CURRENT LITERATURE

VISCERAL NEUROLOGY.

1. VEGETATIVE NERVOUS SYSTEM.

Albert Kuntz and O. V. Batson. EXPERIMENTAL OBSERVATIONS ON THE HISTOGENESIS OF THE SYMPATHETIC TRUNKS IN THE CHICK. [Journ. of Comp. Neurol., 1920, xxxii, December, 335.]

As the findings of Kuntz that cells of medullary origin play an important part in the development of the sympathetic nervous system have been doubted by others, he and Batson have tried by crucial experiments to eliminate the neural crest and the dorsal portion of the neural tube before the spinal ganglia have become differentiated: if this could be done, and it were still found that the embryo continues to develop without spinal ganglia and dorsal nerve-roots, the remaining portion of the neural tube would be the only source from which cells of nervous origin could migrate along the paths of the spinal nerves. If in such embryos the primordia of the sympathetic trunks should arise they would of necessity arise from cells which migrate from the neural tube along the paths of the ventral roots of the spinal nerves. In these experiments chick embryos were subjected to operation at the close of the second day of incubation (48 hours), at which time the spinal ganglia are not yet differentiated: the dorsal portion of the cerebrospinal nervous system was destroyed by electrolysis throughout a limited portion of the trunk region. By this means the writers obtained conclusive evidence that cells of medullary origin, which advance peripherally along the ventral roots of the spinal nerves, enter the primordia of the sympathetic trunks. The spinal ganglia are not excluded as a source from which cells may enter the primordia of the sympathetic trunks under normal conditions; however, these primordia may arise from cells derived from the neural tube only, at least when cells which have their origin in the spinal ganglia (or neural crest) are excluded.

"As observed above, the primordia of the ganglia of the sympathetic trunks may be approximately of normal size in segments in which the spinal ganglia and dorsal nerve-roots are absent, but the remnant of the neural tube is relatively large. On the other hand, these primordia are small or entirely absent in segments in which the remnant of the neural tube is small and represents only the most ventral portion of the central nervous system, even though ventral nerve-roots are present. These facts suggest that the cells which normally give rise to the ganglia of the sympathetic trunks are derived largely from those portions of the walls of the neural tube which give rise to the lateral

cell-columns. Theoretical considerations also favor this interpretation; however, we do not feel that the evidence at hand warrants a definite conclusion on this point." [Leonard J. Kidd, London, England.]

Kuntz, A. THE DEVELOPMENT OF THE SYMPATHETIC NERVOUS SYSTEM IN MAN. [Jour. Comp. Neur., October 15, 1920.]

From a study of embryological material, Kuntz shows so far as ontogeny can show that the primordia of the sympathetic trunks arise in human embryos of about 5 mm. They first show as a small group of cells lying along the dorso-lateral aspects of the aorta in the lower thoracic and upper abdominal region. Six mm. embryos show them from the lower cervical to the sacral region. When the embryo reaches a length of 10 to 11 mm., the primordia are then noted in the upper cervical region as well. These primordia arise from cells of cerebro-spinal origin advancing peripherally along the dorsal and ventral roots of the spinal nerves. The vagal sympathetic plexuses arise in similar fashion and advance along the vagi. The more distal enteric plexuses arise from cells derived from the sympathetic supply in the lower trunk region. The ciliary ganglion is derived from the semilunar ganglion *viâ* the ophthalmic nerve. The primitive cells of the sphenopalatine ganglion advance along the greater superficial petrosal nerve, originating also from the semilunar ganglion *viâ* the maxillary nerve. The otic ganglion arise at the growing extremity of the lesser superficial petrosal nerve. The petrosal ganglion also receives cells of trigeminal origin *viâ* the mandibular nerve. The submaxillary and sublingual ganglia arise on the lingual nerve primarily from cells of trigeminal origin. They probably receive some cells of facial origin *viâ* the *chorda tympani*. The smaller ganglia on the glosso-pharyngeal nerve in the posterior portion of the tongue arise from cells which advance into the tongue along the glosso-pharyngeal fibres. The cells which give rise to sympathetic neurones are derived from both cerebro-spinal ganglia and the neural tube. Not all of those cells actually migrate as such, as many arise by mitotic division of the migrant cell along the paths of migration and in the primordia of the sympathetic nervous system.

Eyster, J. A. E., Middleton, W. S. AURICULO-VENTRICULAR HEART-BLOCK IN CHILDREN. [Amer. Jour. Dis. Child., February, 1920.]

Twenty cases of heart-block in children, nearly all of which were definitely or probably of congenital origin or occurred during the course of severe and usually fatal diphtheria, are here reported upon. A personal case of partial auriculo-ventricular dissociation is added. This developed in a child of 2, apparently in connection with an acute nose and throat infection in which the cultures showed only *Staphylococcus pyogenes aureus*. The child, who was kept under observation for two years, developed normally. At the time of writing the cardiac condi-

tion was that of a well compensated mitral lesion associated with a 2 to 1 auriculo-ventricular block, with a ventricular rate between fifty and sixty.

G. C. Bolten. THE VASQ-VAGAL ATTACKS OF GOWERS. [*Psychiatrische en Neurologische Bladen*, 1920, No. 5-6, 341.]

The vaso-vagal attacks described by Gowers are not very common. Bolten agrees with Gowers that they are closely related to epilepsy. These patients are almost always very neuropathic; their attacks occur mostly suddenly, at times apparently wholly spontaneously, at others in connection with exposure to cold or cold-stimuli. There is violent palpitation, the patient is tormented with a very severe and troublesome feeling of oppression, cold, "dead" extremities, paraesthesiae in hands and feet, and a great retardation of the line of thought. During the attack the patient is not unconscious, yet he feels that he cannot move his limbs; there is great inhibition of all psychical processes, and speech is impossible on account of a complete stiffness of the jaws, and in all four limbs he feels great tingling as if he had been sleeping on his arm or leg. In spite of the preservation of consciousness he can think only very imperfectly; he can assimilate all kinds of external impressions, but all the associational processes in his brain are strongly inhibited. So severe is the oppressed feeling that it brings him into a state of great anxiety, so strong that, even although he may have had many previous attacks, he feels always that he will die in this attack (apparently death has never occurred in an attack). Bolten records a case very graphically, and discusses the nature of these vaso-vagal attacks at length. He holds that they are very closely related to angina pectoris vasomotoria and to paroxysmal tachycardia. He strenuously denies that they are the sequel of a simple peripheral disturbance (vascular contraction or spasm), but holds that they are due to a more complex constitution-anomaly, viz., a vasomotor insufficiency. In these three types of attacks — which he regards as merely mutual variants — the usual considerable pulse-acceleration he looks on as a compensation-symptom; by increasing the number of ventricular contractions — which are in part true extra-systoles — the organism attempts to compensate for the harmful effect of the low blood-pressure. Bolten concludes that the vaso-vagal attacks of Gowers do not form an independent clinical entity, but are to be conceived of as a non-essential variant of paroxysmal tachycardia and of "pseudo-angina" (angina pectoris vasomotoria). [Leonard J. Kidd, London, England.]

F. Depisch. PATHOLOGY OF THE VEGETATIVE NERVOUS SYSTEM. [*Wien. Archiv. f. innere Medizin*, March, 1920, I, I.]

A unilateral vegetative nervous system syndrome is here described by the author. He tries to show by it, in comparing with others, that the

fibres of the vegetative system become crossed on their way to the periphery in a somewhat analogous manner to the somatic sensori-motor systems. The primary lesions causing such unilateral symptoms are usually some localized hemorrhage in the medulla oblongata, entailing bulbar paralysis. The unilateral symptoms included a higher local temperature, the unilateral action of drugs, pilomotor reflex variations, sweat differences, etc.

Andre-Thomas. THE PILOMOTOR REFLEX. [Rev. Neurol., 1920, 27, 1139.]

André-Thomas describes the receptive field, appropriate stimuli, and the characters of the response in this reflex under various conditions.

In the normal subject the pilomotor muscles may be excited directly by mechanical stimuli or reflexly. As a reflex it may be obtained by various stimuli applied to the skin, such as cold, friction, and the electric current. To unilateral stimuli the response is unilateral and may spread to the whole of the stimulated half of the body, or it may be restricted to one or more segmental areas. Certain cutaneous areas are particularly receptive, the nape of the neck and shoulders and the lower part of the axilla. When elicited from the neck the response is descending, and stronger stimuli are necessary to produce it over the head and neck. On the limbs, the extensor surfaces give the response more readily than the flexor surfaces. Bilateral stimuli produce a bilateral response.

In lesions of the spinal cord the response is exaggerated immediately below the upper level of the lesion. In total divisions of the cord, there are two forms of response: one above the level of the section, the "réflexe encéphalique," and one below, the "réflexe spinale."

The spinal reflex in total divisions of the cord makes its appearance when the isolated portion of the cord emerges from the spinal shock—that is, simultaneously with the mass reflex. It is elicited by the same stimuli as this reflex, namely, passive movements of the legs, etc. In these circumstances, the factors governing the distribution of the spinal reflex are the same as those governing that of the reflex sweating described by Head and Riddoch—that is, the distribution of the thoraco-lumbar white rami.

In partial lesions and in various diseases of the spinal cord, the response varies and is often difficult of interpretation. It is normal in poliomyelitis. In syringomyelia, variations in the response follow the distribution of vasomotor and trophic disturbances. In peripheral nerve lesions, the reflex response is abolished over the area of sensory changes, but, as Trotter and Davies showed, a local response to stimulation persists.

The cerebral reflex, that is, the response above the level of a cord lesion, varies greatly in facility and distribution. It descends to the level of the sympathetic distribution of the isolated portion of the cord.

In the normal subject the reflex is elicited by peripheral stimuli and also by various emotional states. The quality of the stimulus in both cases is similar and is strongly affective. In the former case, the sensations aroused by stimulation are of the affective variety and are unpleasant. Hence when the skin is anaesthetic no response can be obtained from it. The emotions producing the reflex are also unpleasant in quality. Fright and horror are the most adequate stimuli, but any profound emotion, whatever its quality, may be sufficient to produce it. The reflex of emotional origin is bilateral, that due to peripheral stimulation may be unilateral. The pilomotor reflex may therefore be regarded as an affective reflex. [F. M. Walshe in *Medical Science*.]

Rizzo, C. THE CILIARY GANGLION AND INACTIVITY OF THE PUPIL.
[Arch. biol. norm. e. patol. 1920, 74, 1.]

The much discussed problem of the pupillary reflexes is here taken up. With regard to the seat and nature of the lesion underlying the Argyll-Robertson pupil (alone, or associated with inactivity to accommodation), he quotes Marina and others as having asserted that in this condition the cells of the ciliary ganglion are reduced in number, and show chromatolysis, and that the myelin sheaths of the ciliary nerves are degenerated. Other observers, notably André-Thomas, have been unable to confirm this from the investigation of three cases. The author agrees with André-Thomas. He examined the ganglia from nine normal subjects, from eight paretics and one tabetic with Argyll-Robertson pupils, from two paretics with totally inactive pupils, and from one with defective reactions to both light and accommodation, and from one parietic with normal pupils. He failed to find any noteworthy degeneration of the nerve-cells, though a few cells showed some chromatolysis and swelling of the cytoplasm which were obviously acute and recent lesions attributable to the intercurrent illness which caused death. The roots of the ciliary ganglion, the short ciliary nerves, the nerve plexus of the muscle of Brucke, and the nerve filaments of the iris and cornea were all normal in every case. An exudate of lymphocytes and plasma cells was constant in the ganglia of paretics.

Fraikin. SOLAR PLEXUS SIGN WITH ABDOMINAL NEUROPATHIES.
[Paris Méd., February 14, 1920, 10 No. 7.]

Spontaneous pain or tenderness in the solar plexus is a not infrequent result of disturbance of the plexus. It is a sign that the circulation is hampered in or outside of the viscera, or the nerves are suffering from toxic action or from traction from sagging organs. This solar sign may serve to differentiate a psychogenic conversion pain, from a somatic visceral lesion.

Livierato, P. E. THE ABDOMINOCARDIAC REFLEX. [Grèce Méd., Athens, Dec., 1920. J. A. M. A.]

Livierato reports that his own and others' extensive experience has confirmed the instructive increase in the size of the right ventricle by mechanical stimulation of the abdominal nerves. He taps along the median line in the epigastrium, the patient recumbent, the abdominal walls relaxed. The left ventricle shows no change, and there is no change in the healthy, but when the heart elasticity is below par, the right ventricle enlarges from this tapping, and it is thus a very useful sign of the condition of tonus of the myocardium, revealing insufficiency before it has become manifest in any other way. This reflex was always positive in patients convalescing from various diseases. Livierato reports further that when the right heart shows spontaneous dilation on changing from the recumbent to the erect posture, this is likewise a sign of hypotonicity of the myocardium. The dilation subsides spontaneously as the subject reclines again. This spontaneous increase of the heart area upward and to the right on change from the horizontal to the erect posture is always a sign of pronounced hypotonicity of the heart muscle.

Ant. Hutter. A CASE OF MYASTHENIA GRAVIS. [Psychiatrische en Neurologische Bladen, 1920, Nos. 5-6, 352, (6 figs.).]

The patient, a man of twenty-five, was a typical case of myasthenia gravis, with attacks of fatigue, extending over two and a half years, interrupted by remissions. In the attacks there were also bulbar symptoms and the myasthenic reaction. Death took place from a pulmonary lesion complicated with rapid exhaustion of respiratory and auxiliary-respiratory muscles. Necropsy showed microscopically a normal brain and a strikingly small pituitary. In the thymus-region there was a small fatty lobe; the vocal cords showed a mother-of-pearl glistening. Thyroid gland rather small; tongue appears fatty. A very fatty omentum. Spleen rather large, greenish, and shows plainly on section its follicle-pattern; its pulp is swollen. Liver rather light-colored. Kidney very vascular, with strongly developed fat-capsules. Right adrenal very vascular; left very flabby, its cortex appears too thin. Prostate rather large with definite middle lobe. Microscopically, the central nervous system was normal; so also were the thyroid, adrenals, testis, pituitary, spleen, kidney, stomach, pancreas, and prostate. Tongue-muscle fatty. Rich lymphoid infiltrations in the muscle-tissue of the masseter, quadriceps, deltoid, and diaphragm, but not in the psoas. The biceps muscle showed degeneration, local lack of cross-striation, and marked increase of nuclei. The thymus-like fragment contained no true thymus tissue; the thymus was thus absent. The liver-parenchyma is definitely fatty. [Leonard J. Kidd, London, England.]

Marie, Bouttier and Bertrand. PROGRESSIVE MYASTHENIA. [Annales de Med., 1920, 10, No. 3.]

Here the postmortem findings are given from a myasthenia gravis case which had been treated for some time advantageously by suprarenal extracts. The nervous system was found apparently intact; the classic picture of bulbospinal myasthenia without any appreciable lesions in the bulbar nuclei. Small lymphoid nodules were scattered throughout the suprarenals and the muscles, which the author states is evidence as to the relation of the adrenals to the disease. The improvement under adrenalin had persisted for six months when the woman died in a few days after the onset of pulmonary edema [excess of vagotonia.—Ed.]

Brun, R. THE ORIGIN AND SYMPTOMATOLOGY OF LUMBAGO. [Schweiz. Arch. f. Neurol. u. Psychiat., 1920, 7, 63.]

Brun emphasizes that we have little accurate knowledge of the causes or nature of lumbago. At present, and largely on theoretical grounds, three clinical forms are recognized.

(1) *A myogenic form* in which the muscle is regarded as the primary seat of the disease. Rheumatism and trauma are generally regarded as the factors responsible for this form. Workmen who work half-stripped and subsequently become chilled acquire a rheumatic affection which remains latent until some strain to the lumbar muscles unmasks the condition by the sudden appearance of pain. (Primary rheumatic origin of traumatic lumbago.) Another view is that strain may make the lumbar muscles a "locus minoris resistentiae" upon which a subsequent rheumatic affection fastens. The importance of actual injury to the muscles, such as tearing, bruising, or haemorrhage, as factors in the production of lumbago is generally deprecated.

(2) *Osteo- or arthro-genic forms.* Torsion at the lumbar spinal joints has been suggested as a cause, but is probably exceedingly rare.

(3) *Neurogenic form.* Lumbago has been described as a true neuralgia of the lumbar nerves, as a toxic neuritis, or as due to injury of these nerves.

In the second section of the paper, Brun analyses twelve personally observed chronic cases of lumbago, all of which presented objective physical signs. From the study of these he finds that trivial injuries may give rise to severe lumbar pain ("Lumbalgie") of long duration, and that these cases are more numerous than is commonly believed; that careful clinical examination reveals in the majority of such cases definite objective physical signs in the affected region; these physical signs include unilateral changes in the lumbar and sacral portions of erector spinae—wasting, hardening, tenderness to pressure, changes of electrical excitability such as diminished faradic response and qualitatively altered galvanic response, and in two instances the presence of transverse furrows in the muscle due to localized spasm.

There were also symptoms referable to the lumbar vertebrae, such as tenderness on pressure over the spines, scoliosis convex to the normal side in three out of four observed cases, and also reflex rigidity of the lumbar spine from pain. Finally, nerve-root symptoms were frequently observed; pain and sensory loss in the distribution of the posterior cutaneous branches of the lumbar and sacral nerves and of the ilio-inguinal nerve, and pain along the distribution of the sciatic nerve. The pain and cutaneous hyper-aesthesia in the lumbar region he attributes to this nerve irritation. From these observations he concludes that chronic lumbago is essentially a perineuritis and neuritis of the posterior cutaneous branches of the lumbar nerves, usually of traumatic origin, and but rarely toxi-infective. The sciatic nerve may be secondarily involved. The condition is frequently complicated by neurotic symptoms. [F. M. Walshe in *Medical Science*.]

2. ENDOCRINOPATHIES.

O'Day, J. C. TOXIC GOITRE. [*N. Y. Med. J.*, cxi., 1920, p. 6 et seq.]

In his chapter on toxic goitre O'Day will not admit the existence of a pathological secretion. Toxic goitre is toxic because of an over secretion of the gland's normal product.

He objects to the term Exophthalmic Goitre. He complains that text-book descriptions of the disease leave the impression that it is called exophthalmic goitre because the exophthalmus is a constant symptom. To quote, "It is no more exophthalmic goitre than it is death-dealing goitre for just as it may result in death may it also result in an exophthalmus."

Toxic goitre he found to be unlike all other varieties. The difference, both microscopically and macroscopically, was constant in several hundred specimens examined—that difference depended entirely upon a preponderance of the acini—"a preponderance that admits of little or no intervening stroma."

He holds the belief that because of this the gland is inevitably over-secretive and with so many follicles at work at and during the same time he conceives the idea that proper adulteration is prevented.

This is the way he puts it: "The secretion is thinner, being destitute of those albuminous elements which govern the consistency of that from the normal gland, and, according to the splendid work of Kendal at the Mayo Clinic, contains more thyroxin. We come to regard it as more volatile, more pungent, and more penetrating. It might be argued that the product of a toxic goitre differs so much in these particulars from that of the normal gland that its toxicity depends on the difference. It is not unreasonable to suppose that because of its volatile and penetrating character it is carried away much more rapidly by the lymphatics, but this is not found to be so. We are convinced that the whole trouble results from the actual hyperactivity where every acini has been