Then, again, it was noticed that, in attempting to group such a blood, unsatisfactory results were encountered: the cells of this Group III blood would not properly agglutinate with a definite Group II serum; so that, unless one was alert, a Group III would be classified as a Group I (Jansky) or a Group IV (Moss). This difficulty was never experienced with groups other than Group III. Several instances occurred in which a donor, classified under Group I (Jansky) or Group IV (Moss) was subsequently

TABLE 1.—ORDINARY DIET

	Group II (Serum)	Group III (Serum)
Group I (Cells) ..	A. No agglutination	No agglutination
	B. No agglutination	No agglutination
	C. No agglutination	No agglutination
	Group II (Serum)	Group III (Serum)
Group II (Cells) ..	D. No agglutination	Strong agglutination
	E. No agglutination	Strong agglutination
	F. No agglutination	Strong agglutination
	Group II (Serum)	Group III (Serum)
Group III (Cells) ..	G. Weak agglutination	No agglutination
	H. Weak agglutination	No agglutination
	I. Weak agglutination	No agglutination

TABLE 2.—GREEN-FREE DIET

	Group II (Serum)	Group III (Serum)
Group I (Cells) ..	A. No agglutination	No agglutination
	B. No agglutination	No agglutination
	C. No agglutination	No agglutination
	Group II (Serum)	Group III (Serum)
Group II (Cells) ..	D. No agglutination	Strong agglutination
	E. No agglutination	Strong agglutination
	F. No agglutination	Strong agglutination
	Group II (Serum)	Group III (Serum)
Group III (Cells) ..	G. Very weak agglutination	No agglutination
	H. Weak agglutination	No agglutination
	I. Very weak agglutination	No agglutination

TABLE 3.—GREEN DIET *

	Group II (Serum)	Group III (Serum)
Group I (Cells) ..	A. No agglutination	No agglutination
	B. No agglutination	No agglutination
	C. No agglutination	No agglutination
	Group II (Serum)	Group III (Serum)
Group II (Cells) ..	D. No agglutination	Strong agglutination
	E. No agglutination	Strong agglutination
	F. No agglutination	Strong agglutination
	Group II (Serum)	Group III (Serum)
Group III (Cells) ..	G. Strong agglutination	No agglutination
	H. Strong agglutination	No agglutination
	I. Strong agglutination	No agglutination

* Jansky classification is used. Group IV (Jansky) or Group I (Moss) is not available. The technic is essentially that of Lee and Minot as described in "The Principles of Immunology" by Karsner and Ecker. Letters A, B, C, etc., refer to men of each group.

found to belong to Group III. The error was detected by a direct match with a recipient's blood which was a definite Group I (Jansky) or Group IV (Moss). When this occurred, inquiry always revealed that the Group III subject had been on a diet insufficient in greens. This led to a more or less empiric policy of placing men of Group III on a diet rich in greens before using their serums for grouping purposes. The results obtained were excellent.

In order to arrive at a definite conclusion, three men each of Groups I, II and III (Jansky) or IV, II and III (Moss) were grouped and placed on a green-free diet for a period of four days, and again grouped. These men were then placed on a diet consisting of a large quantity of greens for four days, and grouped again. The results are shown in the accompanying tables.

It will readily be seen that the grouping of the Group III Cells was influenced to a remarkable degree. It cannot be said, however, that there was a definite change of group.

SUMMARY

1. Diet apparently influences the serum of Group III blood.
2. Diet apparently interferes with the proper agglutination of cells of Group III blood in Group II serum.
3. As we were handicapped by lack of sufficient material, the series is, as we realize, entirely too small to form a basis for definite conclusions. However, we consider these findings sufficiently practicable to stimulate investigation along this line.

THE DIAGNOSIS OF EPIDEMIC
ENCEPHALITIS *

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Epidemic (lethargic) encephalitis is one of three important inflammatory, nonsuppurative diseases of the central nervous system which have certain features in common. Each regularly, at least in the more severe cases, affects to some degree the whole cerebrospinal axis and its meningeal coverings; but there is nearly always a maximal involvement of some particular portion which determines the principal symptomatology in any given case. The other two diseases to which I refer are acute poliomyelitis and syphilis. To distinguish the three diseases from one another and from other diseases, it will be helpful to bear in mind a few of their salient pathologic features.

POLIOMYELITIS AND ENCEPHALITIS

Poliomyelitis and encephalitis both affect the interior of the nervous organs more than the meninges, and gray matter more than white matter. In both we have collars of inflammatory cells about blood vessels; but neither tends to cause occlusion of the blood vessels. The meningeal inflammation is slight and only microscopic in both, but more marked in poliomyelitis; hence the greater tendency to meningitic signs, such as Kernig's sign and stiff neck and back, in poliomyelitis. Each disease really is poli-encephalomyelitis, but one principally affects the brain and the other principally the cord. Hence there is little difficulty in distinguishing between the two if we have a pure brain picture or a pure cord picture.

There is a place, however, where brain and cord meet, namely, the medulla oblongata; and with an acute inflammation there, producing an acute bulbar paralysis, we are often at a loss when it comes to making a diagnosis. If the patient should live long enough, the appearance of atrophy, reaction of degeneration, and residual flaccid paralysis of the affected muscles would almost settle the diagnosis in favor of poliomyelitic infection, as the less severe inflammation caused by the encephalitic virus rarely causes sufficient destruction of ganglion cells to produce these sequels.¹ But the patient usually dies in a few days, and as there are no

* Read before the Englewood Branch of the Chicago Medical Society, Nov. 7, 1922.

1. However, localized degenerative muscular atrophy in an unusual case of epidemic encephalitis also complicated by optic neuritis has been described by Julius Grinker: *J. Nerv. & Ment. Dis.* 52: 323 (Oct.) 1920.

distinguishing laboratory tests, we are left to guess. I have seen several such cases; those occurring in summer I called poliomyelitis, those in winter encephalitis, and when I saw one in the fall I just held up my hands. Necropsy was refused, but it is by no means certain that histologic examination would have settled the question. Very likely there would have been too little ganglion cell destruction for typical poliomyelitis, perhaps a trifle too much for typical encephalitis, while the perivascular inflammation which undoubtedly would have been present is common to the two.

Even with the main symptoms relating to the pons and ocular nerve nuclei in the midbrain the same doubt may exist, and only the addition of symptoms referable to the basal ganglia will settle the diagnosis in favor of the encephalitic virus. There have been whole epidemics of such borderline cases, particularly the famous one in Australia, where the nature of the infection remains unsettled. Even animal inoculations on a large scale do not always settle the question, as the disease produced may also be betwixt and between, and the globoid bodies or pleomorphic streptococci likely to be encountered may be claimed for either disease. Neutralization experiments with a known virus might be helpful, but they are laborious and very expensive.

SYPHILIS

Syphilis does not so often produce clinical pictures resembling encephalitis. However, there are a few reports of cases of syphilitic inflammation of the midbrain in which the clinical resemblance was very close.² The most interesting point connecting these two diseases is the apparent existence in encephalitis as well of a "para" stage, i. e., of progressive cerebral disease coming on long after the acute illness, and separated from it by an apparently normal interval. This is particularly true of the postencephalitic parkinsonian syndrome. It is probably not yet known by all physicians that this condition may come on long after the acute illness which often was so mild that it is forgotten, so that it is necessary to ask the patient about it. I have seen many cases in which a diagnosis of previous acute encephalitis could be positively made by the character of the subsequent affection, just as the nature of a doubtful and, at the time, trivial venereal lesion many years later is made clear by the appearance of tabes or paresis. I have now under my care a young woman who had a rather mild acute attack, recovered, married, and had a child. Then she gradually developed a severe parkinsonian syndrome so that she even became unable to feed herself and walk. Another case is that of a stenographer, aged 26, who had a typical and rather severe attack in February, 1919. There was a general tremor at first, and it persisted in the left foot for about half a year. Then she was perfectly well until July, 1921, when the tremor reappeared in the left leg. It was typically parkinsonian, present with the foot at rest, suppressed during voluntary movement. A third case is that of a professional man, aged 38, who came to me a few days ago with a constant side-to-side tremor of one foot, of a few months' standing, of parkinsonian type, and no other symptoms. He told me that he had a mild attack of influenza two years before; he did not have to go to bed, but stayed at home several days because of double vision, which was the first and the most troublesome symptom. Of

course, what he had at that time was a mild acute encephalitis.

If we are not aware that a long normal interval may intervene, we shall make the mistake of regarding these patients as victims of true paralysis agitans. To be sure, such a mistake is not so very serious as the treatment is virtually the same, and in these late forms the resemblance both as to character of tremor and as to progressiveness of course to true paralysis agitans is closer than in cases in which the parkinsonian syndrome develops during the acute stage and persists as a sequel. In these late recurring cases the onset of tremor and rigidity is usually very gradual, and there is no reason to assume that a fresh infection from without has taken place. The view is gaining ground that the virus, like that of syphilis, may remain dormant for years, and then give rise to a more chronic and degenerative process than the one encountered in the typical disease. Necropsy returns from old and prolonged cases begin to come in and substantiate this view. In a case of two years' standing, König³ found in the corpus striatum proliferation of neuroglia and calcareous deposits in the small vessels. In the pons and medulla he found glial proliferation, invasion of ganglion cells by phagocytes, and perivascular infiltrations of plasma cells and lymphocytes. In this country, Globus and Strauss⁴ have described a primarily subacute form in which early diagnosis is extremely difficult. Four such cases were examined postmortem, and the most striking change consisted of proliferation of the adventitia of vessels and organization of new connective tissue and new blood vessels—in other words, changes quite similar to those of the late stages of syphilis. This view, that the so-called sequels are really manifestations of a chronic and often progressive process, has the support of such experienced investigators as von Economo in Vienna and Netter in Paris. The other view has been that the acute disease may leave lesions behind in the basal ganglia analogous to those produced by illuminating gas and manganese poisoning, with the later tissue changes purely degenerative and secondary, and no active virus or toxin operating. Another resemblance between encephalitis and syphilis is the occurrence of the Argyll Robertson pupil. While not common in the former disease, it certainly is encountered.

SEQUELS

Several rare conditions may be sequels of acute encephalitis, and their presence may lead to a retrospective diagnosis of the original infection. I may mention so-called hypophysis syndromes with adiposis, polyuria and genital atrophy,⁵ myasthenia,⁶ progressive lipodystrophy,⁷ and a very oily seborrhea of the face (Salbengesicht).⁸

Certain mental states, especially in children, are found so frequently after encephalitis that their presence should lead to careful inquiries as to a possible acute attack. Thus, Kirschbaum⁹ reports four cases of encephalitis in children followed by a change in personality without mental deterioration. The first three patients showed asocial tendencies, in one case reach-

2. Achard, C., and Rouillard, J.: *Bull. et mém. Soc. méd. d. hôp. de Paris* 45: 130 (Feb. 11) 1921. Guillaumin, G.; Jacquet, P., and Lechelle, P.: *Ibid.*, p. 74.

3. König, Otto: *Ztschr. f. d. ges. Neurol. u. Psychiat.* 75: 221, 1922.
4. Globus, J. H., and Strauss, Israel: *Subacute Epidemic (Lethargic) Encephalitis*, *Arch. Neurol. & Psychiat.* 8: 122 (Aug.) 1922.

5. Stiefler, G.: *Monatsschr. f. Psychiat. u. Neurol.*, August, 1921.
Bertolani, A.: *Riv. sper. di frenit.* 45, 1922. Barkman, A.: *Acta med. Scandinav.* 56: 188 (March) 1922.

6. Sarbo, A.: *Orvosi hetil.* 65, 1921.
7. Sarbo, A.: *Orvosi hetil.* 65, 1921.
8. Stiefler, G.: *Ztschr. f. d. ges. Neurol. u. Psychiat.* 73: 455, 1921.
9. Kirschbaum, Max: *Ztschr. f. d. ges. Neurol. u. Psychiat.* 73: 599, 1921.

ing the degree of actual "moral insanity." The fourth patient presented a hypomanic state, boiling over with activity and happiness. The author is at a loss to explain the relationship between a more or less focal brain disease and the psychic transformation. It is striking that similar conditions have not been reported in adults in whom we find nervousness, depression, disinclination for work, exhaustibility and deterioration, but not this kind of character change.

Psychic sequels in six children are described by Bonhoeffer.¹⁰ In the acute stage there had not been parkinsonian symptoms but delirium, disordered sleep, ocular symptoms and, in some cases, panting respiration. After the recession of the acute phase, the children became talkative, bold, obtrusive, without respect and inhibition and unsocial, with occasional emotional outbursts. There was no hereditary psychopathic predisposition, so the encephalitis itself must be considered the sole cause. No such transformation of the personality was observed in adults, who merely displayed diminution of activity and volition.

Staehelin,¹¹ an assistant in Blueler's clinic, Zurich, has contributed a long article on the psychopathology of the postencephalitic states. A peculiar case of postencephalitic mental disorder in a girl, aged 13 years, observed at the Psychopathic Hospital, was recently presented to the Chicago Neurological Society by Neymann.¹² A normal child prior to the attack of encephalitis a year and a half before, she now was an "inveterate liar, extremely incorrigible, quarrelsome, bit the other children, cuffed them, knocked them down and finally put pins in the tips of her shoes and kicked them."¹³

Kauders¹⁴ describes "moria-like and defective states" as occasional sequels. From the large material of Wagner von Jauregg's clinic in Vienna, he has selected six cases, all in persons from 5 to 17 years of age. Some of these patients showed a typical moria-like mental state with desultory thinking, silly cheerfulness, with fondness for making puns (*witzelsucht*), a condition frequently described in connection also with frontal lobe tumors. In some cases motor restlessness was marked, the picture suggesting the old term "hyperkinetic motility psychosis," coined long ago by Wernicke. Mannerisms and stereotyped movements were also present in these postencephalitic cases. Finally, in several cases a neurasthenic or psychopathic state gradually developed, with interrelation of motor disturbances and psychic peculiarities. Characteristic of the whole group is the organic, neurologic substratum of the psychic manifestation.

Also in adults, conduct disorders indicative of lowering of ethical standards may call attention to preceding encephalitis. One day two women came to my office and related how the husband of one of them had stood up masturbating in front of an open window in full view of the other woman and her daughter across the alley. This had been repeated on several occasions. The wife added that her husband had changed since he had the influenza, had become indolent and shiftless, and had lost his desire for normal sexual gratification.

I sent for the man, and he presented the typical postencephalitic parkinsonian syndrome. He did not have a real psychosis; he was rather anemic, and much discouraged. He rapidly improved when given encouragement, and gave up both exhibitionism and masturbation.

I have dwelt so long on these psychic sequels because I believe they are, as yet, little understood in this community. There are not only postencephalitic pseudoparkinsonians, but also postencephalitic pseudoschizophrenics, even pseudomorons. The possibility of preceding encephalitis should always be looked into. Another advantage to be gained by careful catanenses is the possible recognition of abortive types of the acute illness. These are probably very common, but so far we have lacked criteria for their recognition.

DIAGNOSIS OF THE ACUTE STAGE

The condition of the spinal fluid has been described at length by Kraus and Pardee,¹⁵ and more recently by Eskuchen.¹⁶ The latter summarizes the available data, stating that there are two spinal fluid syndromes which may be considered fairly characteristic:

A. Pleocytosis. Globulin increase. "Luetic" gold curve. Hyperglycorhachis (sugar increase).

B. Cell-globulin dissociation (high cell count in proportion to the amount of globulin.) "Luetic" gold curve. Hyperglycorhachis.

In children, tuberculous meningitis offers great diagnostic difficulty, but in this disease the sugar content is usually diminished, the gold reaction occurs in the higher dilutions, and the cell count is usually higher.

Botulism may closely resemble epidemic encephalitis both clinically and postmortem. Thus, Geiger¹⁷ relates a case from San Francisco in which a clinical and anatomic diagnosis of encephalitis stood unchallenged until *Bacillus botulinus* was isolated from the medulla oblongata.

Tumors of the third ventricle and basal ganglia may be clinically identical with encephalitis, running a febrile course, with rigidity, characteristic tremors, etc. I have reported such a case.¹⁸ The same is true of localized basal meningitis with extension to the third and fourth ventricles.¹⁹

In the early stages I have had difficulty in excluding such diseases as typhoid fever, acute miliary tuberculosis and chorea; and in cases of slow onset with little fever, the differentiation from catatonic dementia praecox, hysteria, and episodes of excitement in the feeble-minded. In one child, even muscular dystrophy has been troublesome. Cases with predominating spinal cord involvement seem to exist and are most difficult, as are those complicated by preexisting chronic nervous diseases, such as cerebrospinal syphilis and multiple sclerosis. Altogether, no disease makes greater demands on the all-around knowledge of the diagnostician than epidemic encephalitis.

15. Kraus, W. M., and Pardee, I. H.: Serology of Spinal Fluid and Blood in Encephalitis, *Arch. Neurol. & Psychiat.* **5**: 710 (June) 1921.

16. Eskuchen, Karl: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **76**: 568, 1922.

17. Geiger, J. C.: *Pub. Health Rep.* **36**: 1663 (July 22) 1921.

18. Basoe, Peter: A Case of Third Ventricle Tumor Mistaken for Lethargic Encephalitis, *Arch. Neurol. & Psychiat.* **4**: 118 (July) 1920.

19. Loeper, M., and Forestier, J.: *Progrès méd.* **36**: 111 (March 12) 1921.

10. Bonhoeffer, K.: *Klin. Wchnschr.*, July 15, 1922.

11. Staehelin, J. E.: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **77**: 171 (June 20) 1922.

12. Neymann, C. A.: Postencephalitic Mental Disorder, *Arch. Neurol. & Psychiat.* **8**: 582 (Nov.) 1922.

13. Important contributions by American observers on this condition have been made by Hohman, L. B.: *Bull. Johns Hopkins Hosp.* **33**: 372 (Oct.) 1922. Leahy, S. R., and Sands, I. J.: *Mental Disorders in Children Following Epidemic Encephalitis*, *J. A. M. A.* **76**: 373 (Feb. 5) 1921.

14. Kauders, Otto: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **74**: 431 (Feb. 16) 1922.

Ophthalmology in Ecuador.—As late as the seventies, ophthalmology was unknown in Ecuador. It was introduced by Dr. E. Gairaud of the Montpellier (France) school, who was appointed dean of the Quito Medical School in 1873. The first cataract extraction was made by Dr. Antonio Falconi in 1878.