adjustment and a readiness to co-operate to the full during the first crucial years of the disease. Above all, the impression must not be left on the patient's mind that he or she is suffering from a mysterious disease about which nothing is known and for which there is no treatment.

Summary

An air of defeatism tends to surround the subject of multiple sclerosis. The reasons for this attitude are examined in some detail.

Aetiology is discussed with especial reference to the allergic theory.

A diagnosis of multiple sclerosis is usually not made until the fourth or fifth year of the disease. The causes of this delay are discussed and early symptomatology is reviewed.

The traditional belief that multiple sclerosis inevitably leads to increasing disability and premature death requires modification in the light of recent studies. A brief reference is made to the concept of varying degrees of resistance and to a benign form of the disease.

A plea is made for a more positive attitude towards treatment. In an early case more attention should be paid to the constitutional and environmental background of the patient. Stress is laid on the importance of rest during active phases of the disease, followed by prolonged convalescence. In responsible adults an explanation of the nature of their illness is essential in order to obtain intelligent co-operation in treatment.

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A new British Standard (2805 : 1957) specifies a range of straight artery (haemostatic) forceps of the Spencer Wells pattern having a screw joint and made of stainless steel. Provision is made for the 5 in., 6 in., 7 in., 8 in., and 9 in. (12.7–22.8 cm.) sizes. The preparation of this Standard was recommended by the Ministry of Health and authorized by the Surgical Instruments and Medical Appliances Industry Standards Committee of the British Standards Institution. British Standard 2805 may be obtained through the Sales Branch of the British Standards Institution, 2, Park Street, London, W.1, price 3s. 6d.

POLYRADICULITIS (LANDRY–GUILLAIN-BARRÉ SYNDROME)

TREATMENT WITH CORTISONE AND CORTICOTROPHIN

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The clinical and pathological identity of Landry's ascending paralysis and the Guillain–Barré syndrome (polyradiculoneuritis; acute infectious, toxic, or febrile polyneuritis; or infectious neuronitis, polyneuritis cranialis, etc.) was fully established by the important review of Haymaker and Kernohan (1949). Since then the non-specific nature of the syndrome and its relation to other acute inflammatory diseases of the nervous system, and possibly to allergy, have been noted (Stanton *et al.*, 1953), while the results of treatment with corticotrophin and cortisone have been described by Clarke *et al.* (1954) and others.

In the past three years we have treated five deteriorating cases of this condition with corticotrophin or cortisone, and in this paper we present these cases, review the available literature on the subject, and endeavour to draw some general conclusions.

Case 1

On November 6, 1953, three weeks after a brief attack of acute febrile bronchitis, a 28-year-old docker developed an illness initially regarded as influenzal, characterized by fever, backache, and headache. Twenty-four hours later he developed tingling in the tip and dorsum of the tongue, and, after a similar interval, ascending paraesthesiae and weakness in both lower limbs. By November 9 he could progress round the room only by holding on to the furniture. Weakness of the legs increased further, and on November 13 he noticed paraesthesiae in both hands. His speech became slurred, and food stuck in his throat; there was slight dysuria and some constipation. At this stage he came under medical observation, and over the course of the next few days all deep reflexes disappeared, asymmetrically bilateral facial weakness developed, and loss of superficial sensibility became evident over the whole of both lower limbs, the forearms, and a band round the trunk between nipples and umbilicus. The legs were much weaker than the arms, and when he was admitted to our care on November 18 he was unable to stand. Weakness and muscle tenderness were generalized, but were most marked in the proximal limb muscles. Blood examination, E.S.R., E.C.G., and *x*-ray films of the chest and spine showed nothing abnormal. The only abnormality in the spinal fluid was a protein content of 240 mg. per 100 ml., rising subsequently to [illegible]0 mg.

In view of continuing deterioration, and especially pharyngeal pooling of mucus owing to bulbar weakness, oral administration of cortisone was begun at once. He was initially given 300 mg. daily, but this was reduced gradually to 100 mg. daily by the fifth day, and the smaller amount administered daily for a further five days.

Within 18 hours of first receiving cortisone (225 mg.) the patient claimed to feel much better and that his voice was stronger, but examination failed to substantiate his claim of improvement, which was attributed to pharmacologically induced euphoria. Within 36 hours of instituting treatment, however, the facial diplegia showed unequivocal improvement and he could whistle feebly. His voice appeared to be somewhat stronger, but his own claims of improvement in swallowing and improved power in the limbs were unsupported by objective evidence, which showed no change in power, reflexes, or sensation.

Reassessment at the end of 10 days' treatment suggested that the condition had ceased to progress. Facial weakness was greatly improved, the voice stronger, and swallowing normal. Sensory loss was less extensive on limbs and trunk, while pain and muscle tenderness had disappeared. Motor weakness in upper and lower limbs, however, was as severe as on admission, while there was no return of reflex activity.

Subsequent improvement was slow but steady, and five months later his sole disability was some weakness of the left leg. By August, 1954, there was no residual disability and all deep reflexes had recovered.

Comment.—In this severe case of polyradiculitis the administration of cortisone was followed by unequivocal clinical improvement within 36 hours, and at the end of 10 days' treatment this improvement was striking. From this point onwards, however, further improvement was slow, and it was eight months before recovery was complete.

Case 2

A 33-year-old housewife was admitted to hospital under the care of Dr. H. A. Dewar on November 16, 1955, with an eight-weeks history of tingling of the fingers and hands, followed by slight loss of grip. Five weeks before admission she had developed weakness of her ankles and legs and had been put to bed for one week, following which she got up again, but was able to get about the house only with the aid of sticks. The weakness increased and two days before admission she was unable to walk. Constipation had been troublesome throughout the illness.

On examination she appeared to be in good health. The cranial nerves were normal, but there was some loss of power in the forearms and marked loss of power in both legs, with severe bilateral foot-drop. All deep reflexes were absent, and muscle pains and tenderness were conspicuous. There was loss of superficial and deep sensibility in both feet. The spinal fluid contained 800 mg. of protein per 100 ml. Other ancillary investigations were negative.

During an initial period of two days' observation there was no improvement in her condition, and after this time cortisone was administered. She was given 200 mg. daily for three days, 100 mg. daily for 18 days, the drug being then gradually withdrawn over the course of a week. Within 48 hours subjective and objective improvement was evident: her grip was improved and toe movements had returned. Within eight days she could move the legs weakly, and improvement of power in the hands permitted knitting. There was improvement in sensibility in the legs, and the deep reflexes had returned weakly in the arms.

From this time progress was slower. Within three weeks, however, she was able to walk with assistance, and was discharged from hospital on December 31—six weeks after admission.

When the case was reviewed six months later, progress had been continuous but slow, and the patient was able to walk without the use of sticks. Her only complaint was that she put her feet down rather heavily when walking. All limbs showed absence of deep reflexes, but power was good apart from slight weakness of dorsiflexion of the feet; sensation was normal.

Comment.—This patient, an example of mild polyradiculitis, showed what appeared to be a satisfactory initial response to treatment with cortisone, followed by slow improvement indistinguishable from that seen in untreated cases which recovered.

Case 3

A 40-year-old driver was well until a month before admission to hospital, when he noticed some tingling in his fingers and toes. A week later he developed stiffness and pain in the neck and shoulders, and four days after this sharp cramping pain occurred in the muscles of the left thigh. During the next two weeks muscle pains became severe and generalized; he developed a moderate fever, a right facial palsy of peripheral type, mistiness of vision, and slurred speech.

On his admission to hospital on December 30, 1955, examination revealed slurring dysarthria, neck stiffness, bilateral facial palsies more evident on the right, loss of power in the limbs which was more marked peripherally in the lower and proximally in the upper limbs, general hypotonia, and loss of reflexes with widespread muscle tenderness, especially marked in the calves. There was no sensory loss, but the soles were hyperaesthetic. The left plantar response was flexor, the right variably extensor. Ancillary investigations were normal, but spinal fluid examination yielded as the only abnormality a protein content of 290 mg. per 100 ml., a figure which was almost doubled on subsequent examination.

Clinical deterioration continued, and, as the patient developed slight difficulty in swallowing, 200 mg. of cortisone was administered at once, followed by 100 mg. thrice daily. Some improvement was evident within 18 hours of the first dose and was quite marked in 48 hours, by which time the facial palsies were much less conspicuous (the severe right-sided paralysis showing the more striking response), he was able to whistle a little, and speech was distinctly clearer. The dose of cortisone was gradually reduced to 200 mg. and then to 100 mg. daily, and its administration was stopped on the seventh day. At this time, in addition to the improvement already noted, there was increased power in the lower limbs; he was able to dorsiflex both feet, while both plantar responses had become clearly flexor.

Twenty-four hours after the last dose of cortisone the patient, a phlegmatic individual, complained of suddenly feeling very ill with intense pain in the limbs, particularly the legs. These symptoms cleared up within half an hour, but by this time a marked deterioration in physical signs was evident. His right facial weakness was again much more pronounced, vision in the left eye was blurred (without objective findings), his legs were weaker, and the plantar responses were unobtainable. By the next day this deterioration was more evident. His speech had once more become slurred, and he was having some difficulty in passing water.

In view of these developments he was given a further course of cortisone, beginning five days after the end of the previous treatment. On this occasion no dramatic response was obtained. He claimed to feel better, and there was certainly no further deterioration, but on re-examination after 10 days on cortisone his objective improvement was negligible.

From this time the slow but progressive improvement in function which occurred was similar to that with which we are familiar in a severe case of the untreated disease. It was four months before he was able to stand, and even in September, 1956, walking was still much impaired, while the deep reflexes had not yet reappeared.

Comment.—This patient showed unequivocal improvement within 24 hours of the administration of cortisone, but a relapse which occurred when the drug was withdrawn showed little response to a further course of treatment given within four days of the onset of deterioration. On this second occasion the drug relieved pain but produced no improvement in physical signs.

Case 4

A 5-year-old girl was admitted to the Sunderland Children's Hospital under the care of Dr. J. B. Heycock on February 7, 1956. Six weeks before admission she developed tonsillitis, and after this had subsided she remained unwell in herself for a week, during which time she was kept in bed. At the end of this period she developed spontaneous pains and painful cramps in the legs and was observed to limp. These symptoms became more pronounced during the fortnight preceding admission, and she was noticed to lie down a good deal with knees flexed in order to obtain relief.

On admission she was unwell and miserable, with flaccid weakness of all limbs, especially in the legs, and with total disappearance of deep reflexes. The muscles were tender, but no loss of sensation was demonstrable. The spinal fluid protein was 100 mg. per 100 ml., and all other ancillary investigations were negative.

Over a period of 48 hours' observation there was unequivocal deterioration with increasing weakness and at the end of this period (February 17) cortisone was started by mouth, 100 mg. daily being given in four doses for five days, and then 200 mg. daily for seven days, after which time the drug was gradually withdrawn over the course of four days.

The response was not dramatic. What had previously been a steadily deteriorating course ceased to progress, and pain disappeared within 24 hours and the child's general condition also appeared to be improved. There was, however, no convincing change in the neurological findings, except for the disappearance of the muscle tenderness, until seven days after the end of treatment, when the condition began to show unequivocal improvement with increase in power in the legs which was rapidly progressive. Within a month there was a marked degree of improvement in the power of all limb groups, though the deep reflexes remained absent.

She was discharged from hospital on April 4, and continued with exercises and physiotherapy for a further month, when she was again reviewed. Re-examination showed steady but slow improvement; there still being some weakness of the arms and legs, most pronounced in the quadriceps. The deep reflexes were still absent.

Comment.—Except for disappearance of pain and muscle tenderness, this child showed no evident improvement on cortisone, the administration of which coincided, however, with the arrest of progressive deterioration in her condition. Objective improvement in physical signs was not evident until after treatment was withdrawn, following which the course of the illness was characteristic.

Case 5

A 40-year-old stone-mason presented on August 24, 1956, with a story of four days' numbness and weakness spreading up the arms and legs. His weakness rapidly became so profound that the condition was at first thought to be hysterical, but he was seen by a physician, who discovered absence of reflexes, muscle tenderness, and peripheral sensory loss. The condition remained fairly static for three weeks and then quite rapidly became worse, with ascending intercostal pareses, facial diplegia, and bulbar weakness with pooling of mucus in the pharynx. At this stage spinal puncture showed 800 mg. of protein per 100 ml. In view of increasing difficulty in breathing, the patient, who was now under the care of Professor G. A. Smart, was given intermittent assisted respiration and 40 units of corticotrophin twice daily. Improvement was dramatic. Within 24 hours he was able to whistle, there was a surprising improvement in the power of the left arm, and his cough was stronger. Six days later breathing was normal, both arms were stronger, there was some return of power in the legs, and sensory change was beginning to shrink. Within a month of beginning treatment the only disability was partial bilateral foot-drop, rapidly improving, though knee- and ankle-jerks remained absent. As a safeguard against possible relapse, 5 mg. of prednisone was given three times daily for three weeks after the completion of 10 days' treatment with corticotrophin.

Comment.—An apparently dramatic response to corticotrophin in a severe and advancing case of polyradiculitis was followed by unusually rapid recovery.

Review of Literature

Since Stillman and Ganong (1952) first reported a case of Landry–Guillain–Barré syndrome treated with corticotrophin, there have been 34 papers in which the treatment of 68 cases has been described. Review of these shows that in 50 there has been some response and in 18 the response has been negligible. In one case the disease developed during cortisone therapy (Grant and Leopold, 1954), but in no other case was deterioration reported during treatment.

In evaluating the treatment of the Landry–Guillain–Barré syndrome with corticotrophin and cortisone, it would be desirable theoretically to have a sufficiently large series of cases under the care of one observer, so that variations in the amount, duration, and route of treatment could be standardized, allowance made for the wide variation in clinical picture, and a control series of non-treated cases arranged. Practically, however, this is impossible, and we would emphasize that we have been able to draw only a few general conclusions; the wide variations in the degree of detail of these reports have made it impossible to study the exact clinical pictures. In general, cases in which there has been a gratifying response have been reported in greater detail than those in which the response has been less dramatic, and it must be borne in mind also that there is always a tendency to report successfully treated cases while the unsuccessful ones remain in obscurity.

One further difficulty in analysing the literature of such a pleomorphic condition as Landry–Guillain–Barré syndrome is to know whether in fact the cases reported are examples of this condition. There is no easy solution to this problem, for it is difficult to draw up a list of criteria which have to be fulfilled before a particular case can be accepted as Landry–Guillain–Barré syndrome, and we have therefore included in our survey all cases so described.

In analysing the literature note has been made of the following points: sex, age, association with any other disease (such as infective hepatitis, periarteritis nodosa, etc.), severity of the disease, protein level of the spinal fluid, length of history prior to admission to hospital, duration of observation before treatment, dosage and mode of administration of the drug, and duration of treatment. These have been assessed to see if they have any bearing on the response to treatment. The severity of the disease has been arbitrarily defined as "severe" if there have been bulbar or cranial nerve involvement, phenomena such as sudden circulatory collapse sufficient to endanger life seriously, and extensive and profound limb involvement. "Moderate" is used to describe cases in which there has been no bulbar involvement but in which the limb paralysis has proceeded as far as a complete paralysis or tetraplegia. "Mild" means limb weakness only, without complete paralysis, even if widespread. It is felt that some such classification as this is useful in relating severity of disease to response to treatment.

As mentioned above, 18 cases failed to respond, but 12 were unsuitable for analysis as no details are given, and it is felt that the six cases in which details are available do not constitute a large enough group to make an analysis valid. It is not possible, therefore, to compare a group of cases which respond to treatment with a group which failed to respond. Consequently we have no means of predicting which cases are likely to respond.

The 50 cases showing response seem to fall naturally into three types: type 1, in which there is an immediate and sustained response to treatment, and in which recovery is complete within one month of starting treatment (21 cases);

type 2, in which there is a brisk initial response with early partial improvement, but in which final recovery does not occur so rapidly and the tempo of the initial improvement is not sustained (full and final recovery is usual—this type comprises 23 cases and also Cases 1 to 3 described above); and type 3 (6 cases), a miscellaneous group in which the type of response is not readily classified under the other two headings. Examples of types 1 and 2 are as follows:

Type 1.—A 17-year-old girl (Seltzer *et al.*, 1952) developed symptoms of Landry–Guillain–Barré syndrome 10 days before admission to hospital. During the next 10 days she continued to deteriorate and required tracheotomy. On the twentieth day of the illness—that is, 10 days after admission—treatment with 25 mg. of corticotrophin eight-hourly for five days was started, with effect within 24 hours. Improvement was maintained and she progressed so satisfactorily that the tracheotomy was closed nine days after the commencement of treatment, walking was possible seven days later, and 28 days after admission the patient was well enough to be discharged; a fortnight later she returned to school.

Type 2.—A boy aged 15 (Hay, 1955) was admitted to hospital with a five-weeks history of difficulty in walking, paraesthesia for a fortnight, and complete inability to walk for two days before admission. Examination showed generalized lower-limb weakness associated with loss of deep tendon reflexes. Steady deterioration, manifested by increasing weakness in both arms associated with loss of tendon reflexes, was noted during the initial period of nine days' observation, at the end of which time a course of cortisone was started. Subjective and objective improvement in both hands was noted within 24 hours. The patient was able to sit up on the fifth day, and by the 26th day was able to walk, although some weakness persisted until five months later, when full recovery of muscle power had taken place (at that time, loss of tendon reflexes in both legs was still evident).

The dramatic and possibly life-saving effect of corticotrophin or cortisone as typified in the type 1 case made us examine the cases further to decide if there were any indications of the type of case which would produce this response. However, there is no obvious difference between types 1 and 2 in sex, age, severity, spinal fluid protein, or in the dosage or route of administration of the drug employed. Cases in which treatment was started within one month of the onset show a slightly increased tendency to show a sudden and sustained response, though the difference is not statistically significant.

In fact, although the occasional dramatic response to cortisone is at least suggestive, statistical proof of its value is lacking. No control trial has been reported in the literature, and information on the natural history of the disease is curiously lacking in terms of concrete figures which can be used for comparison. Brain (1951) states that in the most favourable cases the patient may not be convalescent until from three to six months. Bradford *et al.* (1919) state: "In cases of a severe type it would seem that a period of about two months elapses between the onset of the palsy and a degree of recovery that renders evacuation feasible, and that a period as long as six months may be necessary for the patient to regain his usual health." Holmes (1917), in a series of 12 cases, noted "steady and rapid improvement which usually sets in within two or three weeks of onset. The pains and hyperaesthesia diminish quickly after the first fortnight or so, and somewhat later the power of the paretic muscles begins to return gradually." However, in none of his cases was recovery complete. Gilpin *et al.* (1936) state that the entire duration of the illness in 20 cases varied from 10 weeks to 2½ years, the average being about 6½ months. The fact that 21 cases out of 68 treated with cortisone recovered completely within one month does at least suggest that the rate of recovery is hastened as a result of the treatment.

Reported death rates vary considerably. Guillain *et al.* (1916), in their original paper, laid down complete recovery as one of the criteria for diagnosis, and Guillain (1936) much later affirmed that none of his patients died. Other authors (see Table) give a mortality rate varying from 8.7% to 42% in cases not treated with cortisone.

Of reported cases treated with cortisone or corticotrophin, only two patients have died. In one (Guiang and Leones-Guiang, 1954) the patient, a boy aged 2, was removed from hospital by his mother when he appeared to be responding to treatment. He relapsed and died at home. In the second (Fazlullah, 1956) the patient died of acute pulmonary oedema associated with adrenal failure following a complex and bizarre illness with some features suggesting polyarteritis nodosa.

Mortality Rate of Landry–Guillain–Barré Syndrome

Author	No. of Cases	Deaths
Holmes (1917)	12	2 (16·6%)
Bradford *et al.* (1919)	30	8 (26·4%)
Gilpin *et al.* (1936)	20	4 (20%)
Roseman and Aring (1941)	16	3 (18·8%)
Forster *et al.* (1941)	26	11 (42%)
Von Hagen and Baker (1953)	23	2 (8·7%)

Discussion

The general features of the condition are familiar—a rapidly or insidiously ascending flaccid paralysis, sensory impairment often being inconspicuous or absent, with muscle tenderness, loss of deep reflexes, and, in classical though not in all cases, cytoalbuminological dissociation in the spinal fluid. A considerable number of these cases show meningism, while an occasional transient extensor plantar response bespeaks extension of the lesion into the spinal cord. Frank myelitic or encephalitic concomitants are rare, but such mixed forms undoubtedly occur.

Aetiology is multiple. The condition occurs as an occasional complication, much less frequent than encephalitis or myelitis, of many specific infections, among which measles, varicella, rubella, and mumps are conspicuous examples, while its occurrence in infectious mononucleosis and infective hepatitis has recently attracted attention. Significantly, its occurrence is not limited to such infections, and it is seen also as a complication of serum sickness and of many sterile prophylactic inoculations such as T.A.B. or rabies vaccine, diphtheria toxoid, etc. (Miller and Stanton, 1954). Most cases, however, arise "spontaneously," though in many such patients there is a recent history of banal infection.

According to Von Hagen and Baker (1953), Bergamasco and Bottiglioni have claimed evidence of a neurotropic virus, but Aring and Sabin (1942) found that culture and transmission experiments were unsuccessful (quoted by Chusid and Marquardt, 1945).

Histopathological changes are maximal in the spinal nerve roots; Scheinker (1949) found that the axis cylinders in the nerve roots were enlarged owing to oedema, and that fragmentation and "corkscrew" formation were also present. Changes in the myelin sheaths—beading and irregular swelling—were also found in some cases. In the cord itself there was some swelling of the anterior horn cells and the axis cylinders, and the white matter showed marked swelling. Inflammatory reaction was conspicuous by its absence. These findings agree largely with those of Gilpin *et al.* (1936) and of Roseman and Aring (1941), and are similar to those described in polyradiculitis complicating frankly allergic disorders such as serum sickness.

An explanation of the clinical picture and the cytoalbuminological dissociation is put forward by Scheinker (1949) and by Reitman and Rothschild (1950), who suggest that the oedema of the nerve roots and trunks produces compression within the firm perineural sheaths. Hassin (1948) claims that about one-fifth of the spinal fluid is absorbed through the perineural spaces in the spinal canal, and Aring (1945) showed that in cases of Landry–Guillain–Barré syndrome the spinal fluid protein level in the lumbar theca was much higher than that in fluid obtained by

cisternal puncture. Boshes and Sherman (1953) point out also that if spinal fluid is allowed to drain off during a lumbar puncture the fluid collected in the first tube and therefore coming from low down in the spinal canal has a higher protein content than fluid collected in subsequent tubes coming from higher up. As Reitman and Rothschild (1950) point out, it is possible that the oedema of the nerve roots prevents the passage of the larger protein molecules, but allows selective absorption of the fluid, and this leads to the high protein level that Guillain regards as the *sine qua non* of the diagnosis of the Landry–Guillain–Barré syndrome.

It appears probable that oedema and nerve-root compression may give rise to the clinical features of the syndrome by an immediate loss of function of the nerve, which may be temporary and quickly reversible if the oedema subsides rapidly. If, however, pressure from the oedema persists or is unduly severe, permanent damage is produced with demyelination, recovery from which can take place only by regrowth of axons down the nerve. This view is in agreement with Seddon's (1948) concept of peripheral nerve injury.

Recovery from the Landry–Guillain–Barré syndrome therefore might be expected to take place in two stages: first, an immediate recovery resulting from the regression of the oedema, and, secondly, a more gradual recovery of some months' duration, while the axons are growing down the nerve. This "two-stage" recovery is precisely what appears to happen during treatment with corticotrophin or cortisone; the degree of recovery may be rapid and complete if the lesion is due to oedema only (this corresponds to type 1 cases noted in our analysis of the literature), or a partial recovery may be followed by a longer period of disability during regeneration of the demyelinated axon (type 2).

This perhaps rather speculative interpretation of the histological changes observed in the disease provides at any rate a plausible explanation for the two main types of response to cortisone which we have noted above.

Stanton *et al.* (1953) have pointed out that Landry–Guillain–Barré syndrome can be related closely to other acute "inflammatory" diseases of the C.S.N., and possibly to allergy, and since all these syndromes, whether mixed or pure, arise on a similarly non-specific aetiological basis it seems possible that to regard them as varying manifestations of an "allergic neuraxitis" may be something more than merely a facile oversimplification.

In this context a case seen by one of us (H. M.) is of interest:

A physician aged 49 was given 1,500 units of antitetanic serum into the left upper arm, and six days later developed signs and symptoms of serum sickness, with joint pains, high fever, and sweating. On the second day he developed a headache with neck stiffness and photophobia, and became mentally confused. Within six hours of the onset of the headache he was given cortisone and salicylates, a therapeutic response occurring within six hours. Next morning he was so much better that the cortisone was stopped after 50 mg. had been given. The same evening he developed severe pains and paraesthesiae in all limbs, and 48 hours later marked weakness of the right arm, loss of both ankle-jerks and of the left knee-jerk, and impaired vibration sense in the toes. A diagnosis of polyradiculitis complicating serum sickness was made but no lumbar puncture was done. He was given cortisone again as he appeared to be deteriorating, and again dramatic clinical improvement occurred within 12 hours, power recovering and paraesthesiae and pain completely disappearing. Within three days the deep reflexes had also recovered completely.

The Landry–Guillain–Barré syndrome appears, then, to belong to a spectrum of diseases of the nervous system which are secondary to a wide variety of aetiological agents but which have in common a probable basis of allergy. The effect of cortisone is unpredictable, inconstant, and difficult to analyse statistically, but, as in other allergic disorders, its use is justified on theoretical grounds as well as in practice.

Summary

Five cases are described of the Landry–Guillain–Barré syndrome treated with cortisone or corticotrophin.

The literature recording similar cases is reviewed.

The response to the drugs is unpredictable and inconstant, and the material available is not suitable for statistical analysis. However, in 21 out of 68 recorded cases complete recovery occurred within a month. This is unusual in untreated cases. Twenty-three other patients showed a rapid initial response followed by slower subsequent recovery. Arrest of deterioration is usual, and the mortality in this admittedly heterogeneous collection of treated cases was much lower than in previously recorded series.

The aetiology and pathology of the disease are discussed. It is considered that the clinical features, pathology, and response to treatment are compatible with an allergic pathogenesis.

We thank our medical colleagues in the Royal Victoria Infirmary and in the No. 1 Area, especially Dr. H. A. Dewar, Dr. J. B. Heycock, and Professor G. A. Smart, for kindly referring their cases.

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