

to inhibit rather than to stimulate heat production. There was an apparent fallacy, too, in the statement in regard to atropin. Atropin does not paralyze the vaso-motor centres. The chief action of atropine is upon the motor rather than upon the sensory system.

Dr. GRAY referred to a case of leptomeningitis above the fissure of Sylvius, in which the temperature was at no time above the normal.

Dr. LLOYD referred to a case of subnormal temperature after injury of the cortical motor region.

Dr. OTT explained that he had stated simply that destructive excision of the centres caused a rise of temperature; he had not stated whether those centres were inhibitory or not. In regard to atropin, he had stated that atropin inhibited the vaso-motor reflexes. The subnormal temperature in Dr. Lloyd's case would be explained by irritation of that region of the cortex, by the removal of which temperature was increased. It was not usual to observe the temperature of patients suffering with nervous disease, and variations might be overlooked. The fact that lesions about the fissure of Rolando were not always followed by an elevation of temperature proved nothing one way or the other. Paralysis also did not always result.

Dr. E. D. FISHER, of New York, then read a paper entitled:

CLINICAL REPORT OF CASES OF EPILEPSY FOLLOWING
CEREBRAL HEMIPLEGIA.

Of late much has been written in regard to cerebral hemiplegia in children, or cerebrale kinderlähmung, both in this country, in England, and on the continent. In recording the following cases I would, however, lay particular stress on the epileptic seizures associated with the disease. The picture of these cases has been so often drawn that it has become a familiar one. The hemiplegia unilateral and bilateral, as it is sometimes termed, coming on at birth, or in the first few years of life, is similar in most respects to that of adult hemiplegia, with the addi-

tional symptoms of more or less interference with the growth of the parts and a more marked condition of contracture. These cases are to be found in every almshouse or home for the feeble-minded. There is usually marked contracture of the upper extremity, the fingers closed in the palm and resisting in many cases all efforts to draw them out. The lower extremity is also contracted, the gastrocnemius being involved, causing the patient to walk on the ball of the foot with the heel drawn up; the face is but slightly affected, at least in the latest stages of the disease. There is some interference with the growth of the parts, and while this was present in all of the following cases, in only one was it marked, affording at once a differential diagnosis from infantile spinal paralysis. In reference to this point I would refer to three cases reported by H. Quinke in the *Deutches Archiv. f. Klin. Med.*, bd. xlii., h. 5, of Glioma involving the central convolutions, in which, along with the paralysis, marked and rapid wasting was present, leading the author to believe that a trophic centre must exist in close proximity to the motor centres. The author does not consider it possible that the wasting could be due to inactivity, as is the case usually, or as the result of descending degeneration of the lateral columns, as it occurred so soon after the paralysis, and microscopical examination revealed no changes in the anterior cornua.

We have also present in these cases, imbecility or idiocy, and epilepsy, conditions not existing in the spinal lesion. All these symptoms, with the exception of the last two, may exist wherever the motor tract is interrupted in its course, whether in the cortex, sub-cortical region, basal ganglia, capsule, crura, pons, medulla, or cord. It rarely occurs in other than the cortical and sub-cortical regions, and if accompanied by imbecility and epilepsy, can only be referred to the cortex.

Wallenberg, in *Westphal's Archiv. f. Psychiatrie*, bd. xix., h. 2, reports a typical case of spastic cerebral hemiplegia of childhood, in which the lesion involved the cornua with descending degeneration and as was to be expected there were no mental symptoms or epileptic seiz-

ures. In regard to the epileptic seizures, there is no rule as to their commencement on the paralyzed or on the non-affected side, indeed they more often resembled in their onset idiopathic epilepsy, and if a warning were present, consisted in an epigastric aura, vertigo, &c. As Jacksonian epilepsy may resemble idiopathic epilepsy in its onset and course, so may the idiopathic form simulate in every particular the Jacksonian.

When the hemiplegia is congenital, the cause is probably in the majority of cases, meningeal hemorrhage, induced either through injury in utero or at time of birth. Porencephalus is also a factor in these cases. We find evidence of the hemorrhage in the form of cysts or adhesions, between the dura and the pia mater, with sclerosis and atrophy of the convolution, accompanied by secondary degeneration extending into the cord. Both hemispheres may be involved, and very commonly over the convex surface. This would sustain Gowers' statement that in congenital cases the hemiplegia is more often of the double form than in the acquired cases.

In these latter cases, occurring usually at the age of two or three years, Gowers believes that thrombosis of the longitudinal sinus is a very frequent cause, with thrombosis of the veins entering the sinus resulting in capillary hemorrhages, atrophy and sclerosis of the convolution with secondary degeneration. Embolism is more likely to occur in cases of more advanced years, or to follow the exanthemata rheumatism, &c.

Poli-encephalitis and intra-cerebral hemorrhage are probably not as frequent causes as those previously mentioned.

In an interesting paper read before this Association in July of last year, Dr. P. C. Knapp enters very fully into its etiology, and also gives a complete bibliography of the subject. I would also refer to Dr. Osler's lectures on the subject in the Phila. Medical News. I would differ with Dr. Knapp in reference to the interference with the growth of the members affected, agreeing with Prof. Hensch that as compared with infantile spinal paralysis it is comparatively

small, the wasting of the muscles being almost exclusively one of disuse, as seen in those cases with permanent contraction; whereas, when athetoid or choreiform movements were present, the muscles were often well developed.

In three cases affected with congenital choreiform movements either constantly present or increased by voluntary action, I found the muscles well developed, and although muscular power seemed somewhat decreased, it appeared to be more awkwardness in movement than actual loss of power. These cases represent, to my mind, congenital injury to the brain substance with a multiple sclerosis, not entirely destroying the cortex or involving all the motor fibres and leading thus to irregular motor impulses. These cases are similar to those of the congenital type of spastic hemiplegia, the lesion, however, being more diffuse in distribution and not so destructive in character. I would refer here to an article on intention tremor by Dr. Stephan in the *Archiv. für Psychiatrie und Nerven Krankheiten* bd. xviii., h. 2.

My cases are mostly congenital, or occurring between the ages of one and three. In one case, however, resembling in every particular the spastic hemiplegia of childhood, with marked contractions, equino varus, exaggerated reflexes and epileptic seizures, the paralysis occurred at 23 years of age, followed one month later by epileptic attacks, always commencing on the paralyzed side.

In regard to longevity, in most instances the average of 20 years, as given by Henoeh, was far exceeded. The presence in greater or less degree of mental weakness is always marked; this is accounted for by the occurrence of the disease in the early stage of cerebral development; epilepsy did not occur in many of these cases for some time following the paralysis.

The contractures decreased in some cases in the course of years, so that in one instance the hand became a fairly useful member. Relaxation during sleep of the spastic condition was not observed. The extreme spasticity caused in voluntary acts was illustrated in cases 1 and 18. In the latter the whole of the affected side, including the

muscles of the face were thrown in violent spasm on the slightest attempt to rise or even extend a limb; in the former, flexion of the arm caused flexion at the wrist, while extension produced excessively violent extension of the wrist, this was beyond the control of the patient and was only overcome by great force.

As remarked in the beginning of this paper my special interest in these cases was the associated epilepsy, and it has been my study to inquire into the nature of the attacks, their onset and course, in order if possible to see whether any difference existed between them and those of idiopathic epilepsy, inferring if none can be found, or at least if no well-founded and persistently acting difference exist, that in all probability, the seat of the disease in idiopathic epilepsy must be in the same region and probably of like nature as in spastic hemiplegia, although not so marked in its changes. Perhaps the most distinctive characteristic of epilepsy is the loss of consciousness, coming on suddenly. In Jacksonian epilepsy the consciousness is often involved, but here we have a case of convulsions due to mechanical irritation corresponding to the physiological experiments of the laboratory. The intelligence of the patient is also much less apt to be affected than in petit mal, even when the latter is not associated with grand mal.

In cases of epileptic seizures from tumors of the brain, the convulsions may be due to a general pressure or disturbance of cerebral circulation and its attacks are not localized but commence bilaterally, and are preceded by loss of consciousness.

A statement of Luciani, sustained by many autopsies, may be of interest here, that while a paralysis may be strictly localized as to the cerebral lesion, it is not possible to make more than an approximate localization in epileptic seizures. This would the more readily occur where the tumor was of large size or of such nature, or so situated as to produce a general disturbance of the circulation. Such cases are found in the epilepsy of spastic hemiplegia, and although the lesion is unilateral I find that the attacks are more often of this character than that of the Jacksonian type.

This then, approaches the form of idiopathic epilepsy where the marked feature is the disturbance of consciousness which precedes the convulsion, and is indeed the most serious phase of the case, as here the higher centres, the latest evolved, and rerepresenting all the lower centres, according to Hughling Jackson, are affected, and we have the resulting dementia, or at least feeble mindedness so often present. When, however, the lesion can be localized as due to a circumscribed pachymeningitis following insolation for instance, the attack commencing with a localized tremor or tingling preceding the loss of consciousness, although the convulsion may become universal and of great severity, still such patients may live on for years with but slight if any mental impairment.

My cases as I have said, resemble the idiopathic epilepsy rather than the Jacksonian form, contrary to the expectation from the localization of the lesion; the explanation probably lies however in the consideration that the disease in question is one of early life, during the most active period of cerebral development, and is always accompanied by interference with this development and the growth of the cells of the cortex.

We are justified by analogy to infer that in idiopathic epilepsy the seat of the disease lies in the cortex cells, and consists in a primary change in their nutrition. That it is primary and not induced by any disturbance of circulation following the convulsions would seem to be clinically proven by the fact that in many cases of petit mal, when convulsion can be excluded the mental disturbances are the most marked.

In looking over my cases of idiopathic epilepsy I find mental weakness almost invariably present. As Luciani has well stated, "the central organ for epilepsy upon which its pathology substantially rests is always the complex of the motor centres of the cortex, whether the irritation arises directly or reflexly." Thus, any internal organ or any portion of the periphery may become an epileptogenous zone for the origin of an epileptic seizure, but this does not result unless the cortex is in a condition to

respond to the irritation, in other words, unless we have a morbid condition, in which case the equilibrium or stability of the cells is easily disturbed.

Thus it is that heredity becomes as important a factor in prognosis as in other mental disorders. In conclusion, it is noticeable in these cases that the great majority are of the hemiplegic type, only one side being involved, and in several instances the paralysis was but slightly marked, so that only a careful examination revealed the fact, that there was some slight interference with the growth of one side, with exaggerated patellar reflex of that side.

This would lead us to a more careful examination of all cases of epilepsy occurring in early life, and probably many now referred to hereditary or unknown causes can be more properly classed as due to cerebral injury in utero, at birth, or during the first few years of life. These facts ascertained however, do not improve the prognosis, as we may see from the review of the cases detailed. In regard to treatment I have but little to say. Trephining after changes have occurred in the cerebral and spinal structure would seem useless. The removal of some portion of the cortex where the epileptic seizures are excessive and unilateral in character, at least in the beginning, has proved in some cases beneficial. The bromides appear to act as beneficially as in ordinary epilepsy.

CASE I.—Geo. T., aet. 25, had severe fall of two stories when a child. Paralyzed in right arm; lower extremity unaffected; arm and hand held in position of flexion, but can be voluntarily extended. Seizures occur about once a month. Patient very unintelligent; reflexes somewhat increased, especially of right side. Warning of present tremor commencing in right side.

CASE II.—Henry H., aet. 17, paralyzed at age of two years on right side; patellar reflex exaggerated in affected side; marked contractures; gait spastic; right limbs smaller than left; speech slow, patient unintelligent; left internal rectus weak. Attacks occur every three or four days, commencing on paralyzed side.

CASE III.—James J., aet. 56, paralyzed on right side at six years of age ; one year after had epileptic seizures. Attacks occur every four months ; increased by drinking ; no warning. Patient presents usual spastic condition of these cases.

CASE IV.—Henry D., 43; hemiplegia at 13 years of age; no history of rheumatism, no cardiac lesion. Four years later first attack. Patient has been under observation for the past ten years at the almshouse. Attacks come on without warning ; generally falls suddenly backwards ; attacks vary from eight or ten a month to five in one night ; any excitement, joy or sorrow, will bring one on ; speech unintelligible and general intelligence growing less.

CASE V.—Geo. Y., aet. 25, spastic hemiplegia from 3 years of age. Attacks about once a week ; no warning ; not limited to or beginning on paralyzed side ; dementia.

CASE VI.—James L., 55, right leg amputated six years ago, following injury. Three months later epileptic seizures; now about three in a month. Memory good.

CASE VII.—Arthur C., 50, paralyzed on right side when seven years old. Attacks very frequently ; not beginning or limited to one side ; no warning ; speech unintelligible ; dementia.

CASE VIII.—John K., 19, right hemiplegia following fall at two years of age. Attacks nine to fourteen a month; generally no warning ; dementia. Reflexes exaggerated on both sides, most marked on the right.

CASE IX.—Wm. L., 26, hemiplegia resulting from a fall at age of three. Attacks five to nine a month ; generally warning of dizziness ; reflexes but slightly exaggerated ; dementia.

CASE X.—John H., 24, congenital paralysis of right side. First seizure at age of thirteen; no warning; generally about one a month ; feeble-minded.

CASE XI.—Martin K., 28, congenital left hemiplegia with epilepsy. Attacks six to nine a month ; warning of

dizziness. Attacks not limited to or commencing unilaterally ; weak-minded.

CASE XII.—Wm. H., aet. 26, hemiplegia of right side following blow on left side of head. Arm and leg much contracted. Equino varus ; seizures two or three a month ; warning dizziness ; attacks general ; speech slow, scarcely intelligible ; partial dementia.

CASE XIII.—Patrick C., aet. 49, family history negative. Patient gives history of syphilis and alcoholism, no cardiac lesion. Ten years ago had attack of vertigo and nausea ; unconscious for two months, and gradual loss of power on left side. One year later had first epileptic seizure, which now occur about every three months ; no warning, never bites the tongue. Attacks general, heretofore commencing on the paralyzed side.

CASE XIV.—Frank R., aet. 22, perfectly well up to nine years of age, when he fell, breaking bridge of nose. Left leg dragged slightly in walking ; both patellar reflexes somewhat exaggerated. Patient says that the attacks begin by twitching in the left leg which passes to the right leg, and then he loses consciousness. At outset cries out, "No ! no !" as if in fear. This can be checked. About one year ago, while attacks were very frequent for ten weeks had spasmodic attacks in one arm (?) in which hammer-like strokes were kept up by the hour. The patient semi-conscious and moaning as from fear. Attacks of late under large doses of bromide much reduced ; patient is unable to read well, is slow of speech, feeble minded, but possesses considerable talent in drawing.

CASE XV.—John H., aet. 32. Hemiplegia following scarlet fever at age of three. No history of otitis ; hearing good ; spastic condition excessive ; marked tremor on voluntary action ; athetoid movements of fingers ; equino varus. First fit at fourteen years of age ; generally preceded by dizziness. Patient fairly intelligent.

CASE XVI.—Henry Z., aet. 30. Congenital right hemiplegia ; marked flexion elbow and wrist ; gait markedly

spastic; reflexes exaggerated. Attacks begin on right side and always more severe on that side; no warning; imbecility; athetoid movements of fingers of right hand.

CASE XVII.—John H., aet. 19. Right spastic hemiplegia from a fall at two years of age. Spastic condition marked; constant tremor of right eyelid; patellar reflex not exaggerated. Attacks usually at 5 A.M.; no warning and generally occurring about once a week. Patient unable to read or write.

CASE XVIII.—James B., aet. 48. Left hemiplegia at one year of age, with convulsions. Left leg smaller and shorter than right; left hand tightly flexed until age of fifteen years. Can now open it; arm well developed, showing but slight shortening. Excessive tremor or spasm of left side of face on talking. Patellar reflexes greatly exaggerated on both sides; gait that of spastic paraplegia; no epileptic seizures since childhood; intelligence fair; can read and write.

CASE XIX.—John V., aet. 35. Patient has had epileptic seizures since two years of age. Attack commences with feeling of numbness on left side. On examination slight paresis, with exaggerated patellar reflex on that side. Patient able to read and write a little.

CASE XX.—Mary B., aet. 47. Specific history, left hemiplegia three years ago; reflexes exaggerated; has had two seizures since in hospital; denies any previous attacks; no warning, feels suddenly both sides equally affected. Paralysis more marked for several days following attack. Intelligence impaired.

CASE XXI.—Mary T., aet. 29. Right hemiplegia with convulsions following small pox; intention tremor; reflexes exaggerated; seizures very frequent, beginning with choking sensation; uncertain whether they begin on paralyzed side; patient imbecile.

CASE XXII.—Maria M., aet. 32. Right hemiplegia with convulsions, between two and three years of age. Walk markedly spastic; attacks about once a month, generally no warning. Imbecile.

CASE XXIII.—Ida C., aet. 32. Left spastic hemiplegia with convulsions at six years of age. Attacks about once a month; petit mal daily; feeling of dizziness precedes attack. Patient unable to read intelligently.

CASE XXIV.—Ada C., aet. 34, left spastic hemiplegia, with epilepsy since childhood; attacks about once a month at time of menstruation, warning in nature of peculiar feeling; does not commence unilaterally; feeble minded.

CASE XXV.—Mary C., aet. 19, congenital right hemiplegia; arm flexed at elbow and fingers in hand; impossible to overcome contracture by force; leg flexed at knee and foot in position of equino varus; patellar reflex exaggerated on both sides, especially on right side; right side much shorter than left and muscles atrophied; seizures mild but very frequent and always commencing on paralyzed side; speech scarcely intelligible; dementia.

CASE XXVI.—Louise W., 32, congenital left hemiplegia; contractures very marked; muscles atrophic and considerable shortening of upper and lower extremities; head unsymmetrical. Attacks first came on at two years of age; warning, as of sensation of choking; commences usually on left side. No attacks for past three years; speech intelligible, but patient very stupid, unable to read or count.

CASE XXVII.—Michael S., aet. 31, attacks followed measles at age of two; did not return until twenty-seven years of age; patient says, was never paralyzed, but on examination find paresis of right side, flatness of right side of face; reflexes exaggerated; patient says that he has always used left hand more than right. Although he went to school until fourteen years of age, can scarcely read; patient ascribes this to defective eyesight. Attacks of late becoming more frequent, about one a month; no warning and commence bilaterally.

CASE XXVIII.—A.C., aet. 2½, (the mother, a primipara at 42 years of age, pregnancy not being suspected, had by advice of physicians, bandaged herself rather tightly and taken rather violent exercise to remove the supposed flatulence;

delivery however, natural and easy. The child made no attempt to walk or talk till two years of age). About one year ago had epileptic seizure, commencing on right side and becoming general; remained more marked on that side. Has had two similar attacks since. On examination, child large and well-developed; gait spastic, pushing one foot before the other and walking on the ball of the foot; reflexes exaggerated, especially on the right side; child able to say a few words and beginning to show desire for certain things; expression of face, idiotic.

DISCUSSION ON DR. FISHER'S PAPER.

Dr. KNAPP suggested the existence of trophic cell centres in the cortex. He had seen cases of infantile hemiplegia in which the atrophy was as great as that in many cases of spinal paralysis.

Dr. G. E. WALTON described a local epileptic attack. The patient had been healthy to within a few weeks when the attacks had commenced. The convulsion involved the left side of the face; there was slight dilatation of the pupils, and the head was turned over the left shoulder. The hands were not affected, and there was no loss of consciousness, but the patient talked thick after the attack. This seemed to be due to difficult pronation rather than to aphasia. The speaker referred to the question of operation. In monkeys the centre for turning the head was located in the first and second frontal convolution near the ascending frontal. The centre for the face and mouth would be lower down.

Dr. FISHER did not find the wasting infantile hemiplegia so marked as that in Dr. Knapp's cases. There was always some wasting, but he thought not more than would be explained by disuse. Another proof that the epilepsy of this condition was not of the Jacksonian type and dependent upon local lesion was the fact that there was no aura or only an epigastric aura in these cases. According to Luciani epileptic seizures had but little value for localization compared with paralysis.