is gradually forced forward and upward in line with the external auditory meatus and glabella until the dura is pierced. In an ordinary-sized adult the needle penetrates to a depth of from 4 to 5 cm. before piercing the dura, and in this position there is usually a distance of from 2.5 to 3.0 cm. between dura and medulla. The conclusions of the author are based on forty-three punctures performed in twenty cases. The procedure was found to be of value in the diagnosis of postmeningitic subarachnoid block, and was also employed in the treatment of a selected group of cases of cerebral syphilis and in one case of epidemic meningitis. Used in conjunction with lumbar puncture it proved of value in the diagnosis of cord compression; where there is an obstruction to the free passage of fluid in the spinal subarachnoid space by tumour, Pott’s disease, etc., fluids taken from above and below the site of compression may show marked chemical differences.

R. M. S.

SENSORIMOTOR NEUROLOGY.

[59] The coincidence of cervical ribs and syringomyelia.—Peter Bassoe. Arch. of Neurol. and Psychiat., 1920, iv, 542.
The frequent association of syringomyelia and scoliosis is well known, but the co-existence of cervical ribs with that disease has not attracted much attention, although a number of cases have been recorded in the literature. The author gives the clinical histories of three patients in whom this combination was found. Operative treatment, although successful from the surgical point of view, does not always lead to disappearance of local symptoms, and may be followed by severe hypochondriasis. Cases of cervical rib must be regarded with suspicion, and the patients accepted as possessed of well-balanced minds and structurally normal nervous systems only after close scrutiny.

R. M. S.

In this remarkable case the heart was dilated, its rate was slow, and electrocardiographic tracings showed an increase of the As-Vs interval in all three leads. In a footnote the authors state that they have since found the same changes in two more cases. This is apparently an entirely new observation, with important bearings on the problem of the relation between the ductless glands and the functions of the heart.

W. J. Adie.

In this paper the author gives a clear and succinct account of the somewhat confusing syndromes which may arise from extracranial lesions of the last four cranial nerves, and contributes notes on three cases which came under his observation. The number of syndromes appears to be limited only by the possible combinations of complete or incomplete paralyses of these several cranial nerves, and the descriptive ability of the various observers.

R. M. S.
ABSTRACTS


In a boy, age 8, whose right arm had been weak since birth, the distribution of the palsy was as follows: complete paralysis, with absence of electrical excitability—serratus, rhomboids, trapezius inferior; weakness and diminished excitability—deltoid; slight weakness with normal reactions—biceps, triceps, pectoralis major, latissimus dorsi. The paralysis therefore was greatest in muscles that are usually spared in upper plexus lesions, and the muscles of Erb’s group were only partially injured. A glance at a diagram of the brachial plexus will show that the injury to the roots C 5 and 6 must be higher in this case than in Erb’s palsy, and above the origin of the long thoracic, dorsalis scapulae, and suprascapular nerves; that is, proximal to the point where C 5 and 6 emerge from the scalene muscles and unite to form the upper cord. According to the author the case is unique.

W. J. Adie.


Spiller reports a case of acquired athetosis, resembling progressive lenticular degeneration in some ways, but differing from it in its chronicity and in the absence of tremor. The patient, a girl, when about 5 years of age, was noticed to drag her left lower limb, and to have lost the use of her left upper extremity. Later, the paralysis affected other parts of the body, and assumed a spastic character; marked inco-ordinate movements were observed in the hands and feet. Articulation was difficult and defective. Forty-five years later all the limbs showed contractures, and there was some muscular atrophy. Her muscular system was in a state of continuous and universal athetosis; the lower jaw was frequently drawn down and to the left. The arms, hands, legs, and feet were most involved and to an extreme degree, the neck was less involved, and the trunk only slightly affected. There was a spastic scoliosis. The tendon reflexes were not obtained, possibly because of the spasticity. Both pupils showed myosis. Speech was peculiarly explosive and indistinct, but occasionally she was able to utter words with comparative ease and distinctness. She had difficulty in chewing and swallowing. Her mentality was poor.

At the necropsy the liver was found in a state of chronic passive congestion and red atrophy. Each lenticular nucleus was about one-half the normal size. The globus pallidus and caudate nucleus were both atrophic, but neither showed the peculiar tissue seen in the putamen. The latter had on each side a worm-eaten appearance, and contained numerous small holes. Microscopic examination of these areas showed shreds of tissue containing numerous neuroglia nuclei, with here and there a much altered nerve-cell; the blood-vessels were much thickened, and the perivascular spaces enlarged. The Betz cells of the paracentral lobules showed pro-
nounced chromatolysis. The motor anterior horn cells of the spinal cord were on the whole normal, and the pyramidal tract throughout the brain and spinal cord appeared healthy.

In an interesting discussion the author reviews extensively the literature on the various disorders attributed to disease of the lenticular nucleus. The group is a formidable one, and includes progressive lenticular degeneration, pseudosclerosis of Westphal and Strümpell, Huntington’s chorea, paralysis agitans, spastic pseudobulbar palsy with contractures and choreo-athetoid movements of Oppenheim and Vogt, and Freund and Vogt, Oppenheim’s dystonia musculorum deformans, double athetosis, von Bechterew’s hemitonia apoplectica, certain forms of carbon-monoxide poisoning, the paramyoclonic type of lethargic encephalitis, arteriosclerotic muscular rigidity, and certain forms of senile dementia, which Strümpell thinks belong to his amyostatic syndrome.

R. M. S.

[64] Dystonia lenticularis (dystonia musculorum deformans).—E. W. Taylor. *Arch. of Neurol. and Psychiat.*, 1920, iv, 417.

The author describes two cases of dystonia lenticularis occurring in the same family; the parents were Russian Jews. Both patients were attacked in their 7th year, but otherwise the clinical manifestations were very dissimilar.

**Case 1**, a schoolboy, age 11, appeared, on admission to hospital, quite normal except for the motor disorder, which was said to have commenced in the upper extremities. Variation in muscle tone was a conspicuous feature; some muscles were firm and elastic, others were flabby and toneless. Any voluntary movement of the body or limbs evoked an extraordinary spasm, which remained until the boy again placed himself in a position of rest. When he lay quietly in bed there was no movement of the body, but the right arm was held constantly in extension and extreme inward rotation, with wrist and fingers sharply flexed. In walking he forged forwards with the pelvis sharply tilted, head and chest thrown violently back in a position of opisthotonus, making extreme inco-ordinate choreiform or athetoid movements to retain his balance and progress. This apparently was only possible under certain circumstances by twisting the body violently. Often in accomplishing progression the patient turned half or completely round, and took a step backwards; a short distance was sufficient to exhaust the patient. The facial muscles showed neither spasm nor contortions, and electrical examination of all muscles gave normal reactions. There was no muscular atrophy, and the reflexes were unchanged.

**Case 2**, age 13, sister of the foregoing patient, was a normal child up to the age of 6. In her 7th year choreiform symptoms appeared, manifesting themselves first as a twitching of the neck, and gradually spreading to the body and limbs. Progressive mental enfeeblement was also noted, and eventually she could only utter inarticulate sounds. When the patient came under observation she was in a state of extreme emaciation and weakness. The most obvious disturbance was a constant choreiform
movement extending over the entire body and limbs. The facial muscles also took part in the involuntary movements, and she could only open the mouth by making extraordinary facial contortions. The tongue could not be protruded. All four limbs were held in a flexed position, and could not be passively extended. The disease terminated fatally, but under conditions which precluded a post-mortem examination.

The writer remarks that experience has amply shown that a syndrome, without a definite pathological anatomy and with a still less ascertained etiology, should never be regarded as a ‘disease entity’. It is therefore desirable to give up for the present the attempt to circumscribe this disease further than to regard it as one of the outstanding symptom-groups of lenticular or basal ganglion disease.

R. M. S.


The question of the possible substitution of function by some other motor tracts for that lost by complete interruption of the cortico spinal system is of considerable interest. Such restitution is always incomplete. It was supposed by Mann, who emphasized the familiar predilection-type of pyramidal palsy (extensors of arm, flexors of leg), that this substitution of function affected the extensors of the leg because of their importance in standing and walking, and was procured by way of the homolateral hemisphere and the uncrossed pyramidal tract. Mann’s view cannot be maintained, however, both for experimental and clinical reasons, and many others (Rothmann, Forster, v. Monakow, etc.) hold that such restitution as ensues is of subcortical origin.

The author gives in tabular form a list of symptoms of pyramidal and extrapyramidal lesions respectively, to which, however, especially in regard to the latter, much exception may be taken: notably, his contrast of the contracture-attitudes in the two cases is inaccurate and incomplete. The motor cortex proper is the seat of isolated volitional movement and of all synergies that have been acquired, as also of motor power. Such weakness as may be found in extrapyramidal disease is the result of imperfect action of the peripheral neurones. The spasticity of pyramidal lesions is the result of loss of cortical inhibition. Fine hand and finger movements, being a late acquisition, cannot be replaced, though old reflex automatisms may make their appearance again. Extrapyramidal disease brings about a fixation-reflex, agonists and antagonists being alike involved, in contradistinction to the reciprocal innervation of cortical origin. It is doubtful whether phylogenetic considerations will explain the differences between the motor symptoms of the two types of lesion. The ape is more of a cortical animal than some suppose, and analogies between the attitudes and movements of, say, infantile cerebral hemiplegia and those of the ape, do not necessarily indicate a phylogenetically old and presumably subcortical origin. The author favours the view of Rothmann, which associates the
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Mental disorders with predilection-type with the assumption of the erect attitude. Importance is attached to a diversion from the cortex, in pyramidal cases, of the normal stream of afferent impulses, which pass by a new route and awaken subcortical activity. It is assumed that the subcortical or extrapyramidal motor system serves ordinarily to support or reinforce, and not to counteract, the activity of the main corticospinal system.

S. A. K. W.


The original optimism as to complete recovery in non-fatal cases of this disease has not been justified by experience, and consideration of the intensity of the pathological changes involved makes it unlikely that this should be so. Not only have somatic sequelæ occurred, but abnormalities in the behaviour of the individual have been noted. In a group of six children, four boys and two girls, age 5 to 14½ years, who had passed through an attack of encephalitis with complete somatic recovery, the following mental abnormalities were noted. Their mental status was characterized by purposeless, impulsive motor acts, marked irritability, definite disorders of attention, distractibility and changing variable mood, inadequate and inconsistent emotional reactions, marked insomnia, and, in two cases, precocious sexual feelings and intense eroticisms. The authors consider these changes, which are elaborated in the case-histories accompanying the article, are the result of purely physical changes which result from the destruction of some nerve-cells and the irritation of others by the prolonged resolution of mesodermic inflammatory reactions.

Drug treatment was not found efficacious; but hydrotherapy, suitable light occupation, and a general non-simulating régime helped the patients considerably.

R. G. Gordon.

[67] Choreo-athetoid and choreopsychotic syndromes as clinical types of sequelæ of epidemic encephalitis.—La Salla Archambault. Arch. of Neurol. and Psychiat., 1920, iv, 484.

The writer lays stress on the remarkable protean symptomatology in epidemic encephalitis, which he suggests is dependent on regional variations in the focal predominance of the underlying histological changes. The combination of choreiform twitchings and acute psychotic disturbance as the salient manifestations is not uncommon, and choreo-athetoid or frankly athetoid syndromes may be observed as sequelæ of the disease. Variations in the symptomatology of epidemic encephalitis are possibly due to individual variations in the relative susceptibility of different levels of the cerebrospinal axis, and it is suggested that regional exhaustion from prolonged physiological hyperactivity or from previous disease may be an important factor in the localization of lesions produced by infections of the central nervous system.

Perusal of this paper will repay the reader, although it cannot be said to add much to our knowledge of epidemic encephalitis.

R. M. S.

The author quotes cases to show how relapses may occur and how a chronic state of invalidism and fatigability may be induced. He concludes that: (1) Not all cases of epidemic encephalitis die or recover. Some drift into a subacute or chronic state which lasts many months. In some cases a distinct relapsing tendency may be observed, in which newly-localized symptoms may appear, and in which an almost typical recurrence of the symptoms observed at the onset may be reproduced. (2) The irritant causing the root pains and myoclonic muscle-contractions of epidemic encephalitis undoubtedly involves successively different segmental levels of the cord or its corresponding nerve-roots, giving rise to descending and ascending forms of radiculitis. (3) The motor nuclei and roots of the cranial nerves are not immune to the irritant which causes symptoms of radiculitis in the various spinal levels of motility. (4) Even the most mild attacks of epidemic encephalitis frequently leave the afflicted person incapacitated for full participation in his work for many months. (5) The symptoms of epidemic encephalitis are protean in character, and tend to prove that both the central and peripheral nervous structures are susceptible to some irritant generated by the invading micro-organism.

R. G. Gordon.

[69] **The frequency of albuminuria with casts in epileptics following convulsive seizures.**—Nathan Novick. *Arch. of Neurol. and Psychiat.*, 1920, iv, 546.

The presence of albumin with casts in the urine voided immediately after epileptic attacks has been variously accounted for; by some it is regarded as due to admixture of semen liberated by pressure on the seminal vesicles; by others it has been attributed to muscular contractions. Novick, who investigated the urine in a series of 60 cases of epilepsy, reached the following conclusions: (1) In the series, 66 per cent showed albuminuria with granular casts after every seizure. Two cases, diagnosed clinically as hysteria, gave no evidence of either albumin or casts in the urine after repeated examinations following epileptiform seizures. (2) Albuminuria with casts persists for from twenty-four to forty-eight hours subsequent to attacks in some genuine epileptics. (3) The frequency of seizures, while apparently holding no relation to positive urinary findings, tends in general towards regularity of urinary abnormalities after attacks. (4) The duration of seizures seems to bear no relation to subsequent albuminuria. (5) Seminal fluid as a contamination factor giving rise to albuminuria after epileptic seizures does not play a very great rôle.

R. M. S.

[70] **Wounds of the head and compensation laws.**—Charles L. Dana. *Arch. of Neurol. and Psychiat.*, 1920, iv, 479.

The syndrome of non-fatal, non-destructive wounds of the head is quite a definite one, and is a picture of a generalized traumatic neurosis or psychoneurosis with certain head symptoms which have a possible legitimate
irritability, anxiety, disorders;

THE report Prominent basis.

fortable by it, partial deafness, (2)
centres.

in those especially helpless functional troubles and even grow
cerebellar hemispheres, early and the patient of cerebellar all more
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Hypotonia was not noted, and muscular force was well preserved. There was no constant tendency to deviate in Bárány’s pointing tests, and no spontaneous nystagmus.

The patient died suddenly, and the brain and cord were alone examined. Transverse section of the cerebellum revealed the presence of old symmetrical areas of softening in the central white matter of both hemispheres; on the left side the destruction of tissue was greater, the dentate and roof nuclei being destroyed. Three other lesions were

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basis. Prominent among the latter are headache, vertigo, insomnia, irritability, anxiety, depression, memory defects, fatigability, tinnitus, partial deafness, and loss of weight. Dana makes the following classification: (1) In about 10 per cent of compensation cases with head wounds the patient has the main features of the syndrome, and is just made uncomfortable by it, but there is no constitutional disturbance of the nervous centres. (2) In more than half the cases the patient is anxious, worried, nervous, and depressed, and cannot concentrate or work. He becomes self-centred, exaggerates, and refuses to attempt to work or to do anything but wait for some new treatment that will make him well. (3) In another large percentage of cases a definite attitude of exaggeration or plain malingering develops.

When the question of compensation is settled the symptoms do not always disappear, and in similar head injuries in non-legal cases they may continue and even grow worse. Such cases can hardly be considered victims of neuroses or psychoses. They are suffering from traumatic conduct disorders or from antisocial reactions to a painful or incapacitating injury. Many patients can be relieved entirely if properly and skilfully handled, but re-educational and reconstructive measures must be employed early and energetically. The patient must be told emphatically that his functional troubles will receive no recognition as a permanent disability.

R. M. S.

[71] A clinical and anatomic study of a vascular lesion of both cerebellar hemispheres.—WALTER F. SCHALLER. Arch. of Neurol, and Psychiat., 1921, v, 1.

The report of a case in which there was destruction of tissue in both cerebellar hemispheres, with conservation of the vermis and vestibular system. The patient, a man, age 58, had a ‘stroke’ which rendered him practically helpless owing to great motor inco-ordination in all extremities, especially in those of the left side. When seen three years later there was no disturbance of intelligence or emotion. Speech was typically scanning. The patient could scarcely stand even when assisted, and was quite unable to walk; yet he was able to sit upright for hours at a time without discomfort or difficulty. Analysis of his disturbed co-ordination showed the presence of dysmetria, dysdiadokokinesia, and dyssynergia: all more marked on the left side. The most striking symptom was that of cerebellar catalepsy. When the patient lay on his back, with the thighs adducted and flexed on the pelvis, and the legs flexed on the thighs, after a few unsteady movements, a remarkable immobility of the lower limbs ensued. Hypotonia was not noted, and muscular force was well preserved. There was no constant tendency to deviate in Bárány’s pointing tests, and no spontaneous nystagmus.

The patient died suddenly, and the brain and cord were alone examined. Transverse section of the cerebellum revealed the presence of old symmetrical areas of softening in the central white matter of both hemispheres; on the left side the destruction of tissue was greater, the dentate and roof nuclei being destroyed. Three other lesions were
encountered, the highest being situate in the right crista. Further examination showed a practically complete degeneration of the left superior peduncle, degeneration of the right red nucleus and in Forel's field, also of the left restiform body, and of the right inferior olive. A tract of degeneration on the left side of the spinal cord was identified as Hellweg's bundle. The absence of nystagmus in this case was explained by the practically intact vestibular system, and the conservation of static equilibrium by the normal condition of the vermis. The destruction of the left restiform body was regarded as being related to the lesion of the left dentate nucleus, and the author suggests that the central tegmental tract, circumolivary fibres, internal fibres of the olive, and Hellweg's bundle are more or less intimately connected with each other.

R. M. S.

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The subject of the pharmacological investigations was an adult male who developed two large pulsating cerebral hernæ after double subtemporal decompression. By enclosing the right hernial mass in a plethysmograph, satisfactory pulse-tracings were obtained. The exhibition of amyl nitrite caused a marked dilatation of the brain-vessels. Epinephrin induced a primary constriction, followed by a marked dilatation. Caffeine administered intravenously produced no demonstrable change. Pituitary extract caused a dilatation of the brain-vessels which was accompanied by a distinct general pallor of the face—the so-called 'leuko-reaction'.

R. M. S.

[73] The oculocardiac reflex (Dagnini-Aschner phenomenon)—its use in medicine and psychology.—Santer Naccarati. Arch. of Neurol. and Psychiat., 1921, v, 40.

Compression of the eyeball causes a slowing of the radial pulse, together with lowering of blood-pressure and modification of respiratory rhythm. This phenomenon was first reported by Dagnini, and later in 1908 Aschner published a paper on the same subject. Since this date the reflex has been investigated in many diseases, with somewhat conflicting results. It is customary to speak of a normal oculocardiac reflex when the pulse is retarded 5 to 12 beats per minute. When the pulse is reduced more than 12 the reflex is exaggerated. When the retardation does not exceed 4 the reflex is abolished. When, instead of retardation, acceleration occurs, the reflex is inverted. The use of the terms 'normal', 'exaggerated', or 'inverted' is somewhat misleading and ambiguous, and Naccarati suggests instead that the difference in one minute between the pulse-rate without ocular pressure and the pulse-rate with pressure be always indicated in full with a positive or negative sign. This algebraic difference should be called the reflex index. The pulse-rate should also be given, as the value of a reflex index is not absolutely the same in a bradycardiac as in a tachycardiac.