramine, guanidine, ergotine, hydrastine, digitalis, glucosides, nicotine, S-methylisothiourea, vitamin D, Ca salts, iodine, and adrenal cortical, posterior pituitary, thyroid, and parathyroid hormones (hypertonic agents); (*b*) changes in hydrostatic intravascular pressure, e.g. by consumption of excessive amounts of liquids; (*c*) changes in plasma colloid composition resulting from quantitative and qualitative disturbances of plasma carbohydrates, lipides and proteins, interfering with the exchange of gases and nutritive substances through the interface of blood and intima, e.g. in diabetes mellitus, excessive dietary lipide intake, and CS_2 poisoning, and after administration of goitrogenic substances (sulfaguanidine, thiourea derivatives, thiocyanates), saponin, polyvinyl alcohol, methylcellulose, pectin and arabinose; and (*d*) changes in the O-carrying power and in the O–CO_2 balance of the blood and tissues, e.g. in CO or O poisoning. Primary intimal sclerosis and hyalinosis result from (*a*) or (*b*), fibrosing intimal reactions from (*c*) (plasma protein disturbances), medial calcinosis from severe action of vasotonic and hydrostatic agents or as a late effect of (*c*), cystic medial degeneration of the aorta from severe hypotonic episodes, and atheromatous lesions from (*c*) (lipide and carbohydrate changes). MARION HORN PESKIN (Chem. Abstr.).

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Dicholesteryl Ether in the Spinal Cord of the Ox. Silberman, Henryk, and Silberman-Martyncewa, Sofia (Ermington, N.S. Wales, Sydney, Australia). [*J. Biol. Chem.*, **159**, 603–4 (1945).]

Investigation of the cholesterol prepared from spinal cord revealed, in contrast to that prepared from brain, the presence of an impurity which was identified as dicholesteryl ether. The amount of this compound isolated from the spinal cord of the ox is from 1·5 to 2·0 per cent. on the dry basis.

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

Blood-cholesterol Levels in Elderly Patients. (1) *Relation of Age, Sex, Basal Metabolic Rate, Cardiac Decompensation, and Coronary and Peripheral Sclerosis to Blood-cholesterol Levels in the Aged. Kountz, Wm. B., Sonnenberg, Arthur, Hofstatter, Lilli, and Wolff, Gerhard (St. Louis City Infirmary and Washington Univ. School of Med.).* [*Biol. Symposia*, **11**, 79–86 (1945).]

Among 212 patients aged 40 to 85, the females showed an average blood-cholesterol level of 237 mgm. per cent., the males 196 mgm. per cent. Atherosclerosis, however, appeared earlier and more frequently among the men, and in both sexes calcification of the peripheral arteries was accompanied by a lower blood-cholesterol value than was absence of such calcification. Coronary sclerosis was not associated with hypercholesterolemia in either males or females. Hypercholesterolemia accompanied lowered basal metabolic rate in women, not in men.

MARION HORN PESKIN (Chem. Abstr.).

The Effects of Anoxia and of Haemorrhage on the Metabolism of the Cerebral Cortex of the Rat. Wilhelmi, Alfred E., Russell, Jane A., Long, C. N. H., and Engel, Mildred G. (Yale Univ.). [*Am. J. Physiol.*, **144**, 683–92 (1945); *cf. preceding abstract.*]

Reproducible initial rates of respiration in the absence of substrate and steady rates of respiration for at least 3 hours in the presence of glucose can be obtained in slices of rat cerebral cortex, provided that the medium contains Ca, that the initial dry weight of the tissue is chosen as the basis for calculating the rates of O uptake, and that the time of preparation of the tissue is carefully standardized. The rate of respiration of brain slices from normal rats in the absence of glucose was depressed to an increasing degree with increasing duration of anoxia after incubation in N for 10, 15, 20 and 30 minutes. No consistent effects of shock after hemorrhage on the metabolism of brain were observed. E. D. WALTER (Chem. Abstr.).

Biological Changes Following Encephalography. Delay, Jean, and Soulairac, André (Univ. Paris). [*Compt. rend. soc. biol.*, **138**, 951–3 (1944).]

Encephalography in man is followed by hyperglucemia, acidosis, hyperleucocytosis, arterial hypertension and bradycardia. These effects are similar to those of electric shock. L. E. GILSON (Chem. Abstr.).

Protein Metabolism in the Nerve Cell During Growth and Function. Hydén, Holger. [*Acta Physiol. Scand.*, 6 *Suppl.*, **17**, 136 pp. (1943).]

During embryonic development as well as during the second period of growth of the nerve cell the total amount of cytoplasmic protein in the cell increases rapidly. The nucleolar and nuclear apparatus show signs of intense activity, as the mechanism for the formation of cytoplasmic protein, but in the adult nerve cell the apparatus exhibits a unique condition not seen in any other cell of the adult organism with the exception of oöcytes. In motor anterior horn cells, from animals which had been completely exhausted physically, the protein content of the cytoplasm and nucleus is markedly decreased and the nucleolar system shows symptoms of intense irritation. In spinal ganglion cells irritated by induction shocks new proteins are formed with surprising rapidity but, if the irritation is sufficiently prolonged, the cytoplasm will be eventually drained of these substances.

S. MORGULIS (Chem. Abstr.).

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Riboflavin and Temperature During Experimental Poliomyelitis in the Monkey. Kolochine-Erber, B., and Raffy, Anne (Inst. Pasteur, Paris). [*Compt. rend. soc. biol.*, **138**, 963–4 (1944).]

As previously shown, riboflavin plays an important role in thermogenesis. However, the hypothermia of the final stages of poliomyelitis in the monkey is not the result of a decrease in tissue riboflavin. L. E. GILSON (Chem. Abstr.).

Prevention of Formation of End-bulb Neuromas. Poth, Edgar J., Fernandez, E. Bravo, and Drager, Glenn A. (Univ. Texas Med. Branch, Galveston). [*Proc. Soc. Exptl. Biol. Med.*, **60**, 200–7 (1945).]

Opposing the growth of nerve fibers by encasing the proximal stump of a severed peripheral nerve in a rigid tube, or injection of the nerve with tannic acid or gentian violet, inhibits neuroma formation. L. E. GILSON (Chem. Abstr.).

Mechanism of the Action of Sympathomimetic Amines. (1) *The Effect of Various Amines on Synthesis of Cocarboxylase. Govier, Wm. M., Bergmann, Vera, and Beyer, Karl H. (Sharp and Dohme, Inc., Glenolden, Pa.).* [*J. Pharmacol.* **85**, 140–2 (1945).]

In 0·00005 M concentration phenethylamine, N-methylphenethylamine, N-dimethyl-β-methylphenethylamine, benzedrine, tyramine, and paredrine markedly increased the synthesis of cocarboxylase by pigeon-liver slices.

(2) *The Effect of Sympathomimetic Amines on the Succinoxidase System as Influenced by the Presence of α-tocopherol Phosphate.* [*Ibid.*, 143–9.]

The activity of the succinic oxidase of homogenized rat muscle and liver is inhibited by addition of α-tocopherol phosphate. The sympathomimetic amines, adrenaline, benzedrine, cobefrin and phenethylamine produce an increase in O uptake by neutralizing the tocopherol effect. The increase in metabolic activity following the addition of these amines to the tocopherol-inhibited succinic oxidase system can be used as a source of energy for synthesis of cocarboxylase from added thiamine and pyrophosphate. L. E. GILSON (Chem. Abstr.).

Anaerobic Glycolysis in Nervous Tissue. Utter, M. F., Wood, Harland G., and Reiner, John M. (Univ. of Minnesota, Minneapolis). [*J. Biol. Chem.*, **161**, 197–217 (1945).]

Extracts and homogenates exhibiting high rates of anaerobic glycolysis were prepared from spinal cord, medulla, cerebellum and cerebrum of rats. One part of tissue was mixed with 4 parts of ice-cold water and the mixture homogenized in a Potter and Elvehjem type grinder. No further treatment followed in the case of the homogenates. If extracts were to be prepared, the homogenates were held for 15 minutes in an ice-bath and then centrifuged for 15 minutes at 2400 r.p.m. For complete activity these preparations need, in addition to glucose, adenosine triphosphate, hexosediphosphate, diphosphopyridine nucleotide (DPN), Mg++, and nicotinamide. The authors present evidence that the " inhibitor " of brain extract reported by Geiger (*C.A.*, **34**, 5517[9]) is a diphosphopyridine nucleotidase. Inhibition of the nucleotidase can be obtained with nicotinamide or by incubation of the preparation with DPN in bicarbonate buffer. Etherization of the animals before the preparation of glycolytic extracts and homogenates did not have any detectable effect on extracts, and had slight or no effect on homogenates. The preparations have approximately linear activity with time and with increasing concentration of tissue for 60 minutes or longer ; they thus prove to be useful in assay of enzymes of anaerobic glycolysis of the central nervous system.

GERTRUDE E. PERLMANN (Chem. Abstr.).

Chemical Changes in the Brain and Gasometric Studies of the Blood in Experimental Head Injury. Stone, W. E., Webster, J. E., and Gurdjian, E. S. (Wayne Univ. Coll. Med., Detroit). [*Proc. Assoc. Research Nervous Mental Disease*, **24**, 226–53 (1945).]

Traumatic head injuries, administered to dogs under morphine and procaine analgesia, did not influence the cerebral arterio-venous differences in O, CO_2, or glucose. In areas of contusion, brain tissue, frozen *in situ*, showed greatly increased

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lactic acid (I) and inorganic phosphate and decreased phosphocreatine (II) and adenosine triphosphate concentrations. A slight loss of acid-soluble P usually occurred. In brains studied 1 to 6 days after injury there appeared to be partial recovery in (I) and (II). In areas of the cortex showing no macroscopic evidence of damage the concentrations of the constituents studied were sometimes entirely normal; occasionally the (I) concentration was slightly increased. The results indicate that generalized cerebral anoxia does not necessarily follow trauma of the head. WARREN M. SPERRY (Chem. Abstr.).

Nervous Disorders Resulting from Manganese Poisoning. Bogaert, Ludo van, and Dallemagne, Marcel J. (Univ. Bunge and Univ. Liége, Belgium). [*Monatsschr. Psychiat. Neurol.*, **111**, 60–89 (1945–46).]

A monkey in a closed cage with a circulating fan was exposed to an aerosol containing MnO_2, $NaClO_3$ and NH_4Cl, in the proportions 50 : 35 : 15 respectively for one hour daily for 95 days. The amount of MnO_2 was increased gradually from 10 to 50 mg. The first abnormal signs appeared at the 40th treatment—hyperexcitability interspersed with periods of torpor. At the 47th treatment massive trembling of the whole body occurred and continued increasingly through the rest of the experiment. Other signs, such as pronation and supination of the hands, appeared as the experiment progressed. After cessation of the treatments the symptoms gradually disappeared within the following 5 months, but then reappeared in a somewhat different and more marked form, with paresis of the legs as an important feature. No Mn was found in any of the organs, including the brain and liver, probably because the animal was killed several months after exposure to Mn. Histopathological examination showed atrophy of the cerebral cortex in accord with the clinical symptoms. Liver, kidney and lungs were normal. Another monkey received $MnSO_4$ by mouth in doses increasing from 10 to 50 mgm. per day for 300 days. The O capacity of the blood dropped gradually during the experiment and returned to normal after $MnSo_4$ administration was stopped, but no signs of nervous disorder were noted clinically or histopathologically. The findings are of interest in their bearing on Mn poisoning in man.
WARREN M. SPERRY (Chem. Abstr.).

A Search for Linkage Between the A, B, O Agglutinogens and Phenylketonuria. Penrose, L. S. (London, Ontaria, Can.). [*Am. J. Mental Deficiency*, **50**, 4–7, (1945).]

A study of 74 patients with phenylketonuria (I) and 97 normal sibs gave a strong suggestion of the presence of a genetic linkage between (I) and the blood agglutinogens A, B, O and A_1. WARREN M. SPERRY (Chem. Abstr.).

The Neuro-humoral Syndrome of Myotonia. Minz, B., and Passouant, P. (La Sorbonne, Paris). [*Rev. can. biol.*, **4**, 510–34 (1945).]

In Steinerts' disease the myotonic muscle is characterized by the myotonic reaction and by trophic troubles. The study of three muscular biopsies revealed that the myotonic muscle is rich in acetylcholine and poor in cholinesterase. The modifications of the myotonia after the action of pharmacodynamic agents acting on cholinesterase and acetylcholine were studied. With vitamin C, which intensifies cholinesterase activity, there was obtained a marked improvement, which was further strengthened by the associated effect of quinine, which decreases the sensitivity of the motor plate to the cholinergic mediator. Myotonia is considered as being of muscular origin and as responding to the richness of the muscle in acetylcholine. As the latter is produced by the nerve, the primary trouble is considered to originate in the myoneural plate. In the first stage the acetylcholine produced at the synapse would seem to be incompletely destroyed as a result of the lowering of the cholinesterase level. In a second stage acetylcholine diffuses from the terminal plate into the muscle, thus creating a condition of "static myotonia," which will give a myotonic response to mechanical excitation, even if the nerve is completely excluded, e.g. after curarization. The diffusion of acetylcholine involves a lowering of muscle K, thereby modifying the ionic medium of the muscle. There follows a decrease in muscular excitability, thereby creating conditions favorable to the onset of an associated muscular atrophy.
A. PAPINEAU-COUTURE (Chem. Abstr.).

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Micro Method for the Estimation of Phosphatidylethanolamine in Nerve Tissue. Edman, P. V., and Aqvist, S. E. G. (Karolinska Inst., Stockholm). [*Acta Physiol. Scand.*, **10**, 144–9 (1945).]

Extract with EtOH for 24 hours finely ground nerve tissue. Dilute extract so that 1 c.c. contains about 0·4 to 1·5 mgm. cephalin. Measure 1 c.c. into a centrifuge tube, add 2 c.c. 2 N HCl, and hold in hot water bath for 3 hours. Cool dry *in vacuo* at room temperature over NaOH. Extract fatty acids with 5 c.c. dry ether, centrifuge, discard ether, and dissolve residue in 0·5 c.c. H_2O. Heat the liberated ethanolamine in a special apparatus with NaOH. The freed NH_3 is collected in 0·5 c.c. 0·1 N HCl. Neutralize with 0·5 c.c. 0·1 N NaOH, add 0·5 c.c. borate buffer (7·32 gm. boric acid + 2 gm. NaOH in 1·1 gives pH 10), then 0·5 c.c. NaClO solution (prepared fresh by adding N NaOH to 0·15 N Cl water to pH 10), and dilute to 5 c.c. The tube and a similarly treated blank are placed for 5 minutes in a vigorously boiling water bath. The stable blue color is examined with filter S_{61} in a step photometer. The value must be multiplied by the factor 12·2 to convert it to cephalin. S. MORGULIS (Chem. Abstr.).

Increase in Blood Pyruvic Acid after the Convulsions of Electric-shock Therapy. Gounelle, H., Raoul, Y., Bachet, M., and Marnay, Ch. (Hôpital Foch, Paris). [*Compt. rend. soc. biol.*, **139**, 166–7 (1945).]

Blood pyruvic acid shows a marked increase within 1–2 minutes after the convulsive crisis. The maximum level observed was 6·58 mgm. per cent. A rapid return to normal then takes place. L. E. GILSON (Chem. Abstr.)

Urinary Acid-base Balance and Electric Shock Convulsions. Delmas-Maraslet, P., Servantie, L., Bannel, F., and Maurice, G. (Univ. Bordeaux). [*Compt. rend. soc. biol.*, **138**, 696–8 (1944).]

During electric shock treatment human urine becomes more acid. This is due to an increase in lactic and pyruvic acids, especially the latter, as a result of the violent muscular contractions. L. E. GILSON (Chem. Abstr.).

Studies on the Protein Concentration of the Cerebrospinal Fluid in Lumbar-disk Prolapse. Friberg, Sten. [*Acta Chir. Scand.*, **87**, 128–37 (1942).]

In lumbar intervertebral prolapses it is recommended that cerebrospinal fluid taken caudal to the prolapse should be examined. Usually it contains 60 mgm. per cent. protein and the globulin/albumin ratio is 0·25 or less.
S. MORGULIS (Chem. Abstr.).

Coagulation-active Substances in the Cerebrospinal Fluid: I. Krafka, V. [*Acta Med. Scand.*, **120**, 147–93 (1945).]

Normal cerebrospinal fluid contains coagulation-active substances, especially the ventricular fluid. Lumbar or cisternal fluid coagulates 0·1 c.c. fresh oxalated plasma at 37° in 22 minutes ± 6 minutes when 1·5 c.c. fluid is used. The coagulating activity is not related to the Ca content, but to the thrombokinase and prothrombin content of the fluid, which is free from fibrinogen. The protein content of the fluid is of no significance. S. MORGULIS (Chem. Abstr.).

Biochemical Study of Bilirubin of Blood and Spinal Fluid. Polonovski, Michel, Fiessinger, Noel, and Gajdos, Alfred. [*Bull. soc. chim. biol.*, **24**, 221–5 (1942); *cf. C.A.*, **39**, 2794[3], 5304[5].] L. E. GILSON (Chem. Abstr.).

Flocculation of Resin Suspensions by Proteins. Mechanism of the Colloidal Benzoin Resin Reaction and a New Flocculation Reaction of Cerebrospinal Fluid. Dognon, A., and Simonot, Y. [*Bull. soc. chim. biol.*, **26**, 277–81 (1944); *cf. C.A.*, **39**, 4339[5].]

The effect of pH on the flocculation of colloidal mastic or benzoin resin suspensions is further discussed, and an application of the method previously discussed to the examination of spinal fluid is described. Normal and pathological spinal fluid cause maximum precipitation of the colloidal resin at different degrees of pH.
L. E. GILSON (Chem. Abstr.).

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Determination of Total Protein in Cerebrospinal Fluid. · *Lindenmeyer, Eugen.* [*Monatsschr. Psychiat. Neurol.*, **109**, 57–73 (1944).]

The determination of total protein (I) in cerebrospinal fluid (CSF) by precipitation with Esbach's reagent and measuring the volume of precipitate after centrifugation (Kafka and Samson (*Z. Neurol. Psychiat.*, **115**, 85 (1928); **117**, 128 (1928)) was compared with the Kjeldahl method in 327 patients. In many cases a wide difference between the two methods was observed, with the Kjeldahl method usually giving higher values, sometimes several fold. Frequently the Kafka-Samson method erroneously indicated normal (I) concentration. The upper limit of normal (I) concentration in CSF is 35 mgm. per 100 c.c. W. M. SPERRY (Chem. Abstr.).

Theory of the Colloidal-gold Reaction. (1) *Reactions Between Gold Sol and Isolated Protein Fractions. Lange, Carl* (*N.Y. State Dept. Health, Albany*). [*J. Lab. Clin. Med.*, **30**, 1006–12 (1945).]

Of the physiochemical factors conditioning the colloidal-gold reaction, the most significant is the pH. This must be fixed, since hemoglobin either coagulates or protects gold sol, depending on whether the pH is below or above 7·0. The loss of sensitivity of citrate gold due to increasing disparity can be eliminated by boiling to a greater turbidity and neutralizing. Temperature is of little influence. The presence of electrolytes is obligatory in the diagnostic test, and their concentrations must be kept constant within the optimal range. At pH 7·4 three qualitatively different reactions with gold sol have been shown: (1) Protection is produced by albumins and normal serum globulins in the prezone, and by hemoglobin at a pH above its isolectric point; (2) sensitization is produced by serum albumin within an optimal range; (3) true coagulation, without the assistance of electrolytes, is produced in cerebrospinal fluid only by pseudoglobulin-like *degenerative proteins*, such as produce the paretic curve. This curve is fundamentally different from the other (prezone) curves, which, in the absence of a true coagulator, result from differences in the globulins, while the albumins have only a decreasing effect. Euglobulin and pseudoglobulin react differently with gold sol, as shown by the difference of their constant optimal ratio. This ratio is the reason why, in prezone curves, the maximum shifts to the right with increasing globulin concentration. The albumin-globulin ratio has little or no effect, since no variation of this ratio produced a paretic curve in the absence of a true coagulator. Nor does the maximum shift to the right with increasing albumin-globulin ratio. The maximum is regulated by the qualitative and quantitative differences of the globulins. Removal of albumins from cerebrospinal fluids produces no qualitative change in either the paretic or prezone curves. DOROTHY A. MEYER (Chem. Abstr.).

Effects of Small Doses of Vitamins on the Nervous System. Chauchard, Paul (*The Sorbonne*). [*Bull. soc. chim. biol.*, **24**, 182–5 (1942); *cf. C.A.*, **38**, 1770⁹.]

In normal rats and guinea-pigs small doses of any of the vitamins have some effect on chronaxia. Vitamins A, C, and D exert an excitant action, and nicotinic acid has the opposite effect. Vitamins B and E first excite, then depress. All the water-soluble vitamins act on the brain only, as regards effect on chronaxia, while the fat-soluble vitamins act on the brain, medulla and spinal cord.
L. E. GILSON (Chem. Abstr.).

Mirror Carp and Vitamin B_1. Nogueira, Cyro Camargo, and Antunes, Luiz Nora. [*O. Hospital*, **25**, 745–50 (1944).]

Chastek's paralysis is a B_1 avitaminosis due to enzymic destruction of vitamin B_1 by raw fish meat (carp). Unlike that of the ordinary carp, the meat of the mirror carp did not destroy the thiamine present in the food of the experimental animals (female albino rats), and caused no incidence of beriberi; it even acted as a source of vitamin B_1. H. ROTHSCHILD (Chem. Abstr.).

Vitamin B Deficiency and Nervous Disease. Spillane, J. W. (*York, England*). [*Lancet*, **249**, 449–50 (1945).]

Apart from relief of pain and tenderness in some acute cases, neither thiamine nor whole vitamin B complex conferred any apparent benefit in 200 cases of poly-

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neuritis (excluding those of obviously infective origin) in malnourished people in the Middle East (Polish refugees, Arab, African and Indian natives, and German and Italian prisoners-of-war), in malnourished German troops from the Channel Islands, and in patients with chronic beriberi from prisoner-of-war camps in the Far East. Rest in bed and "good food" gave as good results as the vitamins mentioned, although these were administered in large doses and by all routes. The vitamin preparations did not hasten the restoration of power, sensibility, or reflex activity, nor shorten the required stay in bed or in the hospital. Conclusion: If vitamin B_1 is the antineuritic vitamin, the results of therapy in chronic polyneuritis cannot be quoted in support of that contention, despite any properties B_1 may possess in combating the acute beriberi of the Far East. The literature on the relationship between vitamin B complex and neuromuscular disorders is critically reviewed, with bibliography. MARION HORN PESKIN (Chem. Abstr.).

Urinary Excretion of Coproporphyrin in Non-alcoholic Pellagra. Rimington, C., and Leitner, Z. A. (Univ. of London, Univ. Coll. Hosp. Med. School, and St. Mary's Hosp., London). [*Lancet*, **249**, 494–6 (1945).]

Normal urinary coproporphyrin values (below 100γ/24 hours) were found in 13 of 15 patients with non-alcoholic pellagra, whose satisfactory response to subsequent nicotinic acid therapy substantiated the diagnosis. The two pellagrous patients with coproporphyrin values above normal were suffering from sepsis and liver dysfunction respectively, which may have contributed to the high figure. The findings indicate that excess porphyrinuria is not an essential feature of pellagra, and hence is not of diagnostic value. Among 7 patients with Korsakow's syndrome (in 6 instances referable to alcoholism) only 2 showed abnormally high urinary porphyrin excretion; this indicates that alcoholism *per se* is not consistently sufficient to produce excess porphyrinuria. An alcoholic patient with beriberi, however, showed extremely high urinary porphyrin (369 γ/24 hours), unaffected by therapy with vitamin B_1 and marmite; this patient presumably had a permanent hepatic lesion resulting from the alcoholism. Apparently a combination of circumstances (e.g. alcoholism plus certain nutritional deficiencies) is needed to produce liver damage with consequent excess porphyrinuria.

MARION HORN PESKIN (Chem. Abstr.).

Urinary Excretion of Nicotinic Acid Among Normal Chinese, Pellagrins and Other Patients. Hou, Hsiang-Chuan, and Dju, Mei-Yu. [*Chinese Med. J.*, **61**, 192–8 (1942).]

The method of Swaminathan (*C.A.*, **34**, 4439[5]) was used to determine urinary nicotinic acid. The average figures obtained in mgm. per day were: 48 normal students 1·621; 6 cases of multiple vitamin deficiency 0·425; 15 cases of pellagra 0·715; 4 cases of beriberi 0·843. Other diseases, tuberculosis, dysentery, etc., also showed low urinary nicotinic acid. There was considerable individual variation in daily nicotinic acid excretion both in normals and in patients, but increased excretion followed promptly upon increased nicotinic acid intake.

WILLIAM H. ADOLPH (Chem. Abstr.).

Disturbances of Neuromuscular Excitability During Dietary Imbalance and Avitaminoses. III. Carbohydrate Imbalance in Rats Compared with Avitaminoses of the B Group and Especially Riboflavin Deficiency. Lecoq, Raoul, Chauchard, Paul, and Mazoué, Henriette (Hôpital Saint Germain-en-Laye, Paris). [*Bull. soc. chim. biol.*, **26**, 347–54 (1944); *cf. C.A.*, **39**, 2784[3], 4120[2].]

Rats were fed diets containing 54–72 per cent. of lactose or 25–35 per cent. of galactose and adequate vitamin supplements. Excitation of the central nervous system and polyneuritic degeneration were produced as in pigeons similarly fed, but in the rats these changes were not as readily detected by chronaximetric methods as in pigeons. The symptoms displayed by the rats resembled those of thiamine deficiency in pigeons, but more closely resembled those of riboflavin deficiency previously studied in rats.

IV. Eutrophic Rickets and the Humoral Rickets of Avitaminosis D ("Invisible" Rickets). [*Ibid.*, **27**, 129–35 (1945); *cf. C.A.*, **38**, 2078[4]].

In rats and in children the onset of rickets can be detected by chronaximetric methods before it can be detected by X-rays.

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V. Dystrophic Rickets and Spontaneously Curable Rickets. [*Ibid.*, 198–206.]

Changes in neuromuscular chronaxia in rats accompanying the production of rachitic symptoms by Ca insufficiency or the addition of $NaHCO_3$ or $SrCO_3$ to the diet are further discussed. L. E. GILSÓN (Chem. Abstr.).

Biochemical Study of Avitaminoses of the Pellagra Group. IV. Urinary Polyphenols with Blue Fluorescence (Alkaptonuric and Normal Urines). Raoul, Y. (Hôpital Foch, Paris). [*Bull. soc. chim. biol.*, **26**, 355–61 (1944); *cf. C.A.*, **38**, 3696[8].]

The urine of alkaptonuric patients has a much more intense blue fluorescence under ultraviolet light than has normal urine. Oxidation of the homogentisic acid (alkaptone) of the alkaptonuric urine to 2,4,5-trihydroxy-α-toluic acid further increases the blue fluorescence. Effects of variations in pH on the ultraviolet absorption spectra of homogentisic acid and its oxidation product are shown in graphs.

V. Protein-derived and Other Fluorescent Substances of Urine. [*Ibid.*, 361–7.]

Among the substances contributing to the blue fluorescence of human urine are polyphenols and indole derivatives of protein origin, some oxidation products of vitamin K, possibly some bile acid derivatives, and other substances of unknown origin and constitution. The addition of $Na_2S_2O_4$ to the specimen may increase the fluorescence by reducing nonfluorescent quinones to fluorescent polyphenols.

VI. Parallelism Between Reactions for Indole Derivatives and the Fluorescence of Normal Urine. [*Ibid.*, 494–5.]

The intensity of the fluorescence and the results of the Nencki-Sieber urorosein test (*J. prakt. Chem.*, **26**, 333 (1892) (red color produced by addition of concentrated HCl to defecated urine containing $Pb(OAc)_2$) were roughly parallel.

VII. Action of β-indoleacetic Acid on Growth. [*Ibid.*, 497–506.]

Indoleacetic acid, 150 γ per day, seemed to have a slight favorable effect on growth of young rats given either a complete diet or one partially deficient in various vitamins of the B group. L. E. GILSON (Chem. Abstr.).

Nutritionally Produced Cerebellar Disorder in Chicks, Not Associated with E Avitaminosis. Bird, F. H. (Univ. of California, Berkeley). [*J. Biol. Chem.*, **161**, 747–8 (1945).]

Chicks receiving a highly purified diet developed a syndrome involving incoordination of movements and convulsions, associated with cerebellar lesions. The diet resulting in this disorder had the following composition per 100 gm.: Water-washed casein 22·2, l(+)-arginine-HCl 0·3, glycine 0·9, l(−)-cystine 0·4, Ca gluconate 5·0, cellulose 5·0, soybean oil 3·0, fish oil 0·25, NaCl mixture (containing 0·49 per cent. Mn, 0·1 per cent. Cu, 0·05 per cent. Zn, 0·05 per cent. Al, 0·002 per cent. Co, and 0·04 per cent. I) 1·0, $Ca_3(PO_4)_2$ 3·5, K_2HPO_4 1·3, KCl 0·3, $MgSO_4$ 0·1, $Na_2SiO_3 \cdot 9H_2O$ 0·25, cholic acid 0·1, choline chloride 0·2, and glucose 56·2 gm. To every 100 gm. of the above mixture were added solubilized liver eluate equivalent to 4 gm. of solubilized liver, thiamine-HCl 0·5 mgm., pyridoxine-HCl 0·4 mgm., riboflavin 0·5 mgm., calcium d-pantothenate 1·5 mgm., nicotinic acid 1·0 mgm. 2-methyl-1, 4-naphthohydro-quinone diacetate 1·0 mgm., synthetic α-tocopherol 1·0 mgm., and biotin 0·01 mg. Chicks fed a compound ration composed of mixed natural foodstuffs showed no signs of disorder of the type described above.
HILDA H. WHEELER (Chem. Abstr.).

An Experimental Study of Disorders in the Permeability of the Cerebral Vessels ("the Blood-brain Barrier") Produced by Chemical and Physicochemical Agents. Broman, Tore, and Lindberg-Broman, A. M. (Lund, Sweden). [*Acta Physiol. Scand.*, **10**, 102–25 (1945).]

When the blood-brain barrier (BBB) is disorganized (vessel lesions), dyes like trypan blue can be seen to penetrate the vascular wall. In experiments on guinea-

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pigs, rabbits, and cats disorders in the BBB were produced either by direct injection into a cerebral artery, local application to pial vessels, or by one minute perfusion of cerebral vessels with test solutions. Distilled H_2O, hypertonic NaCl, acid, alkali, Na glycocholate, Na desoxycholate, EtOH, cobra venom, and bee venom were effective; adrenaline, acetylcholine, and histamine were ineffective. The noxious substances disturb the BBB function in much lower concentration if applied to the intimal rather than to the adventitial layer of the vessel. The former apparently is the site of permeability. Osmotic pressure or pH changes are effective in disorganizing the BBB only if fairly great. It seems probable that proteins, unless they have some toxic influence on the vessels, do not pass from the blood into the brain. Damaged permeability may make their passage possible. Local brain allergic reactions can apparently be produced by antigens (horse serum) injected intracerebrally. As a result of the mechanical injury, antibodies pass from the blood into the brain.

S. MORGULIS (Chem. Abstr.).

Nervous Excitation and the Chemical Dynamics of the Cell. Koshtoyants, Kh. S. [*Bull. acad. sci. U.R.S.S., sér. biol.*, 170–80 (1945) (*in English*, 180–1).]

In spite of qualitative differences between the excitation phenomena of differentiated cells via the nervous route and such phenomena as fertilization, growth, or differentiation of tissues, they are united by the leading role played in all these processes by special chemical compounds of the type of activators. A singular place among these chemical compounds is occupied by neurohumors or mediators of the nervous impulse. Research work during the last three decades resulted in a new classification of the nerves as suggested by Dale. This is based on the fact that one group of nerves exercises its influence through the system of choline esters (cholinergic nerves), the other through adrenaline-like substances (adrenergic nerves), and the third one apparently through histamine or like substances (histaminergic nerves). The leading problems in this domain are as follows: (1) The paths of biological synthesis (breakdown and stabilization) of the mediators; (2) the mode of their being released (" microsecretion " after A. Samojloff) in the course of excitation; (3) the paths and the mode of inclusion in the chemical transformations of the cell linked with their functional activity (the inhibitory phenomena inclusively); and (4) their connection with the decisive factors of generation of the bioelectric potentials. These four problems have been studied by Koshtoyants and his associates during the past years. Starting from comparative-physiological data Koshtoyants raised, in 1938, the question as to the connection between the paths of biological synthesis of acetylcholine and certain stages of carbohydrate metabolism. He showed, in collaboration with his associates, that a block (e.g. fluoride) of definite links of the carbohydrate metabolism results in a block of the impulse transmission of cholinergic nerves. A similar block of the nervous impulse called forth by a number of substances which exercise a definite action on cellular transformations contributes to the formulation of some conceptions regarding the metabolic nature of the nervous impulse. The transition from the " chemodynamics of rest " to " chemodynamics of excitation " is accomplished in different nerves along different enzymo-chemical lines, according to the chemical characteristics of their mediator, the modes of its release upon excitation, and the paths and mode of their inclusion in the metabolism of the excited tissue. As to cholinergic nerves, in the case of skeleton musculature, their mediator, acetylcholine, is intimately connected in its synthesis, breakdown, and action with the system of adenosinephosphoric compounds and with the depot of guanidine-phosphoric compounds. It is concluded from some experimental evidence that acetylcholine favors initially the release in the ionized state of Ca, Mg, and K (from the protein or lipide compounds with which they are combined), the latter activating or inhibiting the enzymic activity of adenosinetriphosphatase with all the implications concerning nervous mediation of the contractile process. In the important process of release of ions there probably participates acetic acid, which is formed through hydrolysis of acetylcholine under the influence of cholinesterase. The physiological significance of the hydrolysis phase of acetylcholine is emphasized, as its consequences for the excitation of protoplasm have not as yet been duly considered. These results were obtained by oscillographic studies.

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

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Metabolism in Different Parts of the Brain, Especially in the Epiphysis, Measured with Radioactive Phosphorus. Borell, Ulf, and Örström, Åke (Wenner-Gren Inst., Stockholm). [*Acta Physiol. Scand.*, **10**, 231–42 (1945).]

The highest P contents in percentage of wet weight were found in the epiphysis, in the anterior and posterior lobes of the hypophysis, choroid plexus, and in the substantia perforata. These experiments suggest a division of the brain parts into a group with very active P metabolism and a group with a slow P metabolism. The latter group consists of the olfactory lobe, parietal and occipital, thalamus, the anterior and posterior parts of tuber cinereum, corpus mammillare, cerebellum, pons, corpora quadrigemina and medulla oblongata, whereas the group with high P metabolism includes the parts enumerated with high P content, except the substantia perforata, which has a low P metabolism. The relative activities are compared by taking that of the cerebellum as 100. Forty minutes after an injection of radioactive P into the epiphysis 25 per cent. of it is in the form of free phosphate, 65 per cent. in acid-soluble esters, and 10 per cent. as compounds, insoluble in CCl_3CO_2H. The activity of the epiphysis is generally higher, and frequently 2–3 times higher than the activity of the hypophysis. Next to the epiphysis in metabolic activity are the choroid plexus and the lobes of the hypophysis, which are 4–5 times as active as the cerebellum, whereas all other components of the brain are generally of the same order as the cerebellum.

S. Morgulis (Chem. Abstr.).

Effect of Transcerebral Dielectrolysis of Certain Ions on the Circulation of the Retina. Bourguignon, Georges, and Baillart, Paul (École des hautes études, Paris). [*Compt. rend. soc. biol.*, **138**, 779–82 (1944).]

In 4 men, 1 woman and 1 rabbit, I, Br, Ca, Mg, Fe, K, and Na ions were introduced by iontophoresis from one electrode placed on the back of the neck and one placed over one eye (positive for metals and negative for halide ions). The retina was examined with an ophthalmoscope. In all cases, except with NaCl solution or distilled water, there was vasodilation of the retina and increase in peripheral blood pressure as measured in the arm. L. E. Gilson (Chem. Abstr.).

The Origin of Electricity in the Nervous System. Beutner, R., and Barnes, T. Cunliffe (Hahnemann Med. Coll. and Hosp., Philadelphia). [*Biodynamica*, **5**, 117–28 (1945).]

The origin of potentials on collodion membranes in contact with salt solutions is explained by the theory of phase boundary potentials. By use of the " oil-cell " method it was found that acetylcholine in an aqueous phase establishes a negative phase boundary potential with cholesterol. Solvents for nerve or brain extract, such as triacetin and tributyrin are being investigated for use in the " oil cell." Results secured furnish a satisfactory explanation of the origin and the pattern of the negative variations observed in the living nerve.

J. E. Webster (Chem. Abstr.).

Contents of Nitrogen and Phosphorus in Various Architectonic Formations of the Cortex of the Great Brain. Gurvich, E. E. (Inst. Mozga, Moscow). [*Byull. Eksptl. Biol. Med.*, **19**, *No.* 3, 60–3 (1945).]

N and P were determined in various regions of the cerebrums of 9 dogs. Analyses were made of brain substance freshly extirpated from the occipital (vision) region, the motor, sensory, and other anatomical divisions. The contents in N and P in different regions apparently depend on the histological structure of the cells.

D. I. Macht (Chem. Abstr.).

Electrical Pulsations in the Human Brain. Barnes, T. C. (Hahnemann Med. Coll., Philadelphia). [*Trans. N.Y. Acad. Sci.*, **7**, 87–9 (1945).]

Mainly a discussion of the physiological and other aspects of electroencephalography. Many physiological conditions (especially blood sugar) influence the brain waves. In normal persons with a blood-sugar value below 130 mgm. per cent., hyperventilation produces the slow delta waves characteristic of certain delinquent individuals. Sugar produces the energy of the brain and may yield AcOH, which combines with choline, forming acetylcholine (I), which is involved in the production

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of brain waves. Artificial electric brain waves can be produced by the contact of (I) with brain extract or with cholesterol extracted from the spinal cord.
W. C. TOBIE (Chem. Abstr.).

Vitamin C and Cholinesterase: II. Herschberg, A. D., Frommel, E., and Piquet, J. (Univ. Geneve). [*Helv. Physiol. Pharmacol. Acta*, **2**, 507–14 (1944) (*in French*); *cf. C.A.*, **38**, 2364⁹.]

The organs of scorbutic guinea-pigs react much more strongly to acetylcholine than normally because lack of ascorbic acid inhibits the activity of tissue cholinesterase. Addition of ascorbic acid to the bath or perfusion fluid restores the activity of the cholinesterase and the organs then react almost normally to acetylcholine. The activating action of ascorbic acid on muscle cholinesterase is also shown by its decreasing sensitivity of frog rectus abdominis muscle to acetylcholine. In guinea-pig serum the cholinesterase activity is lowest in winter and highest in summer. Sex is not a factor. L. E. GILSON (Chem. Abstr.).

Influence of Certain Reduction-oxidation Indicators on the Activity of Cholinesterase. Klein, Paul (Med. Clin. Heidelberg). [*Biochem. Z.*, **317**, 210–16 (1944).]

Thionine, toluidine blue, and methylene blue inhibit cholinesterase activity, but when these are in the leuco form the inhibiting effect is decreased.
S. MORGULIS (Chem. Abstr.).

Effect of Digitalis on Cholinesterase. Miquel, Ovidio, and Riker, Walter F., Jr. (Cornell Univ. Med. Coll.). [*Proc. Soc. Exptl. Biol. Med.*, **60**, 120–1 (1945).]

Digitoxin, ouabain, strophanthin and lanatoside C had no effect on the activity of serum cholinesterase or cholinesterase from the electric organ of the eel. These results confirm the observation that cardiac slowing by digitalis is not due to inhibition of cholinesterase. L. E. GILSON (Chem. Abstr.).

Influence of Amino Acids on Cholinesterase. Anti-acetylcholine Action of Histidine. Aron, E., Herschberg, A. D., and Frommel, E. (Univ. Geneve). [*Helv. Physiol. Pharmacol. Acta*, **2**, 495–505 (1944) (*in French*).]

In concentrations of 0·8–2·0 per cent. glycine, alanine, proline, and especially arginine, lysine and histidine increase the activity of serum cholinesterase *in vitro*. Cysteine and tryptophan have no such effect. Arginine and histidine sometimes double the activity. Histidine weakens the response of isolated frog muscle to acetylcholine by increasing the activity of the muscle cholinesterase. This explains the anti-acetylcholine action of histidine, lysine and arginine reported by other workers. Intravenous injection of histidine-HCl increases serum cholinesterase activity in guinea-pigs and man; in man 0·2 gm. produces an approximate 50 per cent. increase. L. E. GILSON (Chem. Abstr.).

Action of Essential Oils on Serum Cholinesterase. Caujolle, F., Vincent, D., and Franck, C. (Lab. pharm. matière med. faculté med. pharm., Toulouse). [*Compt. rend. soc. biol.*, **138**, 556–8 (1944).]

When added in the proportion of 1 : 10,000 to horse serum containing added acetylcholine, the essential oils inhibited the action of the serum cholinesterase by the following percentages: Peppermint 60, lavender flowers 58, lavender spike 42, bergamot 39, condiment sage 37, thyme 36, marjoram 35, lemongrass 34, petit-grain 32, clary sage 15, and rosemary 13 per cent. Some inhibition was evident with a 1 : 100,000 concentration of the oil in most cases. Menthol acted like peppermint oil; thymol had a much stronger action than thyme oil.
L. E. GILSON (Chem. Abstr.).

Determination of Cholinesterase Activity with Aid of the Glass Electrode. Sanz, M. C. (Hallerianum, Univ. Bern). [*Helv. Physiol. Pharmacol. Acta*, **2**, C29–32 (1944) (*in German*).]

A constant temperature device contains a small cell provided with a stirrer and glass electrode and connected to a saturated calomel electrode. Two or 3 c.c. of the solution to be tested is placed in the cell with 0·1 c.c. of 10 per cent. acetylcholine iodide solution, then 0·02 N NaOH is added from a microburet at such a rate as to keep the pH constant by neutralizing the AcOH as fast as liberated. The

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volume of NaOH solution added is noted every 2 minutes for 10–20 minutes, and a curve is plotted. The results compare favorably with those obtained by other methods. Two views of the complete apparatus are shown.

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

Changes in Cholinesterase Activity During the Rhythmic Polarization of Nerve Fibers. Babskii, E. B., and Minaev, P. F. (Pedagogic Inst., Moscow). [*Byull. Eksptl. Biol. Med.*, **19**, *No.* 6, 14–16 (1945); *cf. C.A.*, **40**, 1545[8].]

Previously it has shown that during the passage of a d.c. through nerve fibers or the central nervous system, the cholinesterase activity was increased at the anode and decreased at the cathode. The same effect is obtained by the rhythmic polarization of nerve fibers with a condenser. H. PRIESTLEY (Chem. Abstr.).

Serum Cholinesterase Activity in Experimental Liver Injury. Steensholt, Gunnar, and Venndt, Helge (Univ. Biskeni. Inst., Copenhagen). [*Acta Physiol. Scand.*, **10**, 23–30 (1945).]

Administration of $CHCl_3$ to dogs greatly increases the serum cholinesterase activity and decreases serum albumin. An interruption in administration of $CHCl_3$ caused an immediate fall in serum-cholinesterase activity, while resumption of the treatment caused it to rise again. It is concluded that liver injury produced experimentally is accompanied by a marked increase in serum cholinesterase activity and a simultaneous fall in serum albumin. S. MORGULIS (Chem. Abstr.).

Effects of Salts on True Cholinesterase. Mendel, Bruno, and Rudney, Harry (Banting and Best Dept. of Med. Research, Univ. Toronto, Can.). [*Science*, **102**, 616–17 (1945).]

Experiments show that the relationship between enzyme activity and substrate concentration is changed by the addition of salt to the medium. A stepwise increase in the salt concentration causes a gradual shifting of the optimal activity of the enzyme to higher levels of acetylcholine. With increasing concentrations of KCl an absolute increase in the rate of acetylcholine hydrolysis occurs at the optimum levels and beyond. When NaCl is used in the medium in equimolar concentrations, the activity-substrate concentration curves show the same general trend, but they are not identical. E. D. WALTER (Chem. Abstr.).

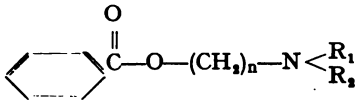
Electrotonic Changes of Activity of Cholinesterase in the Nerve Fibers. Babski, E. B., and Minaev, P. F. (Kafedry Fiziologii Cosudarst. Pedagogicheskogo Inst. im. V. I. Lenina, Moscow). [*Byull. Eksptl. Biol. Med.*, **18**, *No.* 3, 58–60 (1944).]

Frog nerve-muscle preparations were subjected by means of liquid nonpolarizable electrodes to the action of a direct-current of 0·02–0·1 ma. for 5–10 minutes. Acetylcholine was added to the emulsions to a concentration of 1 : 100,000. At the cathode the activity of the cholinesterase is decreased; at the anode it is increased. The content of unhydrolyzed acetylcholine is higher in the cathode emulsion and lower in the anode emulsion than in control emulsions. The changes in the activity of the cholinesterase may be due to the shift of ions in the nerve.

J. DAVIDSON (Chem. Abstr.).

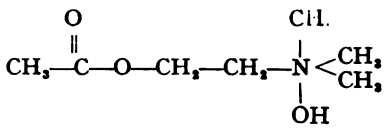
A Theory of Anesthetic Action Based upon Acetylcholine Competition. Schueler, Fred W. (Univ. Colorado, Boulder). [*J. Chem. Education*, **22**, 585 (1945).]

It has been known for some time that compounds having the structure—



will generally possess local anesthetic action. Cocaine is a well-known example. In this structure the essential features for anesthetic action are the Bz group, and the carbon chain terminating in the alkylated N. The problem concerning the local anesthetic class of drugs, illustrated above, therefore (if drug competition is assumed) reduces itself to discovering a substance occurring at the nerve-endings and essential for nerve impulse transference, which resembles the anesthetic drugs structurally. Such a resemblance is exemplified by acetylcholine—

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On the assumption that the local anesthetic drugs may compete with acetylcholine at the nerve-endings, the writer has carried on some experiments which lend support to his theory. In each instance, the rats used that received both acetylcholine and cocaine were freed from symptoms (or these were greatly reduced) possessed by the rats receiving cocaine or acetylcholine separately, i.e. they appeared normal.
R. K. CARLETON (Chem. Abstr.).

Effects of Choline and Acetylcholine Chloride upon Peripheral Nerve Fibers. Lorente de No, R. [*J. cell. comp. Physiol.*, **24**, 85–97 (1944).]

Study of the action of large concentrations of choline and acetylcholine chlorides upon activity in the sciatic nerve of the bullfrog indicates that these substances neither depolarize the nerve fibers nor prevent the conduction of impulses. It is concluded that release of acetylcholine is not the mechanism underlying the conduction of nerve impulses in peripheral fibers, but that ACh is a substance that participates in the metabolic activity of the fibers, although the importance of this participation is at present not understood. F. A. BEACH (Psychol. Abstr.).

Present Views on the Mode of Action of Acetylcholine in the Central Nervous System. Feldberg, W. (*Physiol. Lab., Cambridge, Engl.*). [*Physiol. Rev.*, **25**, 596–642 (1945).]

Acetylcholine (I) has both stimulating and depressant actions on the central nervous system, and both actions appear to be sensitive to atropine, which also antagonizes the central actions of eserine and prostigmine. (I) is bound in the tissue or cell granules, probably through the action of lipides and proteins. Synthesis of (I) is a property of both nerve cell and fiber; the process may occur in two stages—(1) anaerobic, depending upon the presence of adenosinetriphosphate, and (2) aerobic, depending on the presence of O. The release of (1) from cholinergic nerve fibres is generally regarded as the result of a K-ion mobilization during passage of nerve impulse; this is followed by a resynthesis, which is independent of the passage of the impulse. But it is believed that the K-ion mobilization has also a direct effect upon the resynthesis. The synthesis does not apparently bear any relationship to phylogenetic development and is roughly in this order: Frog>rat, guinea-pig>cat, dog, monkey brain. In different parts of the brain the activity is in the order: cerebral cortex>brain stem>medulla>spinal cord>cerebellum. The theory of the transmission across a number of synapses in the central pathway through the mediation of (I) is discussed. S. MORGULIS (Chem. Abstr.).

Effect of Inhibition of Glycolysis and Compounds Related to Glycolysis on Acetylcholine Synthesis. Torda, Clara, and Wolff, Harold G. (*Cornell Univ. Med. Coll., New York, N.Y.*). [*J. Biol. Chem.*, **162**, 149–54 (1946).]

The effect of various products of glycolysis, substances involved in the esterification of carbohydrates, and some substances inhibiting glycolysis, on acetylcholine production in minced frog brain was studied by a method previously described (*C.A.*, **39**, 1918[8]). Intermediary products of glycolysis and organic phosphates may increase the synthesis, and the energy required seems to be supplied by oxidative processes or by glycolysis under anaerobic conditions, although some acetylcholine may be synthesized in the absence of glycolysis and free O.
B. R. MURRAY (Chem. Abstr.).

Acetylcholine in the Mechanism of the Rise of Refractoriness of a Nerve. Zol'nikova, A. I. (*Fiziologicheskoi Lab. Tsentral. Inst. Kurortologii N. K. Zdrava S.S.S.R., Moscow*). [*Byull. Eksptl. Biol. Med.*, **19**, *No.* 3, 70–3 (1945).]

Experiments were made on muscle-nerve preparations of *Rana temporaria*. Acetylcholine 1 : 1000 prolongs both the absolute and relative refractoriness. Eserine and prostigmine 1 : 500 and 1 : 1000 inactivate the cholinesterase of the nerve and prolong the refractoriness. Atropine neutralizes the effect of eserine and prostigmine. D. I. MACHT (Chem. Abstr.).

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The Relation of Adrenaline to Acetylcholine in the Nervous System. *Burn, J. H.* [*Physiol. Rev.*, **25**, 377–94 (1945).]

Adrenaline, which has little action of its own in the nervous system, has a powerful effect modifying acetylcholine action, augmenting it in low concentration or inhibiting it in high concentration. This is also manifested by 2–3-fold increase in transmission of nerve impulses by adrenaline or by sympathetic stimulation, and represents possibly the mechanism for paralysis by fear. Adrenaline or sympathetic stimulation not only improves transmission at the neuro-muscular junction, but also increases the action of prostigmine. Adrenaline in low concentration definitely improves transmission through the sympathetic ganglion and in high concentration inhibits it. Thus, splanchnic stimulation becomes less effective after a large dose of adrenaline. The body becomes, therefore, less capable of maintaining a normal blood pressure after a massive dose of adrenaline, which explains the failures when a sudden stimulus causes a large discharge of adrenaline into the blood. S. MORGULIS (Chem. Abstr.).

Action of Procaine and Some of its Derivatives on the Contraction of Leech Muscle by Acetylcholine; their Cholinesterase Activity. *Hazard, R., Corteggiani, E., and Pelou, A.* (*École des hautes études, Paris*). [*Compt. rend. soc. biol.*, **138**, 427–9 (1944); *cf. C.A.*, **39**, 3587⁶.]

Procaine in 1 : 500,000 concentration makes leech muscle twice as sensitive to acetylcholine and in 1 : 9000 concentration makes it 50–200 times as sensitive. Still higher concentrations provoke contraction by themselves. Procaine acetylated on the N of the p-aminobenzoyl radical had no sensitizing action in high dilution, and in 1 : 4000 concentration had a desensitizing action. Higher concentrations caused contraction by themselves. Procaine iodomethylated on the N of the diethylaminoethyl radical had a slight sensitizing action in 1 : 8000 concentration; higher concentrations caused contraction by themselves. When procaine was both acetylated and iodomethylated, it acted like the acetylated compound mentioned above. Procaine and iodomethylated procaine have about the same inhibiting effect on the action of the cholinesterase of horse serum *in vitro*. Acetylation and acetylation plus iodomethylation greatly decrease the inhibiting action on serum cholinesterase. L. E. GILSON (Chem. Abstr.).

Polarization Effect Under the Action of Acetylcholine on the Nerve Commissure of Anodonta. *Babskii, E. B., Mershchikov, A. G., and Sheikhon, F. D.* [*Byull. Eksptl. Biol. Med.*, **19**, *No.* 4/5, 17–20 (1945).]

Experiments were made with large *Anodonta cygnea*. When acetylcholine is applied in concentrations of 1 : 10,000–1 : 1000 to the interganglionic commissure, a positive electric reaction is noted in the nerve tissue. This polarizing effect is usually equivalent to 5 to 6 mv., but in exceptional cases may be as high as 25–30 mv. The authors believe that this positive reaction is connected with the formation of acetylcholine. D. I. MACHT (Chem. Abstr.).

Antagonism Between Adrenaline and Acetylcholine on the Intestine of the Rabbit. *Gunn, J. A.* (*Univ. of Oxford*). [*Quart. J. Pharm. Pharmacol.*, **18**, 108–16 (1945).]

The physiological antagonism between adrenaline and acetylcholine has been determined on the isolated intestine of the rabbit. Solutions containing a constant amount of one drug were mixed with solutions containing varying amounts of the other drug until a mixture was found which produced complete physiological antagonism. The following conclusions are applicable only within certain limits of concentration of both drugs. On the same segment of intestine, when the concentration of one drug is kept constant, the antagonizing effect of the other drug is proportional to its concentration. On different segments of intestine, the ratio of activities of the two drugs varies. In 10 experiments 5 to 20 parts of adrenaline (average approximately 10 parts) was required to antagonize 1 part of acetylcholine. On the same segment of intestine, when the amount of one drug is increased an approximately proportional increase in the amount of the other is required for antagonism. This can be explained by a coincident corresponding relation between the physiological effects produced. W. O. E. (Chem. Abstr.).

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Acetylcholine Content of Peripheral Nerves During Degeneration. Muralt, A. v., and Schulthess, Gertrud v. (The Hallerianum, Bern). [*Helv. Physiol. Pharmacol. Acta*, **2**, 435–43 (1944) (*in German*); *cf. C.A.*, **39**, 1452⁹.]

Immediately after section of the sciatic nerve in the guinea-pig the acetylcholine content of the peripheral portion decreases rapidly. After 50–70 hours it is less than 10 per cent. of the original value and the nerve can no longer be stimulated to conduct impulses. After 100 hours no acetylcholine can be detected in the nerve.

Thiamine Content of Peripheral Nerves During Degeneration. Muralt, A. v., and Wyss, F. [*Ibid.*, 445–8.]

In the above experiments the thiamine content of the severed nerve began to fall soon after cutting, and in 50–70 hours had decreased to 40–50 per cent. of the original level. The ratio of combined to free thiamine was 9 : 1.

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

2. Pharmacology and Treatment.

Experimental Cocainism. I. General Toxicology, Addiction and Sensitization. Gutiérrez-Noriega, C., and Ortiz, V. Zapata (Inst. nacl. hig., Lima). [*Rev. med. exptl. (Peru)*, **3**, 279–306 (1944).]

Chronic oral, subcutaneous, or intravenous administration of cocaine to dogs resulted in increased sensitivity to the drug. The dogs became addicted to the injections. The effects were manifest most rapidly by intravenous injection, least rapidly by oral administration. The convulsive dose and lethal dose often coincided, and the minimal lethal dose was only twice the optimal stimulating ones. The minimal lethal dose given orally was 2–2·5 times as great as that intravenously or subcutaneously.

Action of Cocaine on Nonaddicted Human Sujbects. Ortiz, Vincente Zapata. [*Ibid.*, 307–16.]

Cocaine in doses of 1 to 3·5 mgm./kgm. body-weight caused unimportant changes other than the hyperthermic effect, which might reach subfever values with the larger doses.

Action of Coca and Cocaine on Addicted Human Subjects. Mendizábal, Francisco Riemberg. [*Ibid.*, 321–8.]

Chewing of coca leaves or administration of 4 mgm./kgm. body-weight of cocaine results in slight hyperthermia, moderate tachycardia, rise in blood pressure, and stimulation of the nervous system. An increase in basal metabolic rate greater than normal might be observed.

Action of Cocaine on the Resistance to Fatigue in the Dog. Gutiérrez-Noriega, Carlos. [*Ibid.*, 329–40.]

Subcutaneous administration of 4 mgm./kgm. body-weight of cocaine resulted in 69–150 per cent. increase in resistance to fatigue in the swimming test. Increasing the dose to 8 mgm./kgm. resulted in increases of 3·9–75 per cent., and to 10 mgm./kgm. resulted in increases of 36·6–103 per cent. Cardiazole, 5 mgm./kgm., did not modify the resistance to fatigue.

Historical Data on Cocaine Addiction in Peru. [*Ibid.*, 341–53.]

H. L. Williams (Chem. Abstr.).

Effect of Phenamine on the Chronaxia of Human Peripheral Nerves and Skeletal Muscle. Filippova, A. G., (Tsentral. Inst. Kurortologii i Klin. Pervnykh Bolezne, Viem, Moscow). [*Byull. Eksptl. Biol. Med.*, **18**, *No.* 4/5, 69–70 (1944).]

The chronaxia was investigated in 17 wounded patients with injured spine and spinal cord and spastic paralysis, as well as in healthy individuals. With neck wounds the muscles and nerves of the upper and lower extremities were investigated, and with chest wounds only the muscles and nerves of the lower extremities. The chronaxias of the median, radial, ulnar, peroneal and tibial nerves and the biceps, triceps, gastrocnemius and tibialis anterior muscles were determined.

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Phenamine was given internally in dosage of 10 to 30 mgm. 2–3 times a day several days in succession or every other day. It was found to shorten the chronaxia of motor nerves and skeletal muscles, in some cases to $\frac{1}{10}$ or $\frac{1}{15}$ the original value. The clinical picture also changed, for spasms decreased and tonus was lessened. The intensity and the duration of the effect depended on the doze of phenamine. For comparison, the effect of ephedrine (50 mgm. given subcutaneously) was studied. This increased the chronaxia, spasms became stronger, and muscular tonus increased. Healthy individuals given single doses of 10–40 mgm. of phenamine showed a definite shortening of the chronaxia of the peripheral nerves and skeletal muscles.
G. LEBEDEFF (Chem. Abstr.).

Anticonvulsant Effects of Steroids. Spiegel, E. A., and Wycis, H. T. (Temple Univ. School of Med., Phila., Pa.). [*J. Lab. Clin. Med.*, **30**, 947–53 (1945).]

Desoxycorticosterone acetate, progesterone, testosterone, acetoxypregnenolone, androstenedione, and dehydroandrosterone increased the threshold of electrically induced convulsions in white female rats of 100–150 gm. wt. Male rats were found resistant to the anticonvulsant activity. The last three of the above compounds showed a definite margin between anticonvulsant and hypnotic doses. The following compounds had no, or only slight, anticonvulsant effect: Cholesterol, allocholesterol, cholesteryl bromide, epicholestanol, stigmasterol, stigmasterylacetate, α-spinasteryl acetate, ergosterol, ergosteryl acetate, α-ergostenyl acetate, dehydrocholic acid, desoxycholic acid, Δ^5-3-acetoxycholenic acid, sarsasapogenin acetate, pseudosarsasapogenin acetate, diosgenin acetate, pseudodiosgenin acetate, α-estradiol benzoate, theelin in oil, 6-acetoxy-α-progesterone, etiocholan-3(β)-ol-17-one acetate, Δ^5-pregnen-3(β)-ol-20-one acetate, $\Delta^{5}16$-pregnadien-3β-ol-20-one acetate, stilbestrol.
WM. M. GOVIER (Chem. Abstr.).

The Central Stimulant Action of Some Vasopressor Amines. Warren, Marshall R., and Werner, Harold W. (Wm. S. Merrell Co., Cincinnati, O.). [*J. Pharmacol.*, **85**, 119–21 (1945).]

In adult male white rats, at 26°, the subcutaneous injection of 7 common vasopressor amines in doses equal to fractional parts of the LD 50 as determined at 26° showed that benzedrine, ephedrine and propadrine (norephedrine) caused the greatest central stimulant action as measured by the total activity of the rats. Maximal stimulating effects were observed with ephedrine at 5, 10 and 20 per cent. of the LD 50, with propadrine at 10 and 20 per cent. of the LD 50, and benzedrine at 20 per cent. of the LD 50. Vonedrine (dl-1-methylamino-2-phenylpropane) produced a slight stimulation at 20 per cent. of the LD 50 and privine, tuamine (dl-2-aminoheptane) and neosynephrine had no significant effect at 20 per cent. of the LD 50.
L. E. GILSON (Chem. Abstr.).

A Cycle of Morphine Addiction. Biological and Psychological Studies. 1. Biological Investigations. Williams, Edwin G., and Oberst, Fred W. (U.S. Pub. Health Service Hosp., Lexington, Ky.). [*U.S. Pub. Health Repts.*, **61**, 1–26 (1946).]
J. A. KENNEDY (Chem. Abstr.).

Marihuana Activity of Cannabinol. Loewe, S. (Cornell Univ. Med. School, Ithaca, N.Y.). [*Science*, **102**, 615–16 (1945).]

The availability of larger quantities of cannabinol and the finding that propylene glycol is an effective solvent for intravenous preparations have made possible a study of the effects of larger doses. Cannabinol, generally believed to be an inert component of hemp oil, is shown to have marihuana activity. The significance of this observation with regard to the relationship between structure and activity in the class of cannabinols is discussed.
E. D. WALTER (Chem. Abstr.).

Effects of Application of Potassium Chloride to the Motor Areas of the Cerebral Cortex in the Dog. Moussatche, H. (Inst. Oswaldo Cruz, Rio de Janeiro). [*Rev. brasil biol.*, **5**, 407–12 (1945).]

Fibrillation and clonic convulsions of the muscles controlled by the centers were produced. In this respect KCl acts like acetylcholine, but not so strongly. Possibly acetylcholine acts by mobilizing the K ion.
L. E. GILSON (Chem. Abstr.).

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Inhibition by Sugars of the Excitant Action of Caffeine and Theobromine on the Nervous System. Chauchard, Paul, Mazoué, Henriette, and Lecoq, Raoul (École hautes études, Sorbonne, Paris). [*Compt. rend. soc. biol.*, **139**, 12–13 (1945).]

In rats the disturbance of chronaxia ordinarily produced by intraperitoneal injection of 5–10 mgm. of caffeine or theobromine is prevented by simultaneous or subsequent subcutaneous injection of 0·5 c.c. of a 5 per cent. solution of lactose, sucrose, galactose, or glucose, or by giving small amounts of these sugars orally.
L. E. GILSON (Chem. Abstr.).

Metrazole Convulsions. Leonardi, Félix Grillo (Facultad med., Lima). [*Rev. med. exptl. (Lima, Peru)*, **4**, 37–51 (1945).]

Subconvulsive doses showed a tendency towards habituation. Some cases of sensitization were observed. Increasing the dose increased the length of the attack, reduced the latent period, and increased the post-convulsive depression. The repeated convulsive dose was two or more times the threshold dose. The lethal dose was 2·5 to 5 times the threshold convulsive dose. This indicated an ample safety range. H. L. WILLIAMS (Chem. Abstr.).

Ammonium Phenobarbital: Preliminary Note. Quesada, Rafael. [*Rev. asoc. bioquim. argentina*, **12**, 195–6 (1945).]

The NH_4+ salt of phenobarbital was precipitated by bubbling NH_3+ through a saturated solution of ethylphenylbarbituric acid in acetone, forming white crystals, which in water solution are stable for at least 20 days. The pharmacological effect seems superior to that of phenobarbital. F. FROMM (Chem. Abstr.).

Studies on Barbiturates. XXVIII. Effect of Succinate and Fumarate in Experimental Barbiturate Poisoning. Corson, Samuel A., Koppanyi, Theodore, and Vivino, A. Earl. [*Anesthesia and Analgesia*, **24**, 177–92 (1945); *cf. C.A.*, **39**, 2330³.]

Neither disodium succinate nor fumarate in small or relatively large doses exhibited any life-saving effect on dogs, cats, rabbits, and rats poisoned with long- or short-acting barbiturates. Although these drugs increased urinary output and in many instances augmented the percentage excretion of barbiturate, this effect did not appreciably shorten recovery. G. H. W. LUCAS (Chem. Abstr.).

The Antidiuretic Action of Barbiturates (Phenobarbital, Amytal, Pentobarbital) and the Mechanism Involved in this Action. Bodo, R. C. de, and Prescott, K. F. (N.Y. Univ. College of Med.). [*J. Pharmacol.*, **85**, 222–33 (1945).]

Normal dogs, in the post-absorptive state and in water equilibrium, excreted ingested water (40 c.c./kgm.) or intravenously infused water (25 c.c./kgm.) almost quantitatively within 3 hours. Phenobarbital-Na given intravenously in half the anesthetic dose of 0·04 gm./kgm. either 40 minutes after the administration of water by stomach or a few minutes before the intravenous infusion of water, inhibited water diuresis about one-half. Smaller doses had no consistent action, and full anesthetic doses had no more effect on water diuresis than the half anesthetic dose. Still larger doses caused a greater degree of inhibition. In similar experiments with amytal-Na and pentobarbital-Na the results were irregular, and inhibition of water diuresis occurred only in some of the dogs. The effects of repeated doses of pentobarbital-Na were inconsistent. In dogs in which the entire neurohypophysis (infundibular process, infundibular stem, and media eminence) was destroyed and in which, as a result, permanent diabetes insipidus developed, water excretion was not inhibited by any of the barbiturates, even in full anesthetic doses. In dogs in which ordinary hypophysectomy was performed, leaving some parts of the neurohypophysis intact (no permanent diabetes insipidus produced), the barbiturates inhibited water diuresis. Phenobarbital-Na in half or full anesthetic doses did not inhibit saline diuresis (after injection of isotonic Na_2SO_4 solution), or inhibited it only in part of the dogs (after isotonic NaCl solution).
L. E. GILSON (Chem. Abstr.).

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