

DR. F. A. KLEINHANS, Milwaukee, Wis., said that he has been a sufferer from psoriasis for 18 years, and has tried practically everything in the line of treatment both internal and external, the x -rays alone excepted. He is firmly convinced that psoriasis is not of parasitic or microbic origin, but is a cutaneous manifestation, due to faulty metabolism, in fact a sort of auto-intoxication. In sections of most recent psoriatic lesions excised from his own person, he has found a parakeratosis, i. e., a thickening of the horny layer and dilatation of the vessels of the corium and round cell infiltration surrounding the dilated vessels and the sweat coils. Among other methods he has tried the hypodermic use of arsenic (as Fowler's solution) and found it very painful. At times he can rid himself very readily of the lesions in the course of a week or two by taking 5 grains of bicarbonate of potash 3 times daily; at other times this remedy has no effect. Sometimes Fowler's solution proves beneficial and again it fails, or even aggravates the condition. Thyroid extract has also been tried without any effect. He believes that there is no one remedy that can be depended on in all cases at all times.

DR. WILLIAM L. BAUM, Chicago, said he has always regarded psoriasis as a dermato-neurosis. In the first place, there is a distinct hereditary tendency to the disease, as it can be traced from generation to generation, following practically the same type as does a psychosis. The mere fact that so many remedies have been recommended by different authorities indicates that the opportune moment effected the cure rather than the remedy. He has frequently observed exacerbations of the disease after violent emotional disturbances. The eruption may disappear rapidly when the patient is placed under a different environment. This does not depend so much on the climate, as the disease is observed in both warm and cold climates. In the majority of cases, the patients should be made to understand clearly that the disease is incurable, although in some instances it is limited to a single outbreak. Vaccination and injections of antitoxin may be exciting factors in causing an outbreak. In Cook County Hospital, Chicago, about 3,200 patients have been given injections of antitoxin and a decided number of the patients injected have returned with psoriatic efflorescences attributed to the antitoxin. Still, any other form of irritation might have the same effect. In over 70 per cent. of the patients who returned, Dr. Baum was able to get a history of psoriasis either in the parents or grandparents, or in some collateral branch of the family. In the treatment of psoriasis, the possibility of neurotic origin should always be remembered.

DR. DAVID LIEBERTHAL, Chicago, said that all are agreed as to the difficulties connected with the management of psoriasis. It is difficult to have a patient carry out such strict diet as Dr. Bulkley demands. Dr. Lieberthal has under his care a patient with psoriasis who years ago was treated by Dr. Bulkley and freed from the disease through the diet treatment exclusively. But the patient remarked that he would rather keep his skin affection than undergo such a diet treatment again. It can not be denied, however, that regulating the diet of certain patients with systemic derangements will bring about good results. Dr. Lieberthal has under his care a patient who for 16 years was not relieved of his affection in spite of having had the benefit of the best dermatologic care here and abroad. The patient's urine was loaded with urates and uric acid and after his diet was accordingly modified, the skin cleared up and remained so until his death, 18 months later. It is important to see the patients at short intervals in order to observe the lesions and to control the internal administration of drugs. External treatment should be employed with due diligence and if chrysarobin is applied, it should not be allowed to spread to the normal skin, otherwise it is apt to produce a dermatitis, on the basis of which new psoriatic lesions may develop. Arsenic should be administered rather in older cases of limited area. If Fowler's solution is used a fresh preparation should be secured as the older becomes inert. Dr. Lieberthal said that he uses the x -ray in psoriasis, but only in the severe inveterate cases. Any treatment employed should be carried out until the last traces of the disease have disappeared.

DR. L. DUNCAN BULKLEY, New York City, said that he has used carbolic acid in psoriasis, and knows what it will do in certain cases. He thinks it entirely erroneous to regard psoriasis as incurable. He also disagrees with those who claim that it is impossible to get patients to live on a vegetable diet. He had one psoriatic patient who has been a vegetarian for 10 years, and a number who have abstained from animal food for from 3 to 4 years. He has seen psoriatic outbreaks following a shock or mental strain. In the treatment of psoriasis, patients should be kept under observation for a long time after the eruption itself has actually disappeared. The same thing is done in syphilis and in gout. Psoriasis can be cured in a certain proportion of cases. Those not cured are the ones in whom the basic cause of the eruption can not be discovered.

UNILATERAL ASCENDING PARALYSIS AND UNILATERAL DESCENDING PARALYSIS.

THEIR CLINICAL VARIETIES AND THEIR PATHOLOGIC
CAUSES.*

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In December, 1899, I presented a paper to the Philadelphia Neurological Society in which I called attention to a new clinical type which I designated as unilateral progressive ascending paralysis.¹ Somewhat similar cases have since been described under such terms as progressively developing hemiplegia, chronic progressive hemiplegia, and ascending unilateral paralysis. Unilateral ascending paralysis is probably the simplest and, therefore, the best designation for the symptom-complex.

Since the publication of the first contributions on this type of paralysis, the subject has attracted some attention, but not perhaps as much as its importance warrants. Probably not a few cases of progressively ascending hemiplegia or hemiparesis have failed of record because of the slowness of development of the affection and the overlooking of the essential feature of upward progression. Some cases have, no doubt, been passed by as lacking in interest, or as probable instances of focal disease not worthy of elaborate report.

Practical medicine is advanced by directing attention to clinical types or entities which are peculiar in their method of development, their symptomatology or their pathologic causes. One point which has been brought out during the brief history of the existence of progressively ascending hemiplegia is that unilaterality and upward progression of the paralysis are exhibited by affections differing in their pathology.

A close study of the recorded cases of unilateral ascending paralysis shows that this clinical type in a more or less pure form may occur, 1, as the result of primary degeneration of the pyramidal tracts to which may be added other degenerative lesions; 2, as the early stage of disseminated sclerosis; 3, as the form assumed by unilateral amyotrophic sclerosis; 4, as the order of progression in unilateral paralysis agitans; 5, as the expression

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1. Jour. Nerv. and Men. Dis., April, 1900.

of a focal lesion either cerebral or spinal; 6, as a clinical type in cerebrospinal syphilis; and as a peripheral or a hysterical affection.

In my description of the first case (rather of the first two cases of unilateral ascending paralysis, for brief account was given of a second case seen many years before the publication of the paper), I suggested that the disease probably represented a new clinical type due to slowly increasing degeneration of the pyramidal fasciculi or of the cerebral motor neuron systems.

The cases which have been recorded by others as well as by myself, since attention was first called by me to the clinical type of ascending hemiplegia, even those cases which have been regarded as instances of pyramidal degeneration, have varied somewhat in their clinical features. In some, for example, spasticity has been present from an early period; in others it has been absent or has been but slightly marked. The reflexes, usually exaggerated, have in one or two instances been diminished or only moderately marked; the wasting usually present but moderate has varied in amount in different cases. As the literature of this affection is still meager, I shall take occasion to give a résumé of all the cases so far published, unless, indeed, some reported case has escaped my investigations.

Before doing this, however, I desire to direct attention to unilateral descending paralysis, which is to be regarded as simply the same type reversed. Unilateral ascending paralysis due to degeneration of the pyramidal tracts, or to this and some added condition of degeneration, is the more common form of progressively developing hemiplegia, but an affection essentially the same sometimes takes a reversed order so far as the limbs or as the face and limbs are concerned, giving us a unilateral descending paralysis or a progressively descending hemiplegia. Through the courtesy of two of my professional colleagues in Philadelphia, Dr. David Riesman and Dr. William Pickett, I shall have the opportunity to present in the concluding portion of this paper two cases of this descending type, the one an example of disseminated sclerosis taking at first the unilateral descending and systemic form, and the other probably an instance of nearly pure pyramidal degeneration.

That the suggestion of a new clinical type is sometimes fruitful in the development of diagnosis is illustrated by the brief history of progressively developing hemiplegia. The report of two or three cases of unilateral ascending paralysis of unusual form has called attention to the question of ascending and descending unilateral types of certain well-known diseases, such as disseminated sclerosis, paralysis agitans and amyotrophic lateral sclerosis. Variations in the symptomatology of these reported cases have also awakened interest as to the probably differing pathology of some of the cases recorded.

After summarizing in the briefest manner possible the cases of unilateral ascending paralysis hitherto published, and after some discussion of the different forms of disease of the nervous system which may present a progressively developing hemiplegia, ascending or descending, I shall record one new case of unilateral ascending paralysis and one new case of unilateral descending paralysis, describing also the case referred to me by Dr. Riesman, when considering the unilateral ascending and the unilateral descending types of disseminated sclerosis.

CASE 1.—A man, aged 52, about two years before coming under observation,¹ began to show signs of weakness in the right lower extremity. Weakness in the arm appeared eighteen months after the impairment in the lower extremity was first noticed. This soon became more and more evident, and was accompanied by the tendency to carry the arm raised against the body and flexed at the elbow.

When first examined the paresis in the upper extremity, although easily determined, had not nearly reached the degree of impairment observed in the leg. The right side of the face was also slightly paretic. Wasting was distinct in the right lower extremity, the measurements showing a difference of one and a half inches for the thighs and five-eighths of an inch for the calves. The various movements of the right leg were distinctly weaker than those of the left, but were nowhere absolutely abolished; similarly all the movements of the right arm were distinctly impaired. Faradic contractility was retained. The affected limbs were not spastic nor contracted. Careful examination showed retention of all forms of sensation. The tendon and muscle phenomena on the right side were all somewhat exaggerated. Knee-jerk was plus on the left side, but considerably more exaggerated on the right. Patellar clonus and slight ankle clonus were present on the right, but not on the left. The plantar reflex was normal on the left; but on the right, while the Babinski response was not present, downward flexion of the toes was distinctly less marked than on the left.

CASE 2.—A woman, aged 43, first noticed weakness in her left leg while pregnant with her last child, three years before coming under observation.¹ A few months later the left arm became paretic. The reflexes were much increased on the affected side. The patient remained under observation for many months, the paresis of the leg and arm slowly increasing. Neither leg nor arm was contracted, and sensibility was preserved. The patient complained at times of pains in the limbs and twitching in the leg and arm.

This woman was under my care more than 20 years ago and had previously been a patient of the late Dr. E. C. Seguin, who believed that there was a cerebral lesion causing secondary changes in the crossed pyramidal fasciculus. The view taken by me was that the alterations in the pyramidal tracts were of the nature of a lateral sclerosis, but were primary and not secondary to any cerebral lesion. I have learned that the patient was alive about fifteen years after she was first seen by me and had become entirely unable to walk. Just how she was affected at this time was not ascertained, except that she had become triplegic, the affection having evidently passed over to the lower extremity on the side opposite to that on which it had started.

CASE 3.—A man, aged 41, four years before coming under the notice of the reporter,² began to have a sense of weakness in the left lower limb in walking. This weakness gradually increased, and in about a year the left upper limb began also to be weak. The patient was positive that a year had elapsed after the impairment in power in the lower limb before he felt any weakness in the left upper extremity. He had never had any symptoms of an apoplectic seizure.

This showed that the movements of the left lower limb were very spastic, but not in the least ataxic. The toes of the left foot scraped the ground, and this foot was inverted in walking. The left knee-jerk was much more marked than the right, which was, however, prompt. Ankle clonus and the Babinski response were present on the left, but not on the right. Sensation was normal. The left upper limb was weaker than the right; it was also spastic, but the weakness and spasticity were less than in the lower extremity. The left upper limb was held slightly flexed at the elbow and against the body, but no contractures were present anywhere. Speech was normal, but the mouth could not be drawn up as well on the left side as on the right. The tongue went slightly to the left on protrusion. Ophthalmoscopic examination showed white atrophy of the left optic nerve. Wasting which was present was like that occurring in hemiplegia.

CASE 4.—A youth, aged 19, about four years before coming

2. Spiller, William G.: Philadelphia Med. Jour., Feb. 9, 1901.

under observation³ began to drag his right foot; it tended to cross in front of the left, and he wore off the sole of his right shoe at the toe. Two years later he had diplopia, which lasted for five months. The right leg at this time was somewhat smaller than the left. About three and a half years after the right foot first began to drag, which was about five months before coming under observation, weakness was noticed in the right arm.

Examination showed that when the patient walked the right leg was rigid and the toe dragged. The movements of the right leg could be performed, but were distinctly weaker than those of the left. The right leg and arm were both somewhat atrophied. The adductor pollicis muscle of the right hand was not so large or firm as that of the left. The muscles everywhere responded to the faradic current; the tendon reflexes on the right were all exaggerated, and a well marked Babinski reflex was present on this side. The reflexes on the left, while marked, were much less active than those on the right. Plantar stimulation caused slight extension of the great toe and flexion of the others. Right-sided facial paresis was present. The muscles of the right side of the throat were also paretic, and the voice had a distinct nasal twang. Nystagmus and paresis of the left inferior rectus, and pallor of the temporal halves of the discs were other symptoms. All forms of sensibility were everywhere preserved. The patient showed a slight Romberg symptom, and at times while standing or walking, a tendency to lose his balance. The case was regarded as one of disseminated sclerosis, beginning as unilateral ascending paralysis.

CASE 5.—A man, aged 60 at the time of his death, about eight years before had some paralysis of the right extremities.⁴ At this time knee-jerk and biceps-jerk were increased on the affected side. Four years before his death hospital notes showed some loss of power in both the right leg and right arm; and all the muscle and tendon phenomena on the right side were exaggerated, patellar clonus and ankle clonus both being present; also the speech was slightly affected, and sensory impairment and astereognosis were absent.

Notes made between two and three weeks before death showed that he was markedly paralyzed in the right leg, and to a less extent in the right arm; also that he was paralyzed in the left leg, but not as seriously as the right. The paralyzed limbs were spastic, and the tendon and muscle phenomena were all exaggerated. He had ankle clonus on both sides, and patellar clonus and the Babinski response on the right. Sensory phenomena were absent. The face was not paralyzed. Contractures and wasting were absent. During two or three years before his death he was treated for cardiovascular symptoms, including attacks of angina.

Postmortem showed advanced disease of the heart, vessels and kidneys, the case being an extreme instance of arteriosclerosis. Chronic pleurisy and tuberculosis with cavity formations were present. Microscopic examination showed intense and long standing degeneration of the right crossed and left direct pyramidal tracts, the degeneration extending into the pons, but not into the left cerebral peduncle; also comparatively recent degeneration of the left crossed and the right direct pyramidal tracts, traced by the method of Marchi into the lower part of the right internal capsule. No lesions, degenerative or focal, were found elsewhere in the brain or spinal cord.

CASE 6.—A young woman, aged 18 at the time of first coming under observation,⁵ was noticed to show slight clumsiness and awkwardness in using the right hand and foot shortly before the age of fourteen. Gradually the weakness of the right lower and upper extremities increased, this from description being more marked proportionately in the former than in the latter. When between seventeen and a half and eighteen years old a slight swelling of the right half of the thyroid was noticed.

Examination showed no abnormality of cutaneous and muscular sensibility nor of the special senses. Pupillary reac-

tions, movements of the ocular muscles and the fundus oculi were all normal. The sphincters were also normal and the patient was mentally sound. The reporter gives in careful detail a description of the musculature and of the movements performed or incapable of proper performance on the right half of the body. Summarized, this indicated marked loss of power and atrophy in both the right upper and lower extremities, considerably greater in their distal than in their proximal portions. The affected limbs were neither spastic nor ataxic, but gave rather the appearance of flaccidity or flabbiness. The paralysis and atrophy were not localized to special groups of muscles. Marked quantitative change to the faradic current was present, being especially noticeable in the muscles which showed the greatest wasting, these being the hand muscles, the anterior tibial and the peroneal. All the deep reflexes were greatly exaggerated on the right side. The Babinski extensor response was typically present, but there was only an indication of ankle clonus. Some paresis of the muscles supplied by the seventh nerve and marked loss of power in the motor distribution of the fifth nerve were present.

CASE 7.—A woman, aged 50, was seen in consultation in October, 1905.⁶ In August, 1904, she first discovered some weakness of the right lower extremity, although it may have been present in mild degree before. The weakness and awkwardness in using the limb very gradually increased until about one or two months before coming under observation, since which time the impairment had increased more rapidly. The patient had suffered no pain in the extremity, in the back or in the head, and had no symptoms of cerebral disease. She had occasional headaches which seemed to be of the migraine type, and now and then had a slight vertigo, but this was probably due either to the condition of her stomach or to some arteriosclerosis.

Examination of the right lower extremity showed general paresis of moderate degree in the entire limb; that is, all movements were impaired in force without definite localized palsy of any group of muscles. The movements of flexion, extension, abduction and adduction of the foot and also intermediate movements were distinctly impaired as compared with those of the left foot, but the reduction in power extended to the entire limb, although it appeared to be more marked distally. The right knee-jerk was greatly exaggerated and patellar clonus could be elicited. Persistent ankle clonus and also a decided Babinski response were present on the right. Front tap was present and marked on the same side and the muscle jerks were also plus on this side. On the left the knee-jerk was somewhat above the usual norm, as were also the muscle jerks, but patellar clonus and front tap were absent. The plantar response on this side was irregular. Careful examination was made for sensory disorders, with absolutely negative results. Examinations seemed to show some slight impairment of the upper extremity, but so slight that it was hardly fair to record it as present.

CASE 8.—A young man, aged 23, complained that for six months his left leg had been growing gradually weaker. He would sometimes trip because he could not clear the ground well with the tip of his left foot. At times also he could not use the left arm well. His condition for about one year is described by the reporter.⁷

His walk suggested a foot drop on the left side, but did not show the circumduction frequently exhibited by hemiplegics. Dorsal flexion of the foot was very feeble, movements of the toes being almost completely lost. Movements at the left knee and hip could all be performed, but with less force than on the right. Knee-jerks and heel-jerks were lively on both sides, but much more marked on the left than on the right, and after three or four months ankle clonus could be elicited. The Babinski response was absent on both sides. The entire left lower extremity was not spastic but flaccid and the seat of general wasting. The left upper extremity was smaller than the right. Atrophy was also distinctly shown in the neck and shoulder. As time progressed the left upper extremity became completely paralyzed. The left face showed no paralysis or atrophy.

3. Potts, Charles S.: Jour. Nerv. and Ment. Dis., xxxviii, No. 10, October, 1901.

4. Mills, Charles K., and Spiller, William G.: Jour. Nerv. and Ment. Dis., July, 1903.

5. Patrick, Hugh T.: Jour. Nerv. and Ment. Dis., xxx, August, 1903.

6. Mills, Charles K.: Jour. Nerv. and Ment. Dis., xxxiii, No. 2, February, 1906.

7. Newmark, L.: Jour. Nerv. and Ment. Dis., xxxiii, No. 3, March, 1906.

Examinations made several years after the first observation of the patient gave, by the Oppenheim method, a flexor response in the smaller toes of the right foot, and a distinct extensor response in the big toe of the left foot. The response to both the faradic and galvanic currents was normal. All forms of sensibility, the special senses, and the bladder and bowels were normal. Absence of exaggeration of the deep reflexes in both upper extremities was an incongruous feature of the case.

These cases represent the literature of this subject as known to me up to the time of the preparation of this paper. They all seem to present in their symptomatology evidences of the probable degeneration of the pyramidal tracts, crossed and direct. I shall recur again to Case 1, when discussing the subject of the unilateral ascending type of paralysis agitans, which this case was believed to be by Patrick. Whatever the case was in its totality, it certainly presented some of the features of a case of pyramidal degeneration. Case 2, seen by me more than 20 years ago, and, therefore, in reality, the first case observed, was without doubt one of progressively ascending hemiplegia due to pyramidal degeneration. Cases 3, 5, 6, 7 and 8 seem to belong to the type first indicated by me, although differing in some features, especially as regards spasticity, the reflexes and wasting. The solution of these differences can only be had as we gain more knowledge by the study of cases, and especially as we acquire additional pathologic data. Case 5, that of a patient originally observed in my wards at the Philadelphia General Hospital, and published by me in conjunction with Dr. Spiller, is the only one as yet in which necropsy and microscopic examination have shown degeneration limited or almost limited to the crossed and direct pyramidal tracts. Case 4, recorded by Potts, was, in all probability, as he suggests, one taking the form of unilateral ascending paralysis in a comparatively early stage of disseminated sclerosis. This case will be presently referred to again when considering the unilateral ascending type of disseminated sclerosis.

I shall next present the details of an unrecorded case of unilateral ascending paralysis—one which does not seem to belong clearly with any one of the 8 cases just summarized. It is apparently an illustration of cerebrospinal syphilis, taking the unilateral ascending form.

CASE 9.—The patient, a man, aged 55, had no family history of importance. He had worked in lead 10 years before coming under observation. Four years before this time his right leg began to grow weak, the impairment of power steadily increasing. The veins of the right leg were markedly varicose. A dull aching pain developed in the heel and in the calf in the region of the varicose veins. The aching had grown worse with the weakness. Three years after the onset he first began to notice weakness and aching in the right arm, these increasing steadily. Shortly after this weakness became noticeable in the left knee and ankle, with some ache in the heel. The left arm remained normal. The bladder and bowels were not involved. He occasionally suffered from backache.

Examination showed that in walking the patient dragged his right leg. Power in the right leg was greatly diminished but normal in the left. The right arm was weaker than the left. The right lower face was paretic. He could not draw the right angle of the mouth up; on smiling the left angle of the mouth was drawn upward more than the right. He could wink each eye separately, the left better than the right. Patient stated that his left arm and leg were beginning to feel as the right extremities felt a year or more before. Muscular atrophy was absent. Standing with his eyes shut he almost fell. Both knee-jerks were exaggerated, the right more than the left. The ankle-jerk was exaggerated on the right but diminished on the left. The Babinski response was obtained on the right, but not on the left. Biceps and triceps reflexes were increased on the right and normal on the left. Sensation

for touch and pain was normal in the extremities and in the face. Speech was normal.

Eye examination revealed extensive chorioretinitis with well advanced optic nerve atrophy. The pupils responded sluggishly to light and more promptly to convergence. No diplopia or muscle insufficiency was present. The fields were much contracted, especially on the temporal side. Patient passed from under observation before an entirely satisfactory investigation of his case was made.

UNILATERAL DESCENDING PARALYSIS.

I had the opportunity of seeing and studying the following interesting case through the courtesy of Dr. William C. Pickett, of Philadelphia, whom I had requested, with others, to be on the lookout for cases of unilateral ascending and unilateral descending paralysis. Dr. Pickett, on first seeing the case, at once recognized that it belonged to the type of progressively developing hemiplegia, in this case taking a descending course.

CASE 10.—A woman, about 45, had been well until three years before coming under observation, when for the first time she noticed a weakness in the left arm on attempting to use it. She soon lost the power of lifting and projecting the arm. The impairment of power became more marked in the distal than in the proximal portion of the limb, and a few months after the onset of the paresis the movements of the hand at the wrist and those of the fingers became much impaired.

About one year from the time when the arm was first affected she noticed some weakness in the left leg. In describing the condition of her lower extremity at this time she said that she felt as if she made miscalculations in stepping up or down. In walking, the toes and fore part of the foot would catch or not be lifted properly, causing her to stumble. She soon had a limping or dragging gait. In the course of five or six months a marked degree of paralysis developed in the left lower extremity. The left upper and lower extremities had been in a condition of gradually increasing helplessness during the last 18 months. The patient said that she felt as if she had to carry them with the other part of the body. She had also a curious feeling of loss of power in the abdomen or bowels of the left side. Her face and ocular muscles had not been affected.

Neither before nor since the coming on of the paralysis the patient had not had any severe headache, paroxysmal or continuous, or any spells of nausea, vertigo or vomiting. She had also had, so far as she knew, no disorder of sight, hearing, smell or taste. She had had no loss of control or other affection of the bladder. She had had no double vision or no difficulty in dragging in the affected limbs. Her menses had continued regular, and her bowels had also been regular with the exception of occasional constipation.

Careful examination gave the following results: Left arm was limp or flaccid, the only place where any contractures were shown was in the fingers, which were half flexed at the first and second phalangeal joints, but this condition of contracture was a passive rather than an active one. The bend of the fingers was evidently due rather to loss of power in the extensors than to any active spastic condition of the flexors. The fingers and thumb dropped into the hand. The loss of power in the limb was greater distally than proximally. She could elevate the entire extremity to an angle of only about 45 to 50 degrees with the shoulder. She could, however, bend the forearm about half way, and extend the arm against resistance, but with about half the normal power; in other words, the movements of the upper arm were considerably impaired but all retained. She could not pronate or supinate the forearm; she could not flex the hand on the wrists; she could not ulnar flex it or fully extend it, but had some power of extension, especially showing itself toward the radial side of the hand. She could not extend the fingers at the metacarpophalangeal articulations or distally. She had some slight flexor power in the movements of the fingers. She could adduct and abduct the thumb which lay helpless toward the palm. Inspection showed some apparent slight wasting of the suprascapular muscles and perhaps of the small muscles of the hand.

There was not, however, the distinct atrophy which is so marked in amyotrophic lateral sclerosis and progressive muscular atrophy of the Aran Duchenne type.

MEASUREMENTS OF THE UPPER EXTREMITIES.

Middle of left forearm...7½ in. Middle of right forearm...7½ in.
Left hand7 in. Right hand7½ in.

She dragged the left leg in walking, but not with the circumduction movement of the foot which is shown in so many cases of hemiplegia. The lower extremity, like the upper, was partially paralyzed, but not in any part quite as markedly affected as the upper extremity. The loss of power was more marked distally than proximally. The movements of the left foot were tested and compared with those of the right, and all were found greatly impaired but none completely lost. Those most affected were dorsal flexion of the foot and dorsal flexion with adduction. The arch of the foot was somewhat flattened; the foot showed a tendency to turn outwards, exhibiting a partial valgus. The movements of the leg on the thigh, of the thigh on the pelvis and all other movements of the thigh could be performed with considerable force, but less energetically than the corresponding movements on the right side. The muscles of both the lower and upper extremities responded everywhere to the faradic current, but with some quantitative diminution, particularly in the upper limb.

MEASUREMENTS OF THE LOWER EXTREMITIES.

Middle of left leg....11½ in. Middle of right leg....12¼ in.
Left foot7¼ in. Right foot7½ in.

The two sides of the face were symmetrical. She had about equal control over the movements of the left and of the right side of the face. The tongue was protruded straight and showed no fibrillary tremor. No loss of power was present in the masseter, temporal and pterygoid muscles. Ocular movements were normal; there was no nystagmus. Articulation, enunciation, phonation and swallowing were normally performed. The pose of the body and head and the facial expression had none of the appearances seen in paralysis agitans. Knee-jerk and quadriceps jerk were much exaggerated on the left; they were present but not increased on the right. Patellar clonus was absent on both sides, as was also ankle clonus, although a slight tendency to clonus was twice shown on the left. Achilles jerk was marked on each side. A typical Babinski reflex was present on the left, the great toe extending distinctly and somewhat deliberately, the other toes more quickly but less decidedly. On the right the small toes were plantar flexed on stimulation of the sole, the great toe sometimes extended and sometimes flexed, but when it extended it was not in the same typical and deliberate manner as on the other side. Examination of the upper extremities was made for the wrist, and elbow jerks and the von Bechterew reflex and its modifications. The jerks were a little more marked on the left than on the right; the most marked responses were the triceps and the infra-spinatus which, while not greatly exaggerated, were undoubtedly more marked on the left than on the right side.

Well marked anesthesia to touch, pain and temperature was nowhere present, but careful and repeated examinations showed slight hypesthesia on most of the left half of the body like that so often seen in hysteria and hysteroneurasthenia of traumatic origin. Her answers, however, were not uniform as to the limitations of this slight hypesthesia. Above the waist, for instance, her replies seemed to show that the sensation was fully retained nearly all over the region of the left scapula and of the deltoid.

Dr. G. E. deSchweinitz made the following report: Vision, right eye 6/5; left eye, 6/5. A., right eye, 6.5 D; left eye, 6.5 D. Pupils normal in all their reactions; equal in size. No diplopia; no limitation of rotations of exterior ocular muscles. Esophoria less than one degree. Optic discs of good color; retinal circulation normal in distribution and carrying normally tinted blood; slight enlargement of retinal veins, but no decided disproportion between size of veins and arteries. No ophthalmoscopic changes in the retina or choroid. Dr. deSchweinitz remarks that the ocular examination is practically negative; that the eyes are normal; that a slight contraction of the color fields found to be present is probably due to lack of concentration from retinal tire in-

duced by long examination. This ocular examination is important as indicating the absence of a focal lesion like a cerebral tumor.

This interesting case, which I examined with great care, is an excellent illustration of a progressively developing descending hemiplegia in all probability due to primary degeneration of the pyramidal tracts, with possibly some slight involvement of other parts of the neuraxis. With the exception of the slight hypesthesia on the affected side, the case was purely motor, and had the usual diagnostic concomitants of a purely motor case, namely, abnormal increase of the deep and superficial reflexes. The hypesthesia, like the sensation of dragging of the limbs, seemed to be due to suggestion. The case in this respect reminded me of the idea long since advocated by Charcot, that the slight or moderate impairment of sensation sometimes observed in cases apparently due to a strictly motor cerebral lesion was sometimes best explained on the hypothesis of hysteria or suggestion. It undoubtedly belongs to the clinical group of unilateral descending paralysis, the pathology being the same as that which is present in at least some cases of unilateral ascending paralysis, the degenerative lesion, however, having developed in the upper part of the spinal cord and later in the lower. It is unlike the cases presently to be described, the one referred to me by Dr. Riesman, in which some of the symptoms seemed to point unerringly to a disseminated sclerosis beginning as a descending hemiplegia. The descending types of amyotrophic lateral sclerosis and of unilateral paralysis agitans could be easily excluded, the former chiefly by the absence of true spasticity, decided wasting, fibrillary tremors, and partial degeneration reactions; the latter by the absence of tremor, masked face and fixed pose and all the truly typical features of Parkinson's disease. Nystagmus, disorders of speech and other symptoms and signs of disseminated sclerosis were absent. The case was inexplicable on the theory of a focal lesion; syphilis, hysterical paralysis and peripheral disease could all be excluded.

UNILATERAL ASCENDING TYPE OF DISSEMINATED
SCLEROSIS.

Disseminated sclerosis may first show itself as a spastic paralysis which, beginning in the one lower extremity, ascends in a greater or less time to the upper. It can not be doubted that Case 4, reported by Potts, is one of this kind, as in addition to unilaterality and progressive ascent, this patient showed several of the typical signs of disseminated sclerosis, as nystagmus, pallor of the temporal halves of the discs and some incoordination in standing and walking. In cases of this description it is altogether probable that the sclerosis at first largely attacks the pyramidal tracts and assumes an almost systemic form. This tendency, to a large degree of systematization, is sometimes observed in cases of bilaterally beginning and progressively increasing disseminated sclerosis.

A case of the hemiplegic form of disseminated sclerosis recorded by Thomas and Long⁸ differs from the case of Potts in that the symptoms were not purely motor.

CASE 11.—A man, aged 47, seven years before his death became paretic in his right leg, the impairment of power increasing and having added to it diminution of sensibility and incontinence of urine and feces. Some improvement in the symptoms in his right leg occurred, but two years before his death these gradually increased and numbness and paralysis in the right

8. Thomas, A., and Long, E.: Compt. rend. hebd. des Seances de la Soc. de Biol., Oct. 13, 1899, No. 28.

arm appeared. The year before his death he had almost complete paralysis in the right lower extremity with contractures, exaggerated deep reflexes on the affected side, including ankle clonus; also slight atrophy of the lower extremity. The right upper extremity was a little paretic, the reflexes at the wrist and elbow were exaggerated in both upper extremities, but more on the right. Marked anesthesia was present in the right lower extremity and the right half of the trunk, and a hypesthesia in the hand and forearm. The special senses were unaffected. He died of an attack of acute pleurisy.

Plaques of sclerosis which, from their position and extent, explained the characteristics of the symptomatology and progress of the cases were found in the cervical and thoracic cord.

UNILATERAL DESCENDING PARALYSIS IN DISSEMINATED SCLEROSIS.

In rare instances, a form of more or less slowly descending paralysis is observed, that is, of paralysis beginning in the arm and face and later involving the leg. This, of course, is known to occur somewhat frequently in paralysis agitans, although after the plastic rigidity with or without tremor has attacked one upper extremity it is perhaps just as likely to cross to the opposite leg as to pass to the leg of the same side. Dr. David Riesman, of Philadelphia, has furnished me with the notes of a case which appears, when closely analyzed, to be one of disseminated sclerosis in which the paralytic, spastic and tremulous phenomena were first most marked in the upper extremity. So far as the limbs were concerned, the affection was, up to the time when the last notes were made, unilateral in its distribution. Some question may arise in the consideration of this case as to whether it is one of disseminated sclerosis of unilateral and descending type or a form of slowly developing unilateral paralysis due to meningomyelitis or some other form of specific lesion. On the whole, however, it would seem to me that the diagnosis which fits the case best is that of disseminated sclerosis, in the stage when the record was made, of unilateral and descending form.

The patient was a man forty-three years old whose father died of nephrolithiasis at the age of sixty-three and his mother of apoplexy at the age of fifty-eight. One brother was in an insane asylum for two periods of six months each.

CASE 12.—Patient had primary specific lesion at the age of 26, accompanied by alopecia and later by secondary eruptions on the abdomen, thorax and arms. He took medicine for this disease for one year. He had pyelitis 8 years before coming under observation, suffering from this affection for one year. According to his statements, he is especially subject to erysipelas, which he had had about six times in all. Two of the attacks of erysipelas he had had within 18 months, the supraorbital and infraorbital regions being affected. The patient's most evident symptoms when he came to the hospital were partial loss of motor power in the right upper and lower extremity, accompanied by violent coarse tremors of the extremities when he attempted any movements of them. He limped somewhat with the right leg. He dated his present trouble to two years before he was seen, when he began to have a dull heavy feeling in the right shoulder. He then began to lose control of the right arm, which loss of power was accompanied by trembling of the arm when he attempted to use it. He stated that within the last year, that is, about a year after the onset of the paralysis and tremor in the arm, the right lower extremity had troubled him because of a loss of power in this part. He had had sharp shooting pains in this lower extremity. He had been able to walk in the dark without falling. Both bowel and bladder control had remained intact. He had never had any girdle sensation. Consciousness had never been lost.

This showed his speech to be somewhat monotonous, with a tendency toward scanning speech; spasticity of right leg, thigh and arm; marked intention tremor of the right upper extremity; fibrillary tremor of the tongue; some

right-sided suprascapular atrophy; nystagmus; increased right knee-jerk with slight ankle clonus; normal reaction of pupils to light and distance; and intact sensation. Later notes made by Dr. Riesman showed that the left pupil was larger than the right; also that the pupils reacted sluggishly both to light and accommodation, the left not as well as the right. There was also no apparent contraction of the visual fields. The tremor in the right upper extremity was most noticeable when moving from a resting position and it seemed to disappear at times when the arms were supported. It was very noticeable when the arm was held free. The tremor was not rotary or rhythmic, but jerky and varying in amplitude. No pill-rolling motion like that seen in paralysis agitans was present. The patient tended to hold the fingers somewhat flexed at the knuckles. The hand, arm and shoulder girdle showed no atrophy. All movements of the upper extremity could be performed, but were affected by the tremor. Slight tremor was present in the right leg on its being moved, with no wasting of this extremity and no apparent shortening, although the patient said that the leg felt shorter than the other. Extension and flexion of the right foot were ample, but more sluggish than similar movements of the other foot. There was marked limitation of rotation of the right leg; other movements were normal. Knee-jerk was increased on the right side; slight tendency to ankle clonus was shown and a marked Babinski reflex was present on this side. Sensation was normal. The gait of the patient was hemiplegic, the right leg being dragged somewhat. Distinct right-sided ptosis is noted in one entry. The lungs, heart, blood, abdominal viscera and all parts of the body were carefully investigated, and notes of these examinations have been furnished me by Dr. Riesman, but as they seem to have no special bearing on the man's nervous disease they have been omitted for the sake of brevity.

Pupils 4 m.; irides react to light and accommodations; tension normal; in the right eye the media were clear and the disc pale, showing atrophic excavations; there was myopic astigmatism, 1 D. The left eye was the same as the right. Ocular movements were unimpaired. It is stated in the history that the patient had diplopia fifteen years before coming under notice, this having been corrected by glasses.

ASCENDING TYPE OF UNILATERAL AMYOTROPHIC LATERAL SCLEROSIS.

Unilateral amyotrophic lateral sclerosis, a comparatively rare form of amyotrophic disease, is occasionally ascending, although this is not the rule. Spiller⁹ succeeded in collecting 10 cases of unilateral amyotrophic lateral sclerosis; of these, four were with necropsy. Three of the ten were of ascending type (Vierordt, Mott, Senator). To these is added a case of his own, with necropsy and microscopic examination.

The paralysis began in the right lower extremity, passed to the upper limb of the same side, and later to the left lower extremity, the left upper extremity being also eventually slightly involved. In this case, besides spastic paralysis and abnormally exaggerated reflexes, considerable atrophy was present in the right upper extremity. Degeneration in this case was found in the ventral horns as well as in the pyramidal tracts. It is probable that, at least, in some cases which begin with pyramidal tract degeneration, if the patient lives long enough the ventral horns eventually take part in the degeneration; although, as already shown in the case recorded by myself in collaboration with Dr. Spiller,⁴ pathologic examination showed that the cells of the ventral cornua were not involved to any appreciable extent in this unique case.

After the publication of the paper by Spiller, an additional case of amyotrophic lateral sclerosis, at first uni-

9. Spiller, William G.: Univ. Penna. Med. Bull., January and February, 1905.

lateral and descending, has been recorded by Potts.¹⁰ Both sides of the body were eventually involved, bulbar symptoms appearing and becoming prominent.

DESCENDING TYPE OF UNILATERAL AMYOTROPHIC LATERAL SCLEROSIS.

Seven of the ten cases collected by Spiller were of descending type (Dejerine, Pick, Leyden, Lennmalm, 3 cases of Probst). Of these, five began with bulbar symptoms, the paralysis of the upper and lower extremities coming on in succession, or at least later. In a case reported by Dejerine, beginning with bulbar symptoms, the lower extremity of the side affected was more markedly paralyzed than the upper. In Pick's case paralysis began first in the left upper limb and extended to the lower of the same side; later the right side became affected. In Leyden's case bulbar symptoms were first; next weakness developed in the left upper limb and later in the left lower extremity. In Lennmalm's case bulbar symptoms were followed by loss of power in the extremities of the right side and later by paralysis in the left upper limb.

In one of the several cases reported by Probst bulbar symptoms began first, the right upper and lower extremities becoming involved. In another case reported by Probst, weakness began in the left upper and lower limbs and later in the right upper. In still another case of Probst's bulbar symptoms began first, followed by weakness in the left upper extremity and in a short time in the left lower extremity.

Recently, I had the opportunity of studying a case of unilateral amyotrophic lateral sclerosis of descending type which it may be worth while to record in this connection, owing to the fact which has just been shown that scarcely more than a dozen unilateral cases of this disease have as yet been put on record.

CASE 13.—A boy, aged about 14, was in good health until about three years before he was seen by me for his family physician, when he began to have slight tremor in the right hand. About eighteen months to two years later some weakness in the right leg was observed.

Examination showed no particular difference in the two sides of the head and face. Ocular movements were normal, as were also movements of the parts supplied by both the facial and trigeminal nerves. The right upper extremity showed distinct and nearly generalized atrophy, and some wasting was present in the right half of the trunk. The right thenar and hypothenar eminences were extremely wasted. The right lower extremity, like the upper, was also generally smaller than the left. Testing for special movements, weakness was found in all parts of the limb, but this was much more decided in the proximal than in the distal portions. The movements of the foot on the leg and the movements of the toes were greatly impaired. Every possible movement of the foot and toes was tested, with the result just given. *Farado*-contractures were well preserved on both sides; on the right side was a quantitative increase, the muscles generally responding to a current of less current strength on the right than on the left side. A moderate current produced widespread contractures in the general musculature of the arm, probably partial degeneration reaction.

On attempting movements of the right or atrophied arm the patient had associated movements of the left. No matter what movement was attempted with the right upper extremity, the left upper extremity responded, sometimes in regular and sometimes in a sort of forced athetoid movement of the hand and fingers. Voluntary movements of the right lower extremity did not cause movement in the left lower, but did cause some movement in the left upper extremity. These, however, were by no means as marked as when the voluntary effort was made with the upper right extremity. Both the deep and superficial re-

flexes were everywhere greatly exaggerated on the right side, the muscle and tendon jerks of both arm and leg being carefully examined. The right great toe remained permanently somewhat extended as in a case of Friedreich's ataxia. A marked Babinski reflex was present on this side, the already extended great toe extending still further on plantar stimulation. Ankle clonus was present on the right side. The wasting and loss of power were not present anywhere in the left half of the body. The reflexes, while prompt, were not exaggerated as on the other side, and irritation of the sole of the foot gave normal plantar flexion. A very slight hypesthesia was present in the right arm and trunk. The patient's mental condition was good. He had kept up with his class in a good preparatory school. He had neither continuous nor paroxysmal headache nor any other evidences of focal disease. The case seemed to be clearly one of unilateral amyotrophic lateral sclerosis beginning first in the upper extremity.

UNILATERAL ASCENDING TYPE OF PARALYSIS AGITANS.

It is, of course, well known that paralysis agitans is often for a long time a unilateral affection; it is not so well understood that the unilateral form of this disease not infrequently is of ascending type, the disease passing from lower to upper extremity of the same side and later to the opposite lower and upper extremity. Patrick regarded one of the cases described by me in 1900 as an illustration of this affection rather than a case of primary degeneration of the pyramidal tracts, but of this I have not been convinced. With regard to this matter I need only reproduce here remarks made in a previous paper written in collaboration with Dr. William G. Spiller:⁴

Among the reasons for believing that the case was not paralysis agitans were the absence of spontaneous tremor; the presence of markedly exaggerated reflexes on the affected side, even including ankle clonus and patellar clonus; the existence of decided wasting on one side, especially of the lower extremity; and the absence of the facies of paralysis agitans and of a fixed position of the body and head. According to Sir William Gowers¹¹ both primary lateral sclerosis and paralysis agitans are probably abiotrophies, and it may be that in the case under consideration the two abiotic diseases are conjoined.

Patrick⁵ reports a case of unilateral paralysis agitans in his paper with the view of indicating the similarity of this case to that reported by me and reinvestigated by him. In this case the disease began in the upper extremity, passing later to the lower. My impression is that this is rather the usual course, although I have not made any collection of cases to demonstrate the point. Whether or not the case reported both by Patrick and myself was one of paralysis agitans or of paralysis agitans and pyramidal degeneration conjoint, it is certain that Parkinson's disease may remain for a considerable time unilateral, and beginning in the lower extremity may take an ascending course. Recently, I have seen two cases of this ascending type.

CASE 14.—A man, aged 55, a pattern maker, first began to notice stiffness or impairment in movement in his left leg in December, 1904. At first and for some time there was no involvement of the left upper extremity, but this gradually came on, and at the time when he was first seen, about 16 months after the beginning of the trouble in the leg, the arm was very markedly affected. He had occasional cramps in the legs, the toes at times contracting when these cramp-like feelings were present. He also had at times, especially after sitting, feelings of numbness or tingling in the left leg and arm.

Examination showed a rather fixed position of the head, slowness of gait with some dragging of the left foot, and a marked tremor in the left upper extremity, especially in the hand, this being the type usually seen in Parkinson's disease. Observed over short periods it was sometimes present and at

10. Potts, Charles S.: *Univ. Penna. Med. Bull.*, July and August, 1905.

11. Gowers, Sir William: *Lancet*, April 12, 1902.

others absent, and it was a little increased by voluntary effort or attention. While both the leg and arm on the affected side showed a little stiffness and awkwardness in movement, real loss of power was slight. The patient still continued to work at his business, even using the left hand. He said that if he could get a good grip on an object with this hand he could go on using it in his work. Sensation was everywhere retained.

CASE 15.—A woman, aged 43, first observed weakness in the left lower extremity four years before coming under observation. Tremor did not appear for three years in the left lower extremity and shortly after this the upper extremity became involved, showing both weakness and tremor. Symptoms appeared in the lower extremity of the opposite side three and a half years after the beginning of the disease. The case was clearly one of paralysis agitans and need not be described at length.

It is not my intention to go with any elaboration into a discussion of the differential diagnosis of unilateral ascending and unilateral descending paralysis from focal lesions, cerebral or spinal, from cerebrospinal syphilis, and from peripheral or hysterical affections. For the sake of completeness, however, a few words may be said about these diagnoses. In a rare case a brain tumor may originate in the cortical leg area or beneath it and develop toward the arm and face regions so as to cause a progressive hemiplegia of the ascending type. The reverse might, of course, take place, and the tumor originating in or near the face or arm area and progressing toward the leg center, give a unilateral descending paralysis. In such a case some or all of the general symptoms of brain tumor would be likely to be present, and other focal signs of this affection, especially Jacksonian epilepsy, would sooner or later appear. A tumor of the internal capsule would most probably produce loss of power in both arm and leg nearly simultaneously. In some cases of arteriosclerosis necrotic areas form first in one and then in another portion of the brain, and it might happen that these would be so placed in succession in the motor region as to give unilateral ascending or unilateral descending paralysis. Such a case would develop clinically by steps rather than by regular progression, and other signs or symptoms indicating the character of the lesions could probably be detected if sought for diligently. A single tumor or necrotic lesion of the spinal cord would not cause a paralysis ascending from the leg to the arm, as the cerebrospinal tracts passing to the nerve nuclei for the upper extremity do not proceed lower than the upper thoracic region. Peculiarly situated and peculiarly progressing focal lesions of the cervical cord might simulate a unilateral descending paralysis, but the other well-known symptoms of such a unilateral focal lesion would be present and the subject hardly needs discussion.

The forms of spinal and cerebrospinal syphilis are protean. A spinal or intracranial gumma or gummatous meningitis or a succession of focal syphilitic lesions might give rise to a one-sided paralysis appearing first in the leg and afterward in the arm, and later taking the form of a triplegia. This is, indeed, what occurs in some cases of meningitis or meningomyelitis with a secondary degeneration of the pyramidal tracts. The new case of unilateral ascending paralysis (Case 9), had some of the features of a cerebrospinal syphilitic affection, although the patient denied venereal disease. Close attention to the history, and the presence of other evidences of syphilis and the variable and irregular manner in which the paralytic symptoms appear will assist in differentiating.

A paralysis of peripheral origin, of ascending or de-

scending type, is rare, but, of course, may occur. Such a case would probably depend either on the degenerative neuritis or on the succession of peripheral lesions. The manner in which each stage of the paralysis appeared, the state of the reflexes, the results of electrical examination, and other well-known methods of determining the existence of a peripheral nerve disorder would serve to separate such a neural case from the forms of progressive hemiplegia under consideration. The absence of hysterical stigmata, as well as the slowly progressive manner of appearance of the paralysis of leg, arm and face would make a hysterical hemiplegia improbable. A pure neurasthenia never shows itself as a slowly developed and permanent hemiplegia.

DISCUSSION.

DR. HERMAN H. HOPPE, Cincinnati, said he was very glad to hear Dr. Mills increase the latitude of the subject. He reported a case perhaps of this kind that he had had under his care for about a year or 18 months. A young child about 3 or 4 years of age developed spastic paralysis, which first appeared in the arm, and then descended to the leg and ascended to the face. The child was absolutely healthy up to the age of 2½ years; it developed, intellectually and otherwise, normally, walked normally and looked like a normal child; then it gradually developed paralysis, spastic in character, of the right arm, then spastic in character of the right leg, and then of the face, during a period of perhaps six or eight months. In other respects the child was perfectly well; there was no headache and the child ate and slept well. One peculiarity about the case was that there was absolutely no syphilis, and the mother gave birth to other children who are well. The mother was addicted to drink, which was particularly manifested before the child was born. Until Dr. Mills spoke of the descending type Dr. Hoppe was very much puzzled about this case. Whether there was any other feature about the case that would develop in the future he could not say. That particular manner of development of spastic paralysis in a child never occurred to him before. There was no history of any infectious disease of any kind which might have caused an encephalitis.

TECHNIC IN THE AFTER-CARE OF THE RADICAL MASTOID OPERATION.*

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To remove diseased tissue thoroughly from the temporal bone, in cases of chronic suppuration, is an easy task compared with the difficulty of obtaining a resultant cavity absolutely free from granulations or discharge, and lined with healthy new epidermis. To establish such a condition speedily is an end toward which we are all striving.

When the first radical operations were done in 1889, it was no uncommon thing to have suppuration continue within the cavity for many months; healing was finally obtained only at the expense of much time and suffering, and was usually attended with considerable deformity. To-day it is possible to have our exenterated cavity thoroughly healed in a comparatively short time, in many cases only five weeks, and with absolutely no post-aural deformity, only a fine linear scar marking the line of incision.

* Read in the Section on Laryngology and Otology of the American Medical Association, at the Fifty-seventh Annual Session, June, 1906.