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10 per cent aqueous gelatin solution, 5 c.c.; warm glycerol, 5 c.c.; warm 1·5 per cent agar suspension, 5 c.c.; 5 per cent aqueous hydroquinone solution, 2 c.c. In preparing the reducing mixture the agar suspension is added after mixing the silver nitrate, gelatin, and glycerol, and the hydroquinone stirred in just before using. (7) After the solution is reduced, and the smears have turned a light brown, remove and rinse in 5 per cent sodium thiosulphate solution. (8) Rinse in distilled water. (9) Absolute alcohol, xylol, balsam.

R. M. S.

VEGETATIVE NEUROLOGY AND ENDOCRINOLOGY.


This is a short and interesting paper, accompanied by a good bibliography. The author brings together briefly the evidence in favour of the theory that scleroderma is of nervous origin, and that the particular part of the nervous system involved is the sympathetic. She says that the first suggestion that sclerodermal changes might be due to nervous disturbance arose from their limitation in occasional cases to the area supplied by a particular peripheral nerve, and she refers to thirty such cases. Many cases of the limitation of the changes to the distribution of a particular spinal root or segment, she states, are on record. Cases in which the distribution is symmetrical, and the rare cases in which the whole of one side of the body is affected, support the theory. Furthermore, scleroderma has sometimes been found in association with undoubted nervous diseases—e.g., herpes zoster, myelitis, syringomyelia—a rather slender argument in favour of its nervous origin.

From the facts that sensory changes are not present in scleroderma, and that section of a peripheral nerve does not cause scleroderma, the author argues that the changes must be produced as a result of irritation of nervous elements rather than of their destruction.

The arguments in favour of the sympathetic being the part of the nervous system involved are many and various: the absence of motor or sensory disturbances; the association, in reported cases, of abnormal pigmentation, of changes in the nails and bones, and of vasomotor and secretory disturbances, are mentioned; but most important is the consideration of the tissues involved in the changes, viz., skin, fat, interstitial tissue, bones, joints, over all of which, the author has reason to believe; the sympathetic exerts a trophic influence.

J. P. MARTIN.

SENSORIMOTOR NEUROLOGY.


Among 25 cases of encephalitis epidemica, Sarbo had 7 in which the onset was purely lethargic, 6 lethargic with psychic disturbances, and 4 lethargic...
with psychic disturbances and chorea. Two cases died in the acute stage, 2 others died within three and a half months, while 6 were considered cured. Of the remaining 15, 10 subsequently presented the 'amyostatic symptom-complex'.

In a short general discussion, Sarbo divides the encephalitic symptoms into five groups: (1) lenticular, (2) myasthenic, (3) cerebellar, (4) trophic, (5) psychic. In the first group he places rigidity, fixed expression, slowness of movement, and pseudo-Parkinsonian tremor. In the second, weakness and tiredness, difficulty in swallowing and chewing; one of his cases had temporarily a myasthenic reaction in some muscles. In the cerebellar group he puts loss of balance in upright posture, retropulsion, and lurching gait; but in a footnote he states that he now believes part of the loss of balance to be due to 'dysfunction of the red-nuclear system'. His fourth group comprises greasiness of the face, changes in the skin generally, and salivation, besides the increase or decrease of fat which sometimes occurs. Of the psychic functions, after the lethargy has passed, the most striking is loss of initiative; perhaps the nocturnal insomnia also falls into this group.

He considers that in the chronic cases the process at work is probably twofold: (1) a sclerosis following the primary acute inflammation, and (2) a continuous chronic inflammation.

J. P. M.


The author describes eight cases, of ages ranging from 1 to 50 years, in which a condition was observed similar to that described by Hofstadt as occurring in 20 children. In these the patients exhibited extreme restlessness directly they were settled down to sleep. In children this took the form of violent movement and grimacing, and even shouting and talking. At other times there was not the slightest sign of any psychic disturbance, and certainly none of mania. On the contrary, there was a certain degree of apathy. The two explanations put forward to account for this are a circulation of toxins and an alteration in a hypothetical sleep centre. Every conceivable remedy had been tried, without the least effect. While some of the cases seemed to show spontaneous cure, the prognosis was most uncertain.

R. G. Gordon.


Of his 39 cases, the author found 27 in the male sex and 12 in the female; 31 occurred in subjects under forty. Although some cases showed a neuropathic history, this was negative in the majority. There did not seem to be any emotional factor constantly present to account for the onset of the Parkinsonian syndrome. A definite acute onset of the encephalitis was
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absent in 8 cases, and the chronicity of the sequel had no relationship to the acuteness of the original disease. There was a definite continuity between the acute attack and the sequel in 28 cases. In one case there seemed to be an interval of a year, though it is not clear that there was ever complete restoration to health. Several associated symptoms not met with in true Parkinson's disease are described as occurring in the cases under observation.

The author is gloomy as to prognosis, though he thinks there is a tendency to spontaneous improvement and even cure. Most remedies have proved ineffectual in his hands, but he thinks that small repeated doses of scopolamine relieve symptoms.

R. G. GORDON.

[112] The amyostatic symptom-complex (Der amyostatische Symptomkomplex und verwandte Zustände).—(1) POLLAK. *Deut. Zeits. f. Nervenhe.*, 1922, lxxiv, 80. (2) JAKOB. Ibid., 47.

The term 'amyostatic symptom-complex' was used by Strümpell in a paper in 1915 as a comprehensive term to include the symptoms of Wilson's disease, of pseudo-sclerosis, and especially of the paralysis-agitans-like condition which occurs as a sequel of epidemic encephalitis—in fact all those symptoms which we attribute to lesions of the extrapyramidal motor system. The term was based on the conception that besides the nervous mechanism which produced (and co-ordinated) the movements of a group of muscles acting on a joint (myomotor), there was another mechanism which innervated and co-ordinated the muscles so as to keep them and the joint at rest (myostatic). It is evident that such a mechanism must be closely associated with the maintenance of normal muscle tone, and so if any part of it breaks down there will occur not only (1) involuntary movements, i.e., inability to maintain steadiness (amyostasis), but also (2) changes in muscle tone and its co-ordination. To all the group symptoms which may be held to arise from such disturbances Strümpell gave the name 'amyostatic symptom-complex', and he attributed it to lesions of the corpus striatum.

Pollak, in opening a discussion on the subject at the Gesellschaft Deutscher Nervenärzte, gave an account of the basal ganglia and their known connections. He treated the matter phylogenetically, onogenetically, and histologically, collating the writing of original workers—Wilson, the Vogts, Spiegel, and others. From the phylogeny several facts can be deduced: (1) That the corpus striatum is probably not directly influenced by the cortex; (2) That its main connection is with the thalamus; (3) That its connection with the hypothalamus is earlier (more elementary) than its thalamic connection (Edinger); and (4) That the putamen and caudate nucleus are essentially one structure. From the embryology we learn that the basal ganglia develop from a point at the base of the lateral wall of the cerebral vesicle; developing from the surface of this vesicle they are related to the cerebral cortex. Coming to the histological structure, Pollak first mentions the different kinds of cells found in the ganglia, and then proceeds to investigate the tracts passing to and from them. The
chief strio-petal connection is with the thalamus—thalamus to putamen; from the putamen fibres course into the globus pallidus, and from there outwards to many parts of the brain—to the homolateral thalamus, hypothalamus, red nucleus, and substantia nigra, and to the contralateral hypothalamus (and hence to the contralateral corpus striatum). The principal secondary connections within the brain would appear to be with the cerebellum and with the nuclei of the pons. The question then arises, By what tract or tracts do impulses from the basal ganglia pass down into the cord? The obvious answer is, By the rubrospinal tract; but Pollak, chiefly on phylogenetic grounds, rejects this, and suggests several minor paths—relays of short fibres from the pontine nuclei, descending fibres from the smaller nuclei in the hypothalamic region, perhaps even the sympathetic tract.

One of the best parts of the paper is that in which the author considers the various influences which act on motor impulses—influences from the anterior and posterior central convolutions, from the frontal lobes, from the corpus striatum, and from the cerebellum—and the points at which these various influences come into association with each other. Here he attributes an important rôle to the red nucleus, and less important parts to the pontine ganglia and the olives.

The paper ends with a warning of the danger of lightly ascribing complex symptoms to lesions of single parts when the whole system is so involved.

The correlation of the amyostatic symptom-complex with pathological findings was dealt with at some length in Jakob's paper. He traced the development of the idea of an extrapyramidal motor system from the assertions of Bonhoeffer and Anton in 1897 that the source of the movements in chorea did not lie within the pyramidal system. In 1911 Mingazzini emphasized the motor function of the lenticular nucleus, and in 1912 Wilson ascribed to it control over muscle tone as well as over involuntary movements. Recently C. and O. Vogt have regarded the 'striatum' and 'pallidum' as the centre for primary automatic movements, the 'pallidum' bringing about the elemental movements of earliest infancy, and later coming under the controlling influence of the 'striatum'. More recently still, however (1921), Stertz has attributed to the lenticular nucleus merely a controlling influence over automatic movements, and supposes the origin of the movements to be in the motor nuclei of the cerebellum.

The first really pathological part of the paper deals with senile chorea, of which the author has examined 6 cases; 5 of them showed pronounced atrophy of the basal ganglia generally, but especially of the caudate nucleus, the small cells of the ganglia being much more affected than the large; the sixth case, which had complications, showed extensive fatty changes in the corpus striatum, including the globus pallidus.

Paralysis agitans presents pathologically a sharp contrast to chorea, because in it the large cells are most affected and 'striatum' and 'pallidum' are about equally involved. Jakob emphasizes the constancy of this finding, and discusses the views of various workers on this subject; he does not attempt to decide whether the degeneration in the 'striatum' or
that in the globus pallidus is the more significant; but he regards part of the changes in the globus pallidus as secondary to those in the putamen.

Wilson's disease and pseudosclerosis are next discussed, and reference is made to Fuchs' experiments with guanidin and with Eck's fistula, intestinal intoxication being regarded as an etiological factor in lenticular degeneration.

Coming to the consideration of athetoid movements, Jakob says "athetosis evidently demands a 'striatum' at least partly capable of function, while progressive degeneration of the 'striatum' and the consequent release of the globus pallidus put a stop to positive motor symptoms by general rigidity".

As regards encephalitis lethargica and its sequelæ, correlation of symptoms and pathological findings is most difficult, owing to the extensive distribution of the lesions, and little attempt to draw conclusions is made. Mention is made of Economo's finding of quite recent patches of inflammation in a chronic case, showing that the active process may continue for years.

Jakob gives particulars of several peculiar cases, and arrives at two conclusions: (1) That if a lesion or degenerative process is limited to the 'striatum', choreic movements develop as a rule, but that under certain conditions, not yet fully understood, athetoid movements may be produced; (2) Very extensive damage to both globi pallidi causes a general rigidity.

Both these papers show the extraordinary difficulties with which the elucidation of the functions of the basal ganglia is surrounded, and neither asserts that the 'amyostatic symptom-complex' has as an entity an anatomical basis.

J. P. Martin.


This is a detailed case report with post-mortem findings, to which is appended a comment upon the group of disorders to which it belongs. A girl of 12 suffering from the disease in question came under the author's observation in July, 1920. The history was that two years previously the parents noticed that her movements were assuming an automatic character. The condition progressed gradually, and there supervened involuntary movements of the extremities which were said to be more violent during sleep. Latterly there had been disturbance of speech and difficulty in deglutition. When she was admitted to hospital in April, 1920, a note was made of general rigidity and choreiform movements. When she was first seen by the author, the striking features of the case were (1) bizarre and grotesque contortions of the trunk and limbs, and (2) great motor agitation.

During the whole of her stay in hospital the patient never for a moment assumed a natural attitude, even in sleep. The abnormal postures varied
somewhat from time to time, and did not lend themselves readily to description. They are illustrated in the text by photographs. It was always possible to manipulate the contorted limbs into other positions, and the author considers that hypotonus rather than hypertonus was a feature of the case. He makes an exception, however, in the case of the arms, which became rigid towards the end.

The involuntary movements approximated more nearly to those seen in chorea than any other recognized type. They were most evident in the limbs, but involved the respiratory muscles, and also the face, in which grimacing and smacking movements of the lips were observed. At first the patient was able to protrude her tongue normally, but later this became impossible. There was considerable dysarthria. Deglutition was normal while she was under observation. Voluntary power was good. There were no defects of sensibility, sphincter control was perfect, and the reflexes were normal. The mental condition was very slightly impaired until the last stages of the illness. Annular pigmentation of the cornea was not present. The liver dullness was notably diminished. The urine reduced Fehling's solution. The blood-sugar was normal. The patient died of pneumonia in August, 1920.

Clinically the case was certainly not one of double athetosis, and in many points it differed from Wilson's disease. It appeared to the author to fit best with the descriptions given by Ziehen, Oppenheim, and Flatau and Sterling of their cases of so-called torsion spasm, a case of which was also described by Thomalla in 1918, with an autopsy recording total bilateral necrosis of the putamen and cirrhosis of the liver.

At the post-mortem in the author's case the most notable findings were multilobular cirrhosis of the liver and an enlarged spleen. The lenticular nuclei appeared greyish-yellow; otherwise the nervous tissues seemed normal to the naked eye.

Histological examination of the brain showed widespread changes, the degree of which varied in different situations, while the general character of the lesions was constant. There were extensive degeneration and destruction of the nerve-cells and of the neurofibrils; but the most striking feature was the increase of glia-cells, which showed every stage of transition between the normal and the so-called Alzheimer cells. The picture corresponded with that described by Alzheimer in pseudo-sclerosis. These changes were most intense in the corpus striatum, especially the head of the caudate nucleus and the putamen, but were also found unmistakably in the thalamus and hypothalamus, in the mid-line of the pons, in the dentate nuclei of the cerebellum, and in the cerebral cortex. There were nowhere any gross foci of necrosis or cysts.

The author draws particular attention to the diffuse character of the lesions in his case. He asks whether it is the lesions of the corpus striatum which are responsible for the principal symptoms. He mentions other cases of Wilson's disease complicated by a lesion of the dentate nucleus, and emphasizes the necessity for thorough examination of all areas of the brain in such cases, lest we be led through faults of omission to attribute to the corpus striatum alone functions which really belong to a system of
neurones in which it plays a part. He considers it probable that Wilson's disease, pseudo-sclerosis, and torsion spasm are all different manifestations of the same disease, various clinical syndromes which depend upon one pathology. In relation to the particular clinical features of torsion spasm, he suggests that this may be correlated with the fact that this syndrome always makes its appearance in childhood. Possibly the nature of the motor symptoms may depend upon the age of the brain. As an instance in point he mentions the well-known fact that post-hemiplegic athetosis is rarely seen to develop unless the hemiplegia dates from infancy.

The paper is clearly written, and the bibliography is up to date. The microphotographs of the brain are inadequate, but this defect is made good by the lucidity of the text.

C. P. S.

[114] An essay on the shaking palsy, by James Parkinson, M.D., Member of the Royal College of Surgeons; with a bibliographic note thereon.—A. J. OSTHEIMER, Arch. of Neurol. and Psychiat., 1922, vii, 681.

A reprint of Parkinson's historic classic, of which only five copies are known to exist.

R. M. S.

[115] Heredity in epilepsy.—CHARLES W. BURR. Arch. of Neurol. and Psychiat., 1922, vii, 721.

Burr's investigations were concerned solely with cases of so-called idiopathic epilepsy; those in which there was evidence of gross cerebral disease were excluded. It was found that only 34 parents were known to be affected, and hence direct inheritance did not appear important. On the other hand, the frequency of insanity, crime, chorea, alcoholism, and epilepsy in relatives pointed towards congenital instability resulting from abnormality in the germ-cell or sperm-cell. From a consideration of all the data, Burr concludes that it is safe to assume that the effect of heredity is rarely direct; that usually it is indirect and general, not specific. In other words, a predisposition to nervous or mental disease is inherited; the resulting specific disease depends on external causes—it is environmental in the broadest meaning of the word.

R. M. S.


Marie and Béhague described a syndrome of disorientation in space met with in cases of deep lesions of the frontal lobe. This occurred without other signs of disturbance of nervous or vestibular functions. Some patients were quite unable to direct any movements, others only failed in the dark, and others found difficulty only when executing special movements. To test slight degrees of disorientation these authors devised the
following method. The patient was taught to distinguish the four sides of a room by some simple means such as the side of the table, the side of the door, of the bed, of the window; his eyes were then bandaged, and he was turned first in one direction, then in the other, made to walk round in a circle several times, then stopped and asked to name which of the walls he was facing. In healthy subjects a mistake was never made, but in cases suffering from deep lesions of the frontal lobes hesitation and error were noted. This experiment was repeated by the author in a case of a lesion of one frontal lobe, apparently cured, and disorientation as described above was discovered. The author next tried the test on a large variety of healthy subjects, taking particular care to avoid possible additional indications of light, heat, and noise, and he found a high percentage of errors, which in his opinion makes the test valueless as a pathological symptom of any sort, and still more so as a pathognomonic sign of lesions of the frontal lobe.

R. G. Gordon.


The study of mutism in infants is very difficult, because the neurological examination of an infant needs limitless patience, because it is difficult to test the hearing in infancy, and because the infant cannot read or write. The symptoms generally show much more variation than in adult life. The causes are simple. Cerebral paralysis or Little's disease of all varieties, biopathic failure of development, and encephalitis from various causes, of which hereditary syphilis is far the commonest, are the chief. The author divides mutisms of infancy thus:

\[
\begin{align*}
\text{Mutism without intellectual deficiency} & : \\
\quad \text{Delayed development.} & \\
\quad \text{Deaf-mutism.} & \\
\quad \text{Mutism due to auditory defects.} & \\
\quad \text{Mutism where hearing is perfect.} & \\
\quad \text{Mutism due to aphasia.} & \\
\quad \qquad \text{"""" anarthria.} & \\
\quad \text{Mutism due to idiocy.} & \\
\quad \qquad \text{"""" prenatal failure of development (microcephaly).} & \\
\quad \text{Mutism with intellectual deficiency} & \\
\quad \qquad \text{"""" prenatal paralysis.} & \\
\quad \text{"""" postnatal early paralysis, or aphasia without paralysis.} & \\
\quad \text{"""" delayed postnatal aphasia without paralysis.} &
\end{align*}
\]

The first group can be diagnosed where there is obvious cause for delayed development or a family history of late talking, and where there is no auditory defect; but the delay should not be present after the age of 2½, or at most 3. The author does not consider adenoids of much importance in this connection, though any interference with hearing certainly is, as
described below. Deaf-mutism is often confused with idiocy. If a galvanic current is passed through the two mastoids the absence of vertigo points to deaf-mutism. The presence of vertigo with a weak current (1 to 2 ma.) in a child who shows no signs of hearing noises points to idiocy. The true deaf-mute shows no signs of paralysis, and can express himself intelligently by gestures. The noise he does make in attempting to speak is without intonation. The prognosis of true deaf-mutism is hopeless; but the author describes a third group in which mutism persists only until the error in the auditory apparatus can be corrected. Such children suffering from middle-ear disease, etc., are often mistaken for idiots, but a careful examination will demonstrate their intelligence and their deafness. The reflex of the external auditory meatus (ticklishness) may be useful in this connection, as it is absent or reduced in deaf children. The mutism of cretins is said to be due to the myxœdematous changes in the middle-ear tissues.

In the fourth group, in which there is no defect of hearing, German authors have thought that this is a form of mild imbecility, while others have supposed it to be a form of sensory or motor aphasia in all cases. Others admit an idiopathic alalia, but regard it as a want of development, due to prenatal causes, of 'an intellectual and affective disposition to talk'. The author thinks that this idiopathic alalia is due to an arrest of development, not of the cortical centres, but of the afferent or peripheral nervous apparatus, so that owing to tone deafness the auditory word centres do not get sufficient stimulation and are delayed in their development. He distinguishes this type of mutism by the following features: (1) Congenital, thereby differing from aphasic mutism; (2) The hearing being apparently normal, but actually often showing bilateral defects of tone perception; (3) Mutism present, but capability of repeating short words at command; mimicry and 'internal language' unaffected; (4) No dysarthria; (5) Sufficient intelligence, though peculiarities may be found; (6) High-grade capacity for education, so that this type is not met with in adult life, but in cured cases it is found that though the intonation of speech is efficient these individuals are taciturn, and sometimes are ungrammatical.

The fifth group, mutism due to aphasia, should be confined to those who have lost the power of employing verbal symbols in thought. Hence all cases in this group are acquired, and due to lesions occurring after the commencement of language development. Several cases of congenital aphasia have been described, but the author thinks that they require reconsideration. Cases of mutism due to aphasia generally show some paralytic symptoms or epilepsy as well, and though there is usually no psychic defect the child is 'unstable'. Transitory aphasias occur after epileptic fits or febrile diseases, but permanent aphasia may occur after trauma or after intracranial disease, and in the latter case may be the only persistent sequel. If the lesion is unilateral such aphasics are capable of re-education, but not if it is bilateral. The sixth group includes those who are mute because of anarthria or dysarthria. Such cases occur in infantile cerebral paralysis and in infantile bulbar paralysis and pseudo-bulbar paralysis. Usually the infantile cerebral paralysis is accompanied by epilepsy and idiocy, but in rare cases this is not so.
The second division, of mutes with defective intelligence, includes the idiots, who show all varieties of mutisms. In congenital idiots there is often a certain capacity for improvement, especially if other complicating conditions can be removed. A more serious condition is that accompanying early prenatal or postnatal cerebral paralysis. Much obscurity surrounds the question of the mutism of idiots, and some authors hold that an idiot cannot talk because he lacks the capacity to form internal speech, others because he lacks the capacity to articulate words. As a matter of fact, although an idiot does not understand much more than he can express, the aphasic idiot seems less intelligent than he really is, and often in the course of development comes to be able to do more than might be expected. The author also describes a delayed aphasia and intellectual inferiority without paralysis. In these cases the progress is gradual and the patient may be left with a few short words. The lesions are bilateral, cortical, and usually syphilitic. It is progressive, but not fatal. The aphasia and the dementia progress together, and one does not depend on the other.

As to treatment of mutism, the author thinks that education is of great value if begun early, and that unilateral lesions are of no great importance in infancy. Antispecific treatment does very little good.

R. G. Gordon.


According to Lhermitte and Cornil, heteresthesia is exceptionally rare, for they have only seen one example of this interesting phenomenon since it was described by Graham Brown in 1920. (See Brit. Jour. Neurol. and Psychopathol., 1920, i, 54.) Their patient, a sergeant, age 25, was wounded on Sept. 2, 1918, during an attack. The bullet entered the left supraspinous fossa 4 cm. from the 2nd dorsal vertebra, and made its exit in the right infraspinous fossa 4 cm. from the internal border of the scapula. The soldier was lying on his face when hit, and at once became paralysed in both lower limbs. Four days later he experienced burning pains in the lower extremities, which occurred from very slight contact, such as pressure of the bedclothes. Eventually they disappeared, to be replaced six weeks later by a new type of sensory disorder, described as lightning pains resembling electric shocks, and excited solely by flexion of the head. After the lapse of six months they too disappeared. Paraplegia was at first complete, but rapidly diminished, and by Jan. 20, 1919, the patient could walk almost without assistance.

When examined on Feb. 26, 1919, there was no inco-ordination, ataxia, or dysmetria: muscle tonus and electrical reactions were normal. A very slight degree of motor weakness in the lower limbs was still present. The application of a soft object to the legs gave rise to a sensation of heat; otherwise sensory functions were normal.

A second examination on Oct. 15, 1920—that is, more than two years after the date of injury—showed an almost complete disappearance of the motor disability; but there were now present new and distinctive sensory
changes. Of his own accord the patient drew attention to certain areas on the right thigh where painful sensations were evoked by contact with some object, or even part of the clothing. A cold stimulus also evoked pain, but of a less intense character. These abnormal sensations never occurred spontaneously, and the boundaries of the areas on which they were experienced could be readily and precisely mapped out by drawing the point of a pencil over the skin, while the patient at the same time indicated with one finger the site of the disagreeable sensation. These zones of heteræsthesia corresponded exactly to the distribution of the 1st, 2nd, and 3rd lumbar, and part of the 2nd sacral sensory nerve roots. In spite of the existence of heteræsthesia, the sensations conditioned by pin-prick, pressure, and temperature remained normal on the affected area; there was, however, a slightly raised threshold for the appreciation of two points of a compass. Localization and deep sensibility were not affected. Contrary to the experience of Graham Brown, the areas of heteræsthesia were very persistent, and were still present when the patient passed out of observation.

In their discussion of this phenomenon the authors state that they consider it certain that heteræsthesia cannot be caused by a lesion of the posterior nerve roots, nor are they able to accept Graham Brown's theory of an alteration of a central mechanism co-ordinating the different spinal segments. They suggest as an alternative hypothesis that the phenomenon is related to an unequal implication of the intraspinal sensory fibres, which are known to retain their radicular grouping in their passage through the spinal cord. Such an explanation seems to square best with the known anatomical facts.

R. M. S.

[119] The nervous symptoms associated with local malformations of the spinal column, especially those of the last lumbar vertebra (Les syndromes nerveux liés aux hétéromorphismes régionaux du rachis, en particulier à ceux de la vertèbre présacrée).—ANDRÉ ROCCAVILLA. Revue neurol., 1921, xxxvii, 39.

MALFORMATIONS of the vertebrae are often found at those levels of the spinal column at which its architecture changes from one form to another, i.e., at the junction of occiput and atlas and at the cervico-thoracic, thoraco-lumbar, lumbo-sacral, and sacro-coccygeal junctions. Any of these may give rise to symptoms. The commonest malformations are those in which either the vertebra above the point of junction possesses characteristics of that below it, or vice versa. The best known of these is that in which the 7th cervical vertebra possesses thoracic characteristics—the so-called cervical rib.

The writer here concerns himself chiefly with the symptoms due to sacralization of the last lumbar vertebra. Anatomically the degree of sacralization varies from that in which the transverse processes of the vertebra are but slightly enlarged, to the extreme in which they extend as far as, and articulate with, the ilia. Supervening inflammatory changes may result in bony union, the sacrum then appearing to be made up of six segments with five pairs of neural foramina. The bony canal for the 5th
lumbar root on either side may be so narrow as to result in compression of its fibres. In addition, secondary changes, mechanical and inflammatory, may cause symptoms referable to the 4th lumbar and 1st sacral roots.

The symptoms may be divided into three groups: (1) Lumbar or lumbo-sacral pain with stiffness of the back. The pain sometimes radiates into the groins and lower abdomen. The objective signs are few—points de Valleix, flattening of the lumbar curve, and, if the malformation is unilateral, a little scoliosis. (2) Diffuse pains over the sacrum and in the buttocks, sometimes accompanied by stiffness, sometimes by weakness in the muscles of these parts. In addition, there may be paroxysms of pain radiating down the thighs and legs as far as the toes. Lasègue's sign is often positive, especially after a paroxysm. (3) Pain of sciatic distribution which corresponds to that described by Dejerine under the name of radicular sciatica. With this are found minor defects of motor and sensory function which follow root distribution, together with a diminution of the tendon-jerks. Tenderness of the nerve-trunks to pressure may be present. The symptoms may be on one or both sides.

Symptoms are not commonly caused by the simple presence of the malformation. As a rule determining factors are present, of which the commonest are the after-effects of trauma. Other causes may be tubercle, syphilis, or non-specific inflammatory processes. The author points out that the malformation may exist without giving rise to any symptoms at all, and issues a warning against the danger of ascribing to this condition, when discovered by the x ray, all pains which may occur in the neighbourhood. The diagnosis is each case must be considered on its clinical merits.

For most cases the best treatment is manipulation by means of passive and active exercises, together with heat in various forms, and electro- and radio-therapy. In some cases of trauma, and in the presence of active inflammatory changes, tuberculous or otherwise, fixation by splints may be required. Finally, in selected instances the surgeon may be asked to clear a new pathway for the compressed nerve roots.

The paper is well illustrated with radiograms and sensory charts, and a number of references to the literature are given.

C. P. S.


The author quotes a phrase of Raymond's to the effect that the causes of so-called idiopathic sciatica remain to be discovered. He believes that in at least 80 per cent of the cases the trouble is due to vertebral disease which may be detected by the x rays. The 5th lumbar vertebra is most often the seat of the trouble. Leaving on one side traumatic lesions and tuberculous caries, he proceeds to discuss:—

1. Ossification of the ligaments, or pseudo-sacralization. This has nothing to do with true sacralization, which is a congenital anomaly
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and non-progressive. Pseudo-sacralization results from calcification of the iliolumbar and sacro-iliac ligaments secondary to chronic inflammatory changes of rheumatic nature.

2. Chronic lumbar arthritis. In these cases the inflammatory changes are not confined to the ligaments, but affect the vertebrae themselves. From this may result a sliding forward of the 5th lumbar upon the 1st sacral, or a condition in which the 5th lumbar is forced downwards so that in the x-ray its transverse processes appear superimposed upon the posterior aspect of the ilium. The author believes that this condition has often been mistaken for sacralization. Although arthritic changes may supervene upon true sacralization, the two conditions are primarily distinct.

3. True sacralization. The aspects of this condition have not yet been sufficiently established upon an anatomical basis by post-mortem dissection. Mere enlargement of the transverse processes of the 5th lumbar is of no account. True sacralization, in the author's opinion, exists only when there is fusion of the lateral elements of the 5th lumbar with the auricular surfaces of the sacrum and ilium. This is usually accompanied by malformations of the sacrum itself, and these congenital anomalies may lead to other static changes affecting the poise of the whole vertebral column.

The title of this paper is misleading in so far as the clinical aspects of the various conditions mentioned are not described in detail.

C. P. S.


The case is reported of a young fisherman who came under observation during military service on account of pains and weakness in the legs experienced on the march or in the gymnasium. Eight years previously he had had an attack of paraplegia, which came on suddenly with pains in the affected limbs. From this he recovered gradually, but during convalescence he noticed an increasing hypertrophy of the muscles of his legs, which after a short time reached the size observed by the author. He had been able to carry on his work as a fisherman, but his lower limbs fatigued rapidly, and he experienced pains in the calves on prolonged exertion. The calf muscles were much hypertrophied, their power being rather less than normal. On palpation they appeared to be of a natural consistency. The electric reactions were quantitatively diminished. No other physical abnormalities were discovered. A piece of muscle was excised during life and examined microscopically. The muscle fibres showed abnormal variation in size, some being considerably hypertrophied. There was no increase in connective tissue, and no fatty degeneration.

The author reviews briefly the stories of 20 cases of true (pathological) muscular hypertrophy which he has collected from the literature. It is remarkable that in no less than 5 of these the condition appeared after an attack of typhoid fever. In some of the others there was a history of general infection or local injury, in some no apparent cause. In several
instances there is mention of pain, and in some there was definite cutaneous anaesthesia.

The best account of this interesting condition previously given is by Léri, who divides the cases into those in which the hypertrophy depends upon vascular disturbances, and those in which it appears to have an independent origin. For the latter, he accepts Talma’s definition of hyperplastic muscular dystrophy.

Krabbe believes that in the majority of cases the primary cause is a neuritis with subsequent exaggeration of the normal process of repair. He compares as similar instances cheloids, amputation neuromata, and hypertrophic osteitis.

C. P. S

Psychopathology.

PSYCHONEUROSES AND PSYCHOSES.


This paper gives the Jungian interpretation of some of the phenomena of the war neuroses in answer to Dr. G. H. Fitzgerald’s exposition from the Freudian standpoint. Young cites two cases of morbid anxiety arising in men who had done prolonged service without receiving any definite trauma, and shows how the patients were reacting against a narrow and one-sided development of their personality. The conflict has to do with the acceptance of and adjustment to the compensating function. The ‘persona’ is essentially narcissistic and maintains a struggle with the unconscious, so that there is little libido available for external relationships; yet the menace from the unconscious has its counterpart in the affairs of everyday life. As the patient cannot find a point of application for his effort against the intangible enemy, his effort only serves to drain him of energy; the effect of which is expressed in his various symptoms. The inimical forces must be brought into and accepted in consciousness, for only after the acceptance of them and the abandoning of the old one-sided ideal can a new, more satisfactory orientation be reached.

Young concludes by saying that, in his opinion, the stress and strain of war simply serve to unmask a tendency to neurosis, and that the so-called traumatic moments are either artefacts or unessential secondary occurrences.

Alfred Carver.


The case reported appeared at first to be one of hysteria due to shock. On analysis it became evident that an x-ray investigation in hospital, the significance of which had been reinforced by important experiences in