The remaining four patients made good recoveries after discontinuance of the infusions. Although one died of congestive heart failure four months after the cardiac infarction, the other three were alive and active at intervals ranging from six to thirteen months afterwards, their lives apparently having been saved by the treatment.

The criteria for infusion of L-noradrenaline are discussed and the methods and precautions necessary for its use described.

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ACUTE DISSEMINATED ENCEPHALOMYELITIS AND ACUTE DISSEMINATED SCLEROSIS RESULTS OF TREATMENT WITH A.C.T.H.

BY

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The treatment of seven cases of acute disseminated encephalomyelitis with A.C.T.H. was reported in a previous paper, in which the theoretical basis for trial of the drug was discussed (Miller, 1953). The results of treatment were held to be suggestive enough to warrant further trial. Three further cases have now been treated, and they are briefly reported below.

We have also used A.C.T.H. in the treatment of seven patients with acute exacerbations of disseminated sclerosis. Glaser and Merritt (1952) have already reported on the use of the drug in disseminated sclerosis. They treated 33 cases of the disease, giving 80 to 100 mg. daily during a course of 14 days' duration. These were chronic cases, and none had experienced significant remission during the three years preceding treatment, or since the onset of the disease in cases of less than three years' duration. Twelve patients showed significant improvement. The syndromes benefited were spastic-paretic, cerebellar-ataxic,

bladder dysfunction, and visual impairment of recent origin. Oculomotor disorders, sensory impairment, and reflex changes remained unaffected. The effects observed appeared during the course of drug administration and rarely persisted for more than seven to fourteen days after the end of treatment. In most cases the disease later resumed its usual course, though these authors believed that subsequent maintenance treatment with 75 to 150 mg. of cortisone daily might possibly retard its progression. They considered that the beneficial effects of the drug represented "secondary physiological phenomena due to the hyperadrenal state," whether produced by interference with abnormal immunological reactions in the central nervous system, suppression of inflammatory and connective-tissue responses, or the effect on nervous excitability of chemical changes related to the profound metabolic disturbance which A.C.T.H. is known to produce. A somewhat similar transitory improvement in cases of established disseminated sclerosis has been reported by Fog (1951).

We have, however, restricted the experimental use of A.C.T.H. to patients with a recent acute exacerbation of unequivocal established disseminated sclerosis. All cases had recently developed an acute neurological episode with some objectively assessable focal disability, either a fresh manifestation of the disease or the sudden aggravation or reappearance of a previous symptom. No patient was showing any sign of spontaneous improvement when treatment was begun, and progressive deterioration was evident in several. It seems reasonable to believe that the onset of an acute neurological episode of this nature signals the development of fresh plaques in the central nervous system. Although there is no general agreement on the pathogenesis of disseminated sclerosis, nor indeed on the histopathological changes present in recent lesions, it seemed justifiable to make a trial of A.C.T.H. in such cases.

We had found apparent improvement in acute disseminated encephalomyelitis, which shows several clinical and pathological similarities to disseminated sclerosis. Moreover, if an abnormal immunological reaction were responsible for such exacerbations (Pette, 1942) there are theoretical grounds for believing it might be aborted by A.C.T.H. There are similar grounds for believing that A.C.T.H. might also lead to the resolution of the acute inflammatory reaction which was considered by earlier authors—for example, Marburg (1906, 1942)—to be the basis of acute episodes of disseminated sclerosis. If, however, the initial change of the disease is demyelination, possibly as a result of enzymatic disorder (Lumsden, 1951), A.C.T.H. would be unlikely to have any effect once the process had begun, although such a view of the disease does not preclude the possibility that long-term administration of the drug might possibly prevent the development of fresh lesions.

It was felt that by selecting our cases on the above lines it would be possible to make an initial assessment of the effects of the drug on a relatively small number of cases, with a view to a more extended trial if the results were suggestive enough to justify this. It may be remarked that the collection of cases satisfying these criteria proved quite unexpectedly difficult.

In all cases of acute disseminated encephalomyelitis and disseminated sclerosis treated, 25 mg. of A.C.T.H. was given intramuscularly every six hours, usually for a period of five days. Repeated eosinophil counts were made, but in all the cases reported adequate adrenal response was obtained with this dosage.

Acute Disseminated Encephalomyelitis

The three additional cases of acute disseminated encephalomyelitis treated with A.C.T.H. comprise one case of special interest—an example of encephalomyelitis occurring during the course of acute rheumatic fever—and two patients suffering from encephalomyelitis following measles.

Case 1

Encephalomyelitic illness arising during the course of acute rheumatic fever in an 11-year-old boy of low average intelligence. Rapid recovery, beginning within 12 hours of administration of A.C.T.H.

Six weeks before his acute neurological illness this boy developed a febrile sore throat with muscle pains. This cleared up within a week but left him off colour, eating badly, and losing weight. Three weeks after the onset of the initial infection he developed a transient coalescent multi-form erythematous rash, and a week later an acute poly-arthritis with which he was admitted to hospital. At this time he was toxic, but only occasionally febrile, with a pulse rate of 122, a systolic apical murmur, triple rhythm, and partial auriculo-ventricular block with occasional dropped beats. During two weeks in hospital the joint pains were greatly relieved by salicylates, there was only an occasional spike of fever, and the E.S.R. fell from 125 to 41 mm. in the hour (Westergren). However, the child remained ill, and a continuing carditis revealed itself by persisting cardiographic signs, an increasingly loud murmur, and an episode of probable pleuropericardial friction.

Seventeen days after admission to hospital he was observed to be drowsy, and within thirty hours passed through stages of deepening stupor, lighter during the day and deeper nocturnally, to become almost unrousable.

Examination revealed slight neck stiffness, unequal sluggish pupils, spontaneous nystagmus, and a generalized increase of deep reflexes with extensor plantar responses. Within the next six hours he became profoundly hypotonic, with absent deep reflexes, and entirely unresponsive even to painful stimuli. The pressure and composition of the spinal fluid were entirely normal. An E.E.G. showed diffuse slow activity, chiefly in the delta range.

At this stage, 36 hours after the onset of neurological symptoms, he was given 50 mg. of A.C.T.H. followed by four injections of 25 mg. at six-hourly intervals. Improvement was evident within twelve hours. The patient was easily rousable, answered questions, and took food. Neck stiffness was less, the pupils were equal but sluggish, muscle tone was normal, and plantar responses remained extensor. Neurological improvement from this time was progressive. For three days the child was unduly drowsy at night, but at the end of this time symptoms and signs had entirely disappeared and the plantar responses were flexor.

Following this the child developed several syncopal attacks accompanied by extreme bradycardia, with a pulse rate as low as 38, which were improved by ephedrine. The E.E.G., however, showed progressive improvement, though the rheumatic process continued in a subacute form, and within seven days an apical diastolic murmur and involuntary movements typical of Sydenham's chorea made their appearance.

Case 2

Measles encephalitis in a boy of 10. A.C.T.H. given from the seventh day of the neurological illness. Improvement slow but progressive to complete recovery.

Four days after the appearance of a typical measles rash, when this previously healthy child appeared to have recovered, he suddenly became quiet and apathetic. He lay almost motionless thereafter, refusing food. On the third day he became mute and his legs stiff. On the fourth he developed retention of urine, and examination on admission to hospital showed him to be flushed and restless, with meningism, general hyperreflexia, extensor plantar responses,

and a normal spinal fluid. Twelve hours later he was barely rousable, and was febrile—99.6° F. (37.6° C.). The signs were unchanged, except that his stupor was deeper and the legs had become flaccid and the arms spastic.

A.C.T.H. was given at once. Twelve hours later he was more easily rousable but remained mute. Twenty-four hours after the first dose he was making semi-purposive movements with his arms and meningism had decreased. Twenty-four hours later the tendon reflexes became normal, and during the next few days tone in the limbs varied from hour to hour. Improvement from this time was progressive but very slow, and it was not until the fifteenth day of the illness (three days after completion of his five-day course of A.C.T.H.) that facial expression became normally mobile. Two days later the plantar responses had become flexor, and 48 hours after this he began to talk. He remained emotionally labile and inattentive for at least three weeks, and convalescence was prolonged.

Case 3

Severe measles encephalitis in a girl of 3½, with myoclonus, coma, hyperpyrexia, and death within four days of onset, after 36 hours' treatment with A.C.T.H.

Five days after the appearance of measles this child was noticed to be unusually quiet and to have apparent brief lapses of consciousness. During the next two days her condition deteriorated, with increasing drowsiness and occasional muscle-twitching. On examination at this time she was deeply stuporous, but resisted violent stimuli. She was febrile—100.2° F. (37.9° C.)—and generally hypertonic, with hyperreflexia, absent abdominal reflexes, and extensor plantar responses. Repeated myoclonic twitching was present in the left hand and foot. There was no meningism and the spinal fluid was entirely normal. A.C.T.H. and suitable sedatives were administered at once. Twelve hours later stupor had deepened to an unresponsive coma. Convulsive twitching was bilateral and continuous, shifting from one group of muscles to another. During the subsequent 24 hours these movements ceased except in both eyelids, all four limbs became flaccid, and death occurred in hyperpyrexia after an illness of four days' duration. Permission for necropsy was refused.

Comment

Cases of acute disseminated encephalomyelitis in association with rheumatic fever similar to Case 1 have been described by van Bogaert *et al.* (1932). Although these illnesses are clinically similar to para-infective encephalomyelitis of more familiar types, a rather different pathological basis has been described by Costero (1949), who found a diffuse cerebral angiitis of "hyperergic" type in fatal cases of rheumatic fever both with and without cerebral symptoms.

By comparison with Case 1 the two cases of measles encephalitis (Cases 2 and 3) were disappointing, especially as success has already been claimed for the use of A.C.T.H. in this condition (Ligterink, 1951). In Case 2 treatment was not begun until the fifth day of the disease, and, although admittedly the patient gradually recovered from this time onwards, there was no dramatic response to A.C.T.H.: the disease ran a course with which we are familiar in untreated cases, convalescence being protracted. Case 3 deteriorated rapidly in spite of treatment. This may well be considered to fall into the second group of cases which we have considered on theoretical grounds unlikely to respond to treatment—"the rapidly fatal encephalomyelitic illness of which the pathological basis is a diffuse cerebral purpura."

Disseminated Sclerosis

Eight acute episodes of disseminated sclerosis in seven patients have been treated with five-day courses of A.C.T.H., as shown in the Table. Each case satisfied the criteria already described. We had hoped that A.C.T.H. might cause rapid resolution of the acute lesions of the

Details of Cases of Disseminated Sclerosis

Case	Age and Sex	Duration of History	Apparent Provoking Factors	Clinical Features of Acute Episode	Background Disability	Results of Treatment
A1	27 M	2 years	0	Spastic weakness and paraesthesiae in right hand for one week	Spastic paraplegia. Difficulty in micturition. Ataxia	Disappearance of acute symptoms. Relief of spasticity and bladder disorder. No change in reflexes.
A2		2½ "		Exacerbation of weakness of legs	As above	Relief of bladder symptoms only. Euphoria
B	38 M	10 "	0	Acute exacerbation of spastic paraplegia 6 weeks before. Later widespread painful paraesthesiae	Spastic paraplegia. Dysuria. Ataxia	Decrease of long-standing spasticity in right leg. No change in reflexes. Euphoria
C	49 M	6 months	0	Ataxia of gait, with flexor spasms in legs	Bilateral pyramidal signs. Paraesthesiae. Little weakness	No change. Euphoria
D	42 F	17 years	Febrile sore throat one week before	"Useless" left arm for 4 days	Spastic paraplegia. Dysuria. Ataxia	No change
E	33 M	9 "	0	Increasing spastic paraplegia and paraesthesiae for two days	Nil (complete previous remission)	" "
F	31 F	16 "	Common cold 23 days before	Spastic tetraplegia developing during a few hours	Spastic weakness of right leg	Aggravation of symptoms during treatment
G	20 F	4 weeks	0	Increasing weakness of legs for three days. Optic neuritis	Nil	Aggravation of symptoms during treatment. Euphoria (subsequent course of illness confirmed diagnosis)

disease, with concomitant improvement in symptoms. It seemed possible that the control of acute episodes might lessen permanent damage to the nervous system and so improve both immediate and long-term prognosis.

The results were, however, profoundly disappointing. The first patient treated appeared to respond (Case A1). His most recent symptoms—spasm and paraesthesiae in the right hand—resolved during the course of treatment, while bladder control was improved and spasticity reduced without change in the reflex signs. Several weeks later, however (Case A2), a second acute episode in the same patient was characterized by a rapidly developing exacerbation of paraplegia, ataxia, and recurrent bladder symptoms. This exacerbation proved entirely unresponsive to a similar course of A.C.T.H., with the sole exception that dysuria was again apparently improved. In no other case was improvement in acute symptoms observed. One other patient (Case B) showed a definite decrease in long-standing spasticity, while his more recent symptoms remained unchanged. Case G had acute visual impairment, but became dramatically worse during the five days of treatment. The one really striking effect of A.C.T.H., observed in four patients, was the rapid development of marked euphoria, sometimes with extravagant claims of subjective improvement in function for which no objective basis could be found. This euphoriant action is well known, and makes clinical assessment difficult, for a cheerful patient may make much more effort than a depressed one in such tests as those for muscular power.

It will be seen that the only definite benefits derived by these patients are among those enumerated by Glaser and Merritt—decrease in spasticity and improved bladder function. We must conclude from this small group of patients that A.C.T.H., given in physiologically adequate dosage over the course of five days, has no specific effect on the acute manifestations of disseminated sclerosis. There seems no reason to believe that cortisone would be any more effective. The place of A.C.T.H. or cortisone in maintenance treatment, with the aim of preventing fresh lesions, remains to be established.

Discussion

In all we have now treated 10 cases clinically diagnosed as acute disseminated encephalomyelitis with A.C.T.H., and the results are on the whole distinctly encouraging. Two cases followed measles, two varicella, and three respiratory infections; one occurred in association with acute rheumatic fever, and one after body-chilling, while the remaining illness developed apparently spontaneously (subacute necrotic myelitis). In seven patients marked improvement occurred during five-day courses of the drug, and in five instances quite unequivocal clinical improvement appeared within 12

hours of the initial injection of A.C.T.H. The two complete failures were in subacute necrotic myelitis and in the very acute measles encephalitis described above (Case 3)—cases which on theoretical grounds would seem very unlikely to respond. These illnesses tend, of course, to spontaneous and often dramatically rapid recovery, and we cannot yet be certain that the course of the illnesses was in fact altered by the administration of A.C.T.H. Nevertheless the evidence in favour of this view is very suggestive, and may perhaps be regarded as more so when it is observed that improvement occurred within twelve hours of instituting therapy in four of the six patients in whom treatment was instituted within forty-eight hours of the onset of neurological symptoms, but in only one of the four cases in which the symptoms were of longer duration. Such rapid improvement in early cases is entirely in keeping with the hypothesis on which this therapy is based (that acute disseminated encephalomyelitis represents a neurological analogue of urticaria or "anaphylactoid purpura"), and is surely the reverse of what might be expected did our case records represent the natural history of encephalomyelitic illnesses unaffected by therapy.

Consideration of these results suggests two further approaches. Clinically it would be of interest to observe the effects of the more rapidly acting intravenous cortisone or compound F on the severe acute encephalitic illnesses in this group. Secondly, observations on the possible therapeutic action of cortisone or A.C.T.H., as opposed to their verified prophylactic effect, in experimental "allergic" encephalitis might yield further information on what might be hoped for in dealing with the disease in man.

It appears, however, that while most cases of acute disseminated encephalomyelitis show definite clinical improvement during a five-day course of A.C.T.H., a similar course of treatment produces no effect on acute episodes of disseminated sclerosis. This finding permits of two possible interpretations. Either acute disseminated encephalomyelitis responds to A.C.T.H. and acute disseminated sclerosis does not, or neither disease is influenced by the treatment and the differences observed are inherent in the natural history of the two syndromes. Although we believe the second interpretation to be the less likely, it cannot be doubted that the two conditions are distinct. It has never been suggested that post-exanthematous or post-vaccinal encephalomyelitis develops into disseminated sclerosis. Nor have we ever encountered in disseminated sclerosis that dramatically rapid spontaneous resolution of gross neurological signs within hours or days which is a common occurrence in the patient with acute disseminated encephalomyelitis. Furthermore, follow-up studies of apparently spontaneous acute disseminated encephalomyelitis have shown that recurrence is unusual and that the prognosis

after survival of the acute illness is good (Thygesen, 1949; van Bogaert, 1950; Miller and Evans, 1953). That the ultimate prognosis of recovered neuromyelitis optica, which we regard as a variant of acute disseminated encephalomyelitis, is similarly favourable has been clearly shown by Scott (1952).

The differentiation of acute disseminated encephalomyelitis from acute disseminated sclerosis is therefore of much more than academic importance, but it may be very difficult in the acute stage. Both illnesses show signs of disseminated lesions in the neuraxis, both tend to occur in young adults, and both often follow non-specific infections, especially of the respiratory tract. In our experience the diagnostic criteria offered by McAlpine (1931) are invaluable, but the need for further study is demonstrated by the following recent case.

Seven weeks before admission to hospital a young man of 19 developed an acute influenzal illness, epidemic at the time in the Army camp in which he was serving, and associated in his case with quite severe pain in the left eye which lasted for three days. He recovered completely from his symptoms within a week, but seven days later vision in the left eye suddenly became blurred. Examination at this time revealed a unilateral retrobulbar neuritis with papillitis; there were no abnormal signs elsewhere. Sight in the left eye became normal within a week and the patient remained well until one month later, when he developed acute backache, followed by weakness and stiffness of the legs, constipation, and retention of urine. Examination at this time revealed some pallor of the left optic disk, nystagmus at the extremes of lateral gaze, and signs of a partial transverse lesion of the spinal cord in the mid-dorsal area, with loss of proprioceptive sensation in the feet and of impairment of superficial sensibility to the seventh thoracic dermatome. The spinal fluid contained 73 lymphocytes per c.mm., but was otherwise normal. Improvement from this lesion was rapid, and a mild spastic paraparesis with some left optic atrophy was at the time of writing the only evidence of his illness.

Was this a case of rapidly healing biphasic encephalomyelitis, showing some similarity to the syndrome of neuromyelitis optica, and with only slight danger of recurrence? Or must it be labelled as disseminated sclerosis, with all the sinister implications of that diagnosis? The answer to such problems seems less likely to depend on further refinements of clinical diagnosis than on the development of such ancillary aids as are suggested by the immunochemical studies of Kabat *et al.* (1948) or the refined colloidal gold reaction described by Lange and Harris (1949), which may ultimately furnish diagnostic instruments of critical importance.

Summary

Three further cases of acute disseminated encephalomyelitis personally treated with A.C.T.H. are reported, bringing the total of cases treated to 10. In seven patients marked improvement occurred during five-day courses of the drug, and in five of these unequivocal clinical improvement was observed within 12 hours of the initial injection of A.C.T.H. The two complete failures were in cases of subacute necrotic myelitis and catastrophic measles encephalitis. Improvement occurred within 12 hours in four of the six cases in which treatment was instituted within 48 hours of the onset of neurological symptoms, but in only one of four cases treated later in the course of the illness. This finding is regarded as a further indication that the course of the illness was favourably influenced by treatment.

Similar courses of A.C.T.H. were given during eight acute exacerbations of disseminated sclerosis in seven patients. Although euphoria was often present and improvement in chronic spasticity and bladder dysfunction occasionally observed (in keeping with results previously reported), acute symptoms showed no improvement comparable to that seen under similar conditions in acute disseminated encephalomyelitis.

The importance and the occasional difficulty of distinguishing between these two clinically similar but biologically distinct acute neurological syndromes are discussed and illustrated.

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CONSERVATIVE SURGERY IN THE TREATMENT OF ULCERATIVE COLITIS

BY

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There is no doubt that the modern improvements in ileostomy bag design have relieved an ileostomy of some of its terrors, but, in spite of this and the reports of many patients who have returned to most of their former activities, it would be idle and false to pretend that the prospect of an ileostomy for the rest of life is one that does not bring dismay and gloom to those of finer sensibility. Many patients for whom an ileostomy is contemplated or performed are in their youth and in their early married and home life, and few can believe that such distortion of the normal channels of evacuation is likely to prosper that alliance and that household. It is surely right that surgical opinion should not accept the inevitability of permanent ileostomy combined with total colectomy and excision of the rectum as the routine surgical treatment of the disease without the fullest exploration of more conservative methods. Devine and Devine (1948), in a series of cases, have put forward a technique whereby the rectum is preserved and continuity of the intestinal track re-established; and Corbett (1952), working along similar lines, has reported successful cases. Both of these methods entail the full discharge of ileal content through an ileostomy that is retained for varying periods of time, but the method of continuity suggested in this paper, dependent as it is upon immediate ileo-pelvi-rectal anastomosis, carries no such disadvantage.

Those antagonists of any attempt to preserve rectal function contend that the inflammatory changes present in the rectal mucosa continue in spite of removal of the rest of the colon and that the mucosa is unable to withstand the proteolytic effect of the ileal contents which are poured into it. Moreover, they regard the possibility of cancerous change in the remaining portion of the bowel as a very real one, and in the event of existing