Fleming and Miller (1900) reported frank Addison's disease in a mother whose four children were pigmented and subject to attacks of diarrhoea and vomiting.

In all these families where post-mortem records are available the essential finding was atrophy of the adrenal cortex in every case.

I wish to record my thanks to Dr. P. E. Thompson Hancock, of the Royal Free Hospital, for his help in presenting these cases.

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Encephalitis Due to the Psittacosis-Lymphogranuloma Venereum Group of Viruses. A Case of Ornithosis Encephalitis

Evidence that the viruses of the psittacosis-lymphogranuloma venereum group can cause infection of the nervous system was first produced in 1942, when Sabin and Aring described a case of meningo-encephalitis due to the virus of lymphogranuloma venereum. Subsequently, de Wilde (1951) described a case of encephalitis due to the ornithosis virus and Giroud *et al.* (1954) described similar cases.

Although meningeal involvement has been recorded as a post-mortem finding in psittacosis (Walton, 1954) no cases of encephalitis of similar aetiology have so far been described in the British literature.

It is well known that only a small proportion of cases in which virus involvement of the nervous system occurs ever exhibit clinical features of such infection. It is hoped that the presentation of the following case of ornithosis encephalitis will draw attention to this group of viruses as possible aetiological agents in virus infection of the nervous system.

CASE REPORT

A farmer aged 37 was admitted to hospital on December 9, 1954. The history obtained from his relatives was that he had been quite well until some two weeks previously, when he complained of general malaise and had influenza-like symptoms. He continued at work for another week, but then had to remain in bed owing to extreme lassitude. Some few days later there was evidence of deranged mental function in that he began talking to himself and was unable to hold a coherent conversation. His mental condition quickly deteriorated and led to his admission to hospital.

On admission he showed marked psychomotor overactivity, throwing himself about the bed and talking and shouting continually. He was doubly incontinent and would not take food or drink. Physical examination was difficult, but no abnormality was detected other than a slight pyrexia and a tachycardia. A lumbar puncture was performed, the pressure being raised, but it was thought that this was probably due to restlessness. The report was as follows: cells, 1 per c.mm.; protein, 55 mg. per 100 ml.; glucose, 51 mg. per 100 ml.; chlorides, 760 mg. per 100 ml. Haemoglobin was 100%; white blood count, 9,800; blood urea, 74 mg. per 100 ml.—no doubt due to inadequate fluid intake. The blood W.R. was $\pm$ (doubtful), Kahn positive.

There was no change in the patient's condition over 36 hours, and heavy sedation with morphine, hyoscine, and paraldehyde had no effect on the great mental unrest and insomnia. In view of this he was transferred to the psychiatric observation ward.

Again there were no abnormal signs on physical examination apart from some dehydration. The clinical picture was that of an organic reaction. A further lumbar puncture was performed which showed no increase in pressure, and the protein content had fallen to 30 mg. per 100 ml. In view of the extreme psychomotor overactivity he was treated with chlorpromazine hydrochloride, 50 mg. intramuscularly six-hourly, and procaine penicillin, one mega unit twice daily prophylactically. With this treatment an improvement was seen within a few hours; the patient became quieter and less restless although still incoherent. The clinical picture continued to improve rapidly, and it was significant that as consciousness became less clouded he complained of loss of vision.

In view of the history of an influenzal type of illness, the gross psychiatric symptomatology, the slight increase in C.S.F. protein, and the complaint of loss of visual acuity, a provisional diagnosis of virus encephalitis was made. The dosage of chlorpromazine hydrochloride was gradually reduced and finally discontinued after ten days' treatment. Penicillin was also discontinued. Within a fortnight of admission there was no evidence of any physical or mental abnormality. His vision had returned to normal and he was fully ambulant. He was able to give a good account of himself. He gave the information that he was a pigeon-fancier. He could not, however, recall illness in any of his birds.

Blood was sent for complement-fixation tests on December 26—that is, roughly one month after the onset of symptoms. The report was as follows:

Influenza	Q Fever	Psitt. L.G.V.	Aggl. Str. M.G.
Sol. A Sol. B			
<1/8 <1/8	<1/8	$\geq 1/128$	<1/10

A Frei test was negative, and both the blood W.R. and the Kahn test were negative by this time.

He was discharged from hospital some three weeks after admission, completely symptom-free.

On March 16, 1955, he was seen for follow-up interview. He had remained well. The complement-fixation tests were repeated, with the following result:

Influenza	Q Fever	Psitt. L.G.V.
Sol. A Sol. B		
<1/8 <1/8	<1/8	1/64

The Frei test was repeated, but was again negative.

In view of the positive Kahn and doubtful W.R. prior to the administration of penicillin, Price's precipitation reaction and a treponemal immobilization test were performed. Both were negative.

COMMENT

We feel that the falling titre against the psittacosis-lymphogranuloma venereum antigen and the persistent failure to obtain a positive skin reaction against the Frei antigen establishes this as a case of ornithosis/psittacosis encephalitis. Although there was a transient doubtful W.R. before penicillin was given, this was shown to be non-specific.

One of the most striking clinical responses to chlorpromazine hydrochloride is seen in its effect in states of acute excitement (Hamon *et al.*, 1952; Chatagnon and Chanoit, 1953). The response in this case after failure of other sedation is, we feel, worthy of note.

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