

## TREATMENT OF POST-ENCEPHALITIS.\*

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HITHERTO the treatment of post-encephalitis has been profoundly unsatisfactory, and for the most part only palliative.

There are at present in mental hospitals some 200 cases ; this is only a fraction of those not certified, and consists of an asocial class impossible to deal with outside a mental hospital.

It is agreed that cases of encephalitis constitute by far the largest proportion of patients attending neurological hospitals ; also that the distinction of greatest incidence has passed from syphilitic sequelæ (such as general paralysis) to the sequelæ of encephalitis, *i.e.*, from one hopeless disease to another. Since acute encephalitis was made notifiable in January, 1919, the following have been the returns for each year :

1919 . . . . .	541	1923 . . . . .	1,025
1920 . . . . .	890	1924 . . . . .	5,039
1921 . . . . .	1,470	1925 . . . . .	2,635
1922 . . . . .	454	1926 . . . . .	2,267

The certifiable children most nearly approach the old Biblical conception of being "possessed of the Devil." They are a living death to the parents. In this connection Dr. Crafts, in his book, *Epidemic Encephalitis*, very aptly quotes Swinburne in the following lines :

" At the door of life, by the gate of breath,  
There are worse things waiting for men than death."

### ÆTIOLOGY.

According to our present knowledge, encephalitis appears to be an organismal infection, probably gaining entrance through the naso-pharynx. In support of this there is the fact that the acute attack often commences with respiratory symptoms, and that naso-pharyngeal washings from infected cases have yielded a virus capable of producing similar symptoms in animals.

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In 1921, Strauss, in New York, Kling, in Sweden, and several others, claimed to have isolated an ultra-microscopic, filtrable virus which, when injected into rabbits and monkeys, reproduced typical symptoms and pathological lesions. At the annual meeting of the British Medical Association in July, 1927, the production in rabbits of these symptoms was disputed. It was suggested that the pathological results were due to the transmission from rabbit to rabbit of a disease peculiar to them, and only resembling the human disease superficially, *i.e.*, in some of its pathological findings.

Apart from all this, we know that clinically the disease behaves like other diseases known to be bacterial in origin, that there are remissions and exacerbations, and that the morbid process can be retarded.

The pathological lesions in chronic encephalitis are to be found chiefly in the mid-brain (especially the substantia nigra), the thalamo-striate ganglia and rubro-spinal tract.

It has been shown that histologically the foci of infection in many chronic cases are still active, and that even in advanced cases there are seldom signs of actual destruction of nerve-cells. This is a very hopeful and important point, and should influence our outlook on treatment.

Clinically, also, the disease is of a progressive nature; one can see evidences of spread from one part of the brain to another, *e.g.*, we have in this hospital a case who showed the Parkinsonian syndrome on admission, but later developed in addition signs of cerebellar ataxia. It is necessary, therefore, to consider chronic encephalitis as a disease in which the virus is still living and active, and which can at least be restrained.

#### TREATMENT.

Medicinal therapy, with hyoscine, belladonna and numerous other drugs, is as a rule only palliative. Certain distressing symptoms undoubtedly relieved, but something more radical is required. In this hospital we have adopted the following general lines of treatment.

(1) In the first instance, each patient is subjected to a thorough physical examination to detect foci of toxic absorption. Nearly every case requires dental treatment, and in many there is advanced pyorrhœa. Intestinal stasis is also very common, and antiseptic lavage has proved useful.

(2) An attempt is next made to increase the patient's resistance by means of special feeding, tonics, and, in particular, a graduated course of ultra-violet therapy. I agree with Dr. Jaffe's findings,

published in the *British Medical Journal*, December 31, 1927, that such a course results in marked improvement in some cases.

(3) In certain cases liver treatment is being employed. I am aware that it is held that there is no hepatic deficiency in encephalitis, but at the same time there is known to be an association between liver disease and disease of the corpus striatum.

(4) Diathermy is being used in the hope that it may, by increased temperature, kill the germ. It has been used with great success in pneumonia, gonococcal infection, and rheumatic conditions, and also in paralysis agitans. So far it has been found to reduce salivation, and if it proves successful it may be useful to the general practitioner who is handicapped in the use of induced malaria.

(5) In one case an autogenous vaccine, made from organisms found in the naso-pharynx, is being used.

Besides these methods, graduated gymnastic exercises are given to selected patients, with the object of assisting muscular co-ordination and preventing contractures. In those patients who are physically fit, some form of occupational therapy has been found useful in giving an aim and interest, and also in overcoming stiffness of the hands.

Finally we now give each of our cases a course of induced malaria. We began this treatment in July, 1927, the *rationale* being that if encephalitis is, as we believe it to be, caused by a germ, and if that germ is still living and active, and the infection is smouldering in the central nervous system, some form of fever therapy may have a beneficial effect.

Malaria is not the only means of inducing pyrexia. The virus of recurrent fever has been used by Marcus, Kling and Høglund in a series of 18 cases of advanced encephalitis. Marked improvement was claimed in two-thirds of the cases so treated, *i.e.*, with recurrent fever.

There is an obvious resemblance between syphilis and general paralysis on the one hand, and acute and chronic encephalitis on the other. The benefit from malarial treatment in early cases of general paralysis is well known, and in this hospital we have obtained 8 complete recoveries. In giving malaria for both these diseases, we are greatly handicapped by the fact that we seldom receive cases in the early stages.

The technique in our malarial treatment of post-encephalitis is the same as that used in general paralysis.

It is interesting to note that in our experience the patients without exception made a rapid and uneventful convalescence, and

that the debilitating action of the malaria was never alarming. This is in direct opposition to the experience of other workers, who found malarial therapy in these cases to be an unjustifiable method of treatment. I notice, however, that their patients were inoculated directly by mosquitoes, in which case quinine has less efficient control, and the course of the disease is often very different from that resulting from blood transfusion.

Only two of our cases died, the cause in one case being phthisis, and in the other fatty degeneration of the heart. Both died a considerable time after the malarial treatment.

*Results of malarial treatment.*—Eighteen cases have been so treated in this hospital; 12 have shown some signs of improvement, and of these 12, 7 have subsequently regressed to varying extents; 5 have maintained their improvement up to the present time; 4 cases showed no improvement whatever; 2 have only recently been infected, so that the result cannot be judged.

One patient improved so much that he was subsequently discharged to his home "on trial." His mother stated that he was never better in his life. The improvement in his case dated from the time of his malarial attack. The first signs of improvement, according to the patient's own statement, appeared while he was undergoing the rigors.

#### *Summary of the Cases Treated.*

CASE 1.—D. M. C—, female, æt. 22; acute onset 1925.

*Condition before treatment.*—Parkinsonian with epileptiform fits. Dull, lethargic, mask-like facies, eyes always closed; impulsive, negativistic and requires spoon-feeding. Habits very faulty. Salivation very troublesome. Skin greasy.

*Two months after treatment.*—Marked improvement. All actions more briskly performed. Eyes kept open. Patient plays the organ, moves voluntarily, and attends to physical needs. Habits quite clean. Salivation ceased. Skin condition improved.

*Present condition* (8 months after treatment).—Regression marked. Has again become lethargic. Salivation has returned. Habits are still clean. Has developed signs of cerebellar ataxia.

CASE 2.—C. V—, female, æt. 31; acute onset 1920.

*Condition before treatment.*—Parkinsonian. Mask-like face, rigidity, severe salivation, dysphagia. Speech is slow and articulation indistinct. Spastic paraplegia with athetoid movements. Habits faulty.

*Two months after treatment.*—Slight improvement. Less retarded, expression brighter, salivation much reduced. Can swallow semi-solid food. Circulation and skin condition improved. Habits clean.

*Three months after treatment.*—Regression. Moans continually. Swallowing difficult. Progressive emaciation. Died 5 months after treatment from acute phthisis.

CASE 3.—G. M—, female, æt. 28; acute onset 1925.

*Condition before treatment.*—Parkinsonian. Unemotional face. Retardation both mental and physical. Fixed and rigid attitude. Greasy skin.

*Two months after treatment.*—Slight improvement. Expression brighter. Movement less slow. Skin more healthy.

*Present condition* (5 months after treatment).—Improvement maintained. Is mentally brighter; is able to read and to do simple housework.

CASE 4.—E. A. D—, female, æt. 28 ; acute onset October, 1918.

*Condition before treatment.*—Parkinsonian with spastic paraplegia and contractions of both lower limbs (knees almost up to chin). Mentally apparently demented. Chatters incoherently, eyes always closed, tremors of eyelids; has to be spoon-fed. Habits very dirty. Emaciated and anæmic.

*One month after treatment.*—Slight but definite improvement. Opens her eyes answers questions, takes food voluntarily. Habits much cleaner.

*Present condition* (5 months after treatment).—Improvement is progressive and definite. She now reads, laughs and converses rationally. Rigidity is much less, lower limbs almost straight; can walk with assistance. Menstruation has returned. Is steadily gaining weight. Habits quite clean.

CASE 5.—M. E. R—, female, æt. 21 ; acute onset 1926.

*Condition before treatment.*—Parkinsonian. Stuporose. Fixed, expressionless face. Has never spoken one word since admission. Muscles very rigid. Habits faulty. Physical health very poor.

*Three months after treatment.*—Slight improvement. Is more animated and smiles at jokes, but is still mute. Habits clean. Physical health improving.

*Present condition* (4 months after treatment).—Improvement is progressive. She now talks a little, reads the paper and does sewing. She also dances and is much less rigid.

CASE 6.—A. A—, female, æt. 14 ; acute onset 1917.

*Condition before treatment.*—Moral delinquent. Disobedient, untruthful and dishonest. Assaults nurses and patients, and at times is almost uncontrollable. Incapable of any useful occupation.

*Three months after treatment.*—Slight improvement. Is more obedient and can now be employed at basket-making, etc. Physical health improved. No permanent improvement: subsequently became as troublesome as ever.

CASE 7.—S. L. D—, female, æt. 11 ; acute onset 1924.

*Condition before treatment.*—General intelligence above average, but is morally unstable, selfish, deceitful and disobedient. Shows marked respiratory disorder (hyperpnœa), and also has hysterical attacks of screaming, etc.

*Present condition* (8 months after treatment).—Improvement was noticed within two months of treatment, and has been progressive. Breathing is normal, behaviour very much improved and hysterical attacks infrequent. Physical health much improved.

CASE 8.—R. C—, male, æt. 22 ; acute onset 1926.

*Condition before treatment.*—Parkinsonian. Lethargic, unresponsive, and with fixed, mask-like face. Rigidity and salivation marked. Has to be dressed and fed.

*One month after treatment.*—Slight improvement. Salivation less marked. Movements less rigid.

*Present condition* (7 months after treatment).—Regression. Is rigid and almost as helpless as formerly. Salivation very troublesome.

CASE 9.—E. C—, male, æt. 20 ; acute onset 1925.

*Condition before treatment.*—Parkinsonian. Dull, retarded and rigid. Behaviour impulsive at times.

*Three months after treatment.*—Slight improvement. Is less rigid; employs himself in workshops and shows much mental improvement.

*Present condition* (6 months after treatment).—Slight regression. Is lethargic and less inclined to work.

CASE 10.—F. E. C—, male, æt. 23 ; acute onset 1925.

*Condition before treatment.*—Fixed, mask-like face, dyspnœa and hyperpnœa. Behaviour irrational and impulsive.

*Three months after treatment.*—Much improvement. Is brighter and more interested. Conversation rational. Breathing normal.

*Present condition* (6 months after treatment).—Apparently recovered and has been discharged to his home "on trial."

CASE 11.—E. J—, male, æt. 25 ; acute onset 1920.

*Condition before treatment.*—Depressed with acute suicidal impulses. Speech and actions retarded. Rigid, unable to walk; has to be fed and dressed. Athetoid movements. Groans continuously.

*Two months after treatment.*—Slight improvement. Is brighter and no longer groans. Rigidity less and athetoid movements diminished.

*Present condition* (5 months after treatment).—Marked regression. Is acutely miserable and becoming helpless. Has also developed signs of phthisis.

CASE 12.—S. H—, male, æt. 20; acute onset 1927.

*Condition before treatment*.—Mask-like expressionless face. Never speaks and is quite disinterested. Salivation troublesome. Habits faulty.

*Three months after treatment*.—Considerable improvement. He now converses with others, is much less lethargic, and does a little ward work. Habits clean.

*Present condition* (4 months after treatment).—Regression. Is again silent, lethargic and rigid.

The remaining 4 cases showed no improvement and have therefore been omitted.

#### CONCLUSIONS.

(1) In the majority of cases—three-fourths of those treated—there has been some temporary improvement, and in the cases which have subsequently relapsed, regression has not been complete.

(2) There has usually been permanent benefit in some particular direction, but chiefly in wet and dirty cases, who have, without exception, become clean in their habits and have remained clean since their treatment.

(3) Improvement has been noted in the following symptoms and signs: Mental condition, rigidity, tremors, salivation, greasy skin, hyperpnœa and incontinence. Also the general physical condition in nearly all the cases has improved.

In conclusion I wish to thank the Medical Superintendent, Dr. G. H. Grills, for his constant stimulation and valuable advice during the carrying out of these experimental methods of treatment, and for permission to publish the notes of these cases.