

REPORTS
ON
PUBLIC HEALTH AND
MEDICAL SUBJECTS.

No. 11.

ENCEPHALITIS LETHARGICA.



MINISTRY OF HEALTH.

LONDON:
PUBLISHED BY HIS MAJESTY'S STATIONERY OFFICE.

1922.

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FOR OFFICIAL USE.

REPORTS

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No. 11.

REPORT ON ENCEPHALITIS LETHARGICA.

Being an account of further enquiries into the epidemiology and clinical features of the disease; including an analysis of over 1,250 reports on cases notified in England and Wales in 1919 and 1920, together with a comprehensive bibliography of the subject.

BY

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with contributions by

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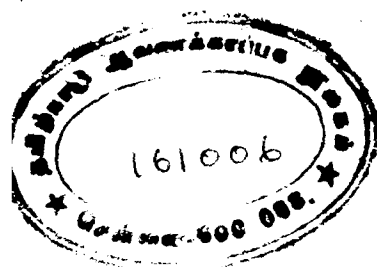
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REPORT ON ENCEPHALITIS LETHARGICA.

TO THE RIGHT HON. SIR ALFRED MOAD, Bt., M.P.,
Minister of Health.

SIR,

1. The Reports on Public Health and Medical Subjects which have been issued by the Ministry since 1919 include important contributions to our knowledge of such well-known and world-wide afflictions of mankind as Syphilis, Plague, Influenza, Smallpox and other diseases. I have now the honour to present a Report concerning Encephalitis lethargica, a comparatively unfamiliar epidemic disease which was first reported from Eastern Europe in the winter of 1916-17 but which has since been noticed in almost every part of the civilised world.

2. While it cannot be said that Encephalitis lethargica has shown any tendency to ravage the peoples of the world or to cause anxiety among administrators to the same extent as some of the long established scourges mentioned above, it is nevertheless true that this strange disease has given rise to a considerable amount of interest and speculation among medical men and, by reason of some of its more striking clinical manifestations, the malady has stirred the imagination of the public at home and abroad.

3. It is worthy of record that in its invasion of this country, the disease had not advanced far before it was challenged and recognised as dangerous at two different points (London and Sheffield) in the first line of public health defence (the practising physician), and the clinical reports of the new disease which were published by observers in April 1918 formed the occasion for a thorough investigation by the Local Government Board which was aided by other medical bodies in this country. This first inquiry was brought to a close in the summer of 1918, not because the disease had disappeared or declined, but because it was thought advisable to make known for immediate guidance all that had been discovered concerning the ætiological, epidemiological and clinical features of the malady and particularly as regards the question as to whether or not it was an independent disease. The results of this initial inquiry, which was conducted by Dr. MacNalty and Colonel James, two of the medical officers of the Ministry, were published by the Local Government Board in 1918.*

4. During and since this first Inquiry the cases were, in truth, increasing in number and the disease was made notifiable as from January 1, 1919; this measure, however, was introduced for the primary purpose of obtaining information and not so much because it was anticipated that the disease would become a public menace requiring widespread sanitary precautions.

* New Series No. 121. "Report of an Inquiry into an Obscure Disease: Encephalitis lethargica."

Having provided ourselves with this most useful instrument (notification) and realising the further help that might be anticipated as the disease became better known among the medical profession, it seemed imperative that we should continue our investigations of this "obscure" malady. It is hardly necessary, I hope, to emphasise the importance of utilising our special facilities for making an extended and intensive study of the natural history of an unfamiliar infective disease, which is so strangely protean as regards its clinical manifestations and so apparently capricious in its epidemic behaviour. Moreover, an intimate inquiry of any one particular clinical syndrome leads to a better understanding of other clinical syndromes or pathological conditions. In the present instance, for example, the close scrutiny of Encephalitis lethargica has led many observers to think that certain forms of *paralysis agitans*, which previously presented many anomalies, may now be considered as part of the symptom-complex of the "new disease." From the standpoint of epidemiology also, the principle holds good; the careful investigations of acute poliomyelitis and of cerebro-spinal fever, which have been made from time to time by the Medical Department of the Local Government Board, have undoubtedly facilitated an understanding of the behaviour of Encephalitis lethargica. Not by frontal attacks on the main position only but by flank movements upon outlying points do we sometimes capture the key to the situation. Knowledge gained concerning one particular infective disease may bring increased wisdom for dealing with infective diseases as a whole.

5. The conduct and scope of the present Inquiry, which was placed in the competent hands of Dr. Allan Parsons, is explained in his introduction to this report and it is appropriate to acknowledge here the valuable service that has been rendered in its publication by Dr. MacNalty, a recognised authority on this disease, by Dr. Perdrau, by the various Public Health Departments throughout the realm, by Hospital Authorities in London and the provinces, and last, but by no means least, by many busy medical practitioners in scattered rural districts and crowded industrial centres, who have voluntarily supplied the clinical data upon which a large portion of the report is based. For the technical information concerning the various, and varying, clinical features of such a disease as this, which is widely but sparsely distributed all over the country, we are fundamentally dependent upon bedside observations. Medical practitioners in their turn, and especially in the case of a comparatively rare and little known disease, have sought and I hope obtained assistance from those who, by virtue of their Public Health or Hospital experience, have had better opportunities for studying the disease in its various aspects. To ourselves has naturally fallen the duty of analysing, weighing and correlating the various reports and returns that have been received and of recording the results of such field-work and

pathological studies as have formed part of the inquiry. The present publication indeed, more than any of its predecessors, is thus a product of concerted thought and labour and a tangible outcome of sound team-work by medical men within the Ministry and outside it.

6. So much by way of explanation of the continued investigations of this disease and the scope and manner of the inquiry. What has this second inquiry achieved and what does the report tell us concerning any such achievements? In general, the extended inquiry which has had reference to the years 1919 and 1920, and this report, which deals with some 1,250 cases, confirm in most particulars the opinions expressed by those who took part in the original inquiry; in addition, some new facts and features have come to light during the last investigations. It is satisfactory to find that the previous view, which were formed as to the essential character of this disease have been confirmed as a result of inquiries lasting over a longer period and dealing with far more material than was possible during the first inquiry.

7. It may be convenient if I draw your attention to some of the more important considerations arising out of the enquiry in the order of subjects followed in the report.

The report consists of four sections, in the first of which the objects of the report and the nature of the material used are more fully explained. Section II. deals with the epidemiology and general ætiology of the disease as studied by means of field-work and the analysis of case reports. In Section III. the symptomatology is dealt with at some length from a statistical and analytical point of view and the case-reports have also been thoroughly searched for all possible information concerning the clinical course, complications and mortality of the disease. Section IV. deals with the pathology of Encephalitis lethargica: Dr. MacNalty provides a useful historical review of the progress that has been made in our knowledge of the virus and of its effects upon the nervous system. Particular reference may here be made to the important advance made by a British pathologist, towards the end of 1919, when Professor MacIntosh succeeded in transmitting the virus of human Encephalitis lethargica to a Patas monkey. This achievement may also be cited as an instance of the value arising out of the co-operation between the Ministry and the Medical Research Council, for the material used by Professor MacIntosh was provided by Dr. MacNalty during his field investigations into the Derby outbreak of August, 1919.

8. There is little calling for comment as regards prevalence, local distribution and seasonal incidence except that there was an unusual rise in the incidence during the summer of 1920. During the first year 541 cases were notified, in 1920 there were 890, and in 1921 this figure rose to 1,470. The winter months

during each year have been those in which the disease was most prominent, but there was a great decline during the winter of 1921-22, when the number of cases fell to 266 as compared with 1,193 for the corresponding period in 1920-21.

9. The distinction between Encephalitis lethargica and both botulism and acute poliomyelitis has been emphasised as the result of the enquiry. Further, no evidence of any clear and direct epidemiological or clinical relationship with influenza has been discovered. A personal history of recent influenzal attacks was only obtained in 8 per cent. of some 1,250 case reports. In passing I may say on the subject of differential diagnosis that among young subjects tuberculous meningitis and among older patients cerebral tumours or cerebral hæmorrhage are the conditions most frequently confused with Encephalitis lethargica. The clinical picture drawn in the report is clear and graphic. During the winter of 1920-21, and at a time when encephalitis was prevalent, there was a curious epidemic of hiccough, but our inquiries in this country failed to discover the close relationship between the two conditions that seems to have been established on the Continent. Again, the accounts in 1921 of various epizootic outbreaks among dogs and cats was inquired into (in this country and South Africa), but no correlation could be found between these outbreaks and epidemic human encephalitis. The opinion formed by the Veterinary Department of the Ministry of Agriculture and Fisheries was that the animals in each outbreak in this country were affected with "gastric distemper."

10. Despite the valuable statistics available nothing definite could be deduced regarding any conditions that might dispose an individual to an attack of Encephalitis lethargica. The disease appears, however, to have selected more particularly those of either sex following sedentary occupations, while, as regards age incidence the period 10-20 years has proved to be the most susceptible. A considerable amount of information concerning encephalitis and the pregnant state has been collected from our series of English cases, and Table A. in Appendix II. contains perhaps more data on this subject than any hitherto published. It would appear that the mortality from encephalitis among pregnant women is no higher than that among women not so circumstanced, nor was the course of pregnancy unusually affected where the mother survived her attack of encephalitis. The occurrence of four cases of "encephalitis neonatorum," however, is of peculiar interest.

11. The means and mode by which the infection of Encephalitis lethargica is naturally carried from person to person remain obscure; very seldom can an attack of Encephalitis lethargica in any individual be traced to the patient's contact with an obvious case of the disease. The Derby outbreak of August, 1919, investigated by Dr. MacNalty and reported upon in my

Annual Report for that year, remains without parallel and no such explosive outbreak in localised communities has since occurred. In Table B, Appendix II., have been collected notes of all the instances in which case-to-case infection possibly took place. The *hypothesis* of the "healthy carrier," put forward as the result of epidemiological and clinical observations, however, has recently received striking support from experimental workers and may now be replaced by the definite *theory* that, most frequently, Encephalitis lethargica is chiefly conveyed by persons who show no clinical signs of the disease, and in other instances through the agency of mild cases.

12. The symptomatology of Encephalitis lethargica receives considerable attention in Section III. of the report, and it is evident that a more comprehensive clinical concept of the disease has become necessary in order to include the mild and typical forms that have now become manifest amid the greater mass of cases. The present report again shows abundantly, however, how common is the occurrence of that classical combination of symptoms (fever, lethargy and ocular palsies) that serve as the primary diagnostic factors in the differentiation of Encephalitis lethargica from other diseases. Among the clinical features that have received special attention are the possibility of sudden onset, the occurrence of signs suggestive of both irritative and paralysing lesions and the question of optic neuritis in relation to lethargic encephalitis.

13. The disease must be accounted a dangerous one for the individual, but, as pointed out in the report the case-mortality of notified cases (approximately 48 per cent.) is inflated, for the obvious reason that the number of notifications does not represent accurately the incidence of the disease. There is some reason to think, however, from evidence collected since 1920, that the disease generally is assuming a milder and less fatal form.

14. The report lays stress on the general absence of complications in Encephalitis lethargica and contrasts this with influenza. As regards so-called relapses and sequelæ we may venture to believe that the repetition or continuance of symptoms may be due in large part at least to the continued action of a long-resident or protracted virus. Certainly, a study of Table E in Appendix II. indicates that the psychical and nervous disorders which mar or postpone the recovery of so large a number of patients resemble very closely the clinical features of the acute disease. It should be added that loss of memory and profound changes in the disposition and character of patients who suffered from the disease are not infrequent sequelæ and have an interest from a medico-legal point of view.

15. It is obvious from what has been said about the mode of dissemination that no very definite lines of prophylaxis can be laid down until we have means of determining the individuals who are the unconscious reservoirs of the virus, and provisionally

the section on preventive measures seems to supply all that can be said on this subject. It does little more than repeat the action suggested in the memorandum issued by the Ministry last year.

16. It would be an incomplete Inquiry that did not include as an all-important part of its work a study of the underlying morbid processes of the disease in question, and in this instance Dr. Parsons was associated with Dr. C. Da Fano, Lecturer on Histopathology at King's College, London, and Dr. J. R. Perdrau, Pathologist of Lambeth Infirmary, who undertook the pathological investigations. We are, in part, indebted to the Medical Research Council for the services of Dr. Da Fano and no less to Dr. Da Fano himself for the time he freely devoted to histopathological examinations. The microscopical examination of the cerebral nervous system of the rabbits used in the course of Dr. Perdrau's experiments has been undertaken by Dr. Da Fano and will form the subject of a separate communication published by himself. He has also in preparation a description of the histological appearances found in human cases of the disease, which is based largely upon the examination of material procured during the joint Inquiry. It is to be regretted, that on grounds of economy, it has not been possible to incorporate with this report the skilful and finely illustrated work of our colleague. Special thanks are also due to the authorities of Lambeth Infirmary, where Dr. Perdrau was enabled to carry out his experimental work, the results of which are embodied in this report. As a result of his investigation of the cerebro-spinal fluid in encephalitis and other conditions with which it may be confused, Dr. Perdrau considers that examination of the fluid withdrawn by lumbar puncture is an important aid in diagnosis, for the reasons explained in his section of the report. In the experiments with the virus of Encephalitis lethargica and of herpes, Dr. Perdrau confirmed some of the results obtained by Levaditi, Harvier and Nicolau. It is significant to note, also, that he succeeded in immunising a rabbit by simple inoculation of the skin with the living virus.

17. After all that has been said, however, it is probable that this report will prove of greatest value as a storehouse of statistical information, and in the large number of pages devoted to the appendices is safely garnered a wealth of facts and figures such as I do not think have been collected in any other similar report. The extensive bibliography and subject-index are new features in these reports. Not often, however, are we present at the birth of a new clinical concept, and it seemed both proper and profitable that an attempt should be made to build up a bibliography of Encephalitis lethargica *ab initio*. Though the report deals almost exclusively with Encephalitis lethargica as observed in England and Wales, its value is enhanced by the fact that the authors have constantly marshalled their facts alongside those collated by other observers abroad. The report,

therefore, while chiefly intended for the information of those in this country who have already shown their interest in the subject by providing much of the raw material of which it is composed, may also make an appeal to a wider fellowship of medicine and serve as a general work of reference to all in search of information upon this particular subject. I hope this may be the case, for the inquiry here reported represents, in extended degree, an effective co-operation between the Ministry of Health and the medical practitioner in the elucidation of new knowledge.

I have the honour to be,

Sir,

Your obedient Servant,

GEORGE NEWMAN.

WHITEHALL,
August, 1922.

SECTION I.—INTRODUCTION.

"Having met with no less than seven cases within a fortnight, I wish to draw attention to a disease which is little known, though dangerous and difficult to treat. The cases were scattered over London between Wandsworth, Clapham Road, Marylebone, Paddington, Harlesden and Palmer's Green."

WILFRED HARRIS.

"During the last two or three weeks a series of cases presenting somewhat remarkable features has come under my notice in this district (Sheffield)."

ARTHUR J. HALL.

Thus, four years ago, and in the same number of the *Lancet* (20th April 1918) was attention first drawn by two independent observers in England to the disease now known as Encephalitis lethargica.*

Since these two preliminary notices appeared, much has been written, both, at home and abroad, concerning encephalitis lethargica, and it is doubtful whether any disease in recent times has in such a short period gathered around itself a literature so cosmopolitan and so extensive.

The main object of the present contribution is to set out in order the various items of information gathered from official reports concerning some 1,300 cases which were notified to the Ministry during the years 1919 and 1920; before attempting this, however, it is advisable to give a short account of previous

* The name "Encephalitis lethargica," given by Von Economo to the disease as observed first by him at Vienna, has received much adverse criticism and many alternatives have been suggested. The only serious rival, however, would seem to be "Epidemic encephalitis," under which term the disease is described in the last edition of Osler's text-book; this expression also occurs frequently in the *Index Medicus*, though not to the same extent as the original name.

In this report we shall follow our predecessors and speak always of "Encephalitis lethargica"—a name which can claim the right of primogeniture and the fortune of illustrious parentage; moreover, the concept is clothed in that language which is common to scientists of all countries and partly, perhaps, for euphonious reasons the term, "encephalitis lethargica" seems to be standing the test of time.

(An American writer has thrown some light on the word "nona," the use of which was formerly advocated by some writers because the word is short and makes no attempt at embracing any clinical considerations. It appears that in 1890, when the African sleeping sickness first came into prominence, there was an epidemic of lethargy among the silkworms of Southern France and Northern Italy—Italian, "nona"; French, "nonne" = silkworm. There was, at the time, some discussion as to a possible common cause of this "malattia de la nona" and human sleeping sickness, and some confusion in the use of the term. "Malattia de la noaa" became abbreviated to "nona," and as such was then applied to the mysterious epidemic which involved many people in N. Italy, Hungary and other parts of Europe in 1889-90.)

official investigations and to discuss, briefly, both the methods used in the present inquiry and the material furnishing the report.

(A) BRIEF ACCOUNT OF PREVIOUS INVESTIGATIONS BY THE LOCAL GOVERNMENT BOARD.

It was early remarked that the clinical manifestations of the "new disease" bore a certain resemblance to those caused by the *Bacillus botulinus* of Van Ermengem, and the conditions that prevailed during the late phases of the war certainly gave plausibility to the suggestion that contaminated food was responsible for the cases that occurred during the early part of 1918. The prompt and exhaustive enquiry which was undertaken jointly by the Local Government Board, the London County Council and the Medical Research Council failed, however, to establish any connection between food infection and the disease in question. This negative finding, which was important in itself, stimulated further curiosity, and it was then suggested by certain authorities that the malady should be included in the Heine-Medin group of diseases; on the other hand, many observers were not satisfied with this suggestion, so a second and more comprehensive inquiry was instituted.

In this inquiry the London County Council was again associated with the Local Government Board, and an effort was made to obtain the fullest epidemiological and clinical information regarding those cases of the disease which were brought to light during the first half of the year 1918.

Dr. A. Salusbury MacNalty (Ministry of Health), who conducted the clinical side of the inquiry, was able to confer constantly with the late Sir William Osler (who at first was of opinion that the new syndrome belonged to the Heine-Medin group), while Major Draper (U.S.A.), with his unique experience of acute poliomyelitis, also helped in the inquiry and personally investigated some twenty cases. At the same time, and in conjunction with Dr. MacNalty, Lt.-Col. S. P. James (Ministry of Health) carried out the epidemiological part of the inquiry, and his consequent report was the first of its kind to be published. No less fortunate was the continued collaboration with the Medical Research Council, and on this occasion Professor Marinesco was associated with Dr. (now Professor) McIntosh in the pathological investigations.

The information gathered from these clinical, pathological and epidemiological inquiries was embodied in a Report to the Local Government Board (New Series, No. 121) entitled *Report of the Inquiry into an obscure disease, Encephalitis lethargica*. All the contributors to this report formed the opinion that the disease they had been studying is not identical with acute poliomyelitis, but is a separate condition embracing the same clinical features as the disease previously reported from Vienna and called by Von Economo *encephalitis lethargica*.

Finally, it may be noted that, largely on account of the findings of this report, *Epidemic encephalitis* (encephalitis lethargica) has been admitted as an independent disease to the ninth edition (1920) of Osler's *Principles and Practice of Medicine*, and this recognition goes far to establish the credentials of the "new disease." Such, in brief outline, is an account of the official steps taken in 1918 to track down and study an "obscure disease"; our comparatively simple task has been to follow in the trail so clearly blazed by the early explorers and to present a further account of the disease as studied during the years 1919 and 1920.

(B) MATERIAL AVAILABLE AND METHODS OF INQUIRY.

1. **Notification.**—In the concluding paragraph of his preface to the report issued in 1918, Sir Arthur Newsholme wrote, "The first necessity for these (further) studies is early and complete information of all cases of the disease in different parts of the country, and for this reason it has been decided as a temporary measure to make encephalitis lethargica compulsorily notifiable for a period of one year throughout England and Wales" (739). This measure was carried out, and by a general Order dated 17th December 1918, and entitled *The Public Health (Acute Encephalitis Lethargica and Acute Polio-Encephalitis) Regulations, 1916*, encephalitis lethargica was made a notifiable disease as from 1st January 1918.*

This, then, was the first step which was taken in order to facilitate the continued investigation; it may be mentioned at the same time that, in the preliminary inquiry, the investigators were dependent for clinical details upon more or less voluntary contributions, though a greater proportion of cases were personally visited and investigated than has been possible in the subsequent inquiry.

In the Regulations quoted above, it will be noted that poliomyelitis is included (poliomyelitis has been a notifiable disease since 1st September 1912), and this was done in order to secure the notification of cases clinically intermediate between poliomyelitis and encephalitis lethargica. It may be added that, besides those of poliomyelitis and polio-encephalitis, the notifications of cerebro-spinal fever also were considered together with those of encephalitis lethargica, because it was thought that the arbitrary grouping together of these four epidemic diseases might help to elucidate some of the epidemiological characteristics of encephalitis lethargica.

* A whole year elapsed before the English example in this matter of compulsory notification was emulated abroad. Among the earliest countries to adopt the measure may be mentioned Prussia, Italy and Switzerland, all of whom framed regulations in February, 1920; in the case of Prussia, these were revoked in the following month. Portugal, Spain, Brazil, Uruguay, some of the States of N. America and the Union of South Africa, are among those which have since followed suit; in France and Belgium, the measure has been advocated but not adopted.

The numbers of cases of these four diseases reported during the period under discussion are shown in the table below.

TABLE 1.

Year.	Encephalitis lethargica.	Polio- encephalitis.	Polio- myelitis.	Cerebro-spinal fever.
1919 -	541	62	553	848
1920 -	890	36	293	582

The figures above represent the official notifications published week by week, corrected monthly, and issued finally in yearly returns by the Registrar-General's Department, and as such they formed the primary data upon which detailed work was begun. As a result of further information, it was inevitable that the diagnosis should be amended in some instances, and the figures in the table above, though approximately correct, do not coincide with those used for analysis.

Thus, by means of various checks, the figures for encephalitis lethargica (1919 and 1920) have been reduced to 1,273, and it is upon the analysis of these cases that this report has, for the most part, been based.* In the matter of multiple cases occurring under one roof, the effect of encephalitis lethargica on pregnancy, and in one or two other instances, however, advantage has been taken of material that has become available during 1921 in order to make the conclusions more valuable.

2. Schedule Reports.—The routine method used for obtaining further information about notified cases was by means of questionnaire papers, or "*schedules of inquiry*." These schedules were sent in by the Medical Officer of Health after he had obtained the usual nominal details, together with information concerning the circumstances of the case and the symptoms displayed by the patient.

The clinical data contained in these reports has been supplied voluntarily by the medical practitioners in attendance, either in the course of private practice or from hospitals, infirmaries and other medical institutions. To all such collaborators a large meed of thanks is due, for without their help the present inquiry would have been comparatively fruitless.

The report forms were prepared for, and where possible used during, the first inquiry; and since their inception, over three years ago, it has been unnecessary to do more than slightly modify the original scheme of questions.

* In addition to the analysis of 228 cases by MacNalty (629a), other analyses that may be mentioned are:—Alexander and Allon (100 cases); Boyd (75 cases); Dunn and Hagey (115 cases); Neal (274 cases); Smith (181 cases); Strauss and Weschler (864 cases).

Thus, under heading 10, specific information was, for some time, sought concerning fever; lethargy; eye palsies; facial paralysis; paralysis or paresis of skeletal muscles and of sphincters; mental condition; delirium, tremors, &c.; speech changes; condition of fundus oculi and rashes. At the beginning of 1921, however, it was decided to omit all the sub-headings of section 10, and space is now left in which the clinician can record the salient features of the case. The complete form as used at present is here shown:—

MINISTRY OF HEALTH.

To be filled in at { Notification No.....
the {
Ministry of Health { Serial No.....

ENCEPHALITIS LETHARGICA.

Sanitary District.....County.....
.....Medical Officer of Health.
.....Officer making Inquiry.
.....Date of Inquiry.

SCHEDULE OF INQUIRY.

In enquiries into cases of disease notified as encephalitis lethargica, it is requested that the following Schedule may be completed as far as possible and returned to the Medical Officer (Med. 1), Ministry of Health, Whitehall, S.W.1.

1. Name and address of patient.....
2. Age and sex
3. Occupation.....
4. When last at work.....
5. Date of onset of symptoms.....
6. Date of notification.....
7. Name and address of medical attendant.....
8. If removed to hospital, state where and when.....
9. Initial symptoms
10. Salient symptoms and signs of the disease and further progress of the case. (The assistance of the hospital medical officer or other medical practitioner treating the case is specially desired for information under this heading.)
11. If fatal, state date of death.....

12. Note any recent illness or condition which may have lowered the patient's resisting power or otherwise predisposed him to the present illness.....
13. Number of persons in the family.....Number of occupants of the house.....
14. Name any recent illness or indisposition among them, which may possibly be regarded as an abortive case :—
.....
15. Note whether any history of sore throat, influenza, cerebro-spinal fever, poliomyelitis or other recent illness in patient or member of family.
.....
16. Insanitary condition of the house (e.g., overcrowding, &c.) :—
.....
17. Name any illness or indisposition known to have occurred among persons with whom the patient has recently been in contact, and when :—
.....
18. How many other cases of a similar nature have you observed in the district ?
.....
19. Results of Examination of cerebro-spinal fluid made.....
.....1921 :—
 (a) Physical.....
 (b) Chemical (including where possible figures as to chlorides and sugar)
 (c) Microscopical
 (d) Bacteriological
.....
20. Results of any blood tests or bacteriological examinations :—
.....
- 21 P.M. examination if made, by whom, and date.....
22. Remarks :
.....

Medical Officer of Health.

Date.....

It should be added that, to ensure uniformity, the forms adopted by the London County Council are replicas of those used at the Ministry; similar forms, with the necessary modifications, have also been adopted by the Army Medical Department. Arrangements, too, have been made with the newly-created Welsh Board of Health, so that copies of similar schedule reports on cases of encephalitis lethargica in Wales are sent to the Ministry as occasions arise.

Somewhat similar schedule report forms concerning poliomyelitis, poliomyelitis and cerebro-spinal fever, which had been in use synchronously with those for encephalitis lethargica, were suspended at the beginning of 1921, as it was considered that sufficient information of the kind asked for had been received and that no useful purpose would be served by continuing to ask for such reports.

3. Annual Summaries.—In order to ensure, as far as possible, that the information previously received from the various districts concerning each case of encephalitis lethargica remains correct, Medical Officers of Health have been requested to fill in and return special tabular forms relating to encephalitis lethargica and the three other diseases considered with it.

These forms, in the first place, were sent out from the Ministry, partially filled in with the nominal details of all cases notified during the year, district by district. Such details are checked, and often amended, by the Medical Officer of Health, who adds any information he can collect concerning the after history of the patients concerned. Moreover, the tables are so arranged that the cases become sorted according to their age and sex groupings, whereby the subsequent statistical analysis is greatly facilitated.

4. Mortality Returns.—Special arrangements, too, have been made with the Registrar-General's Department so that weekly and monthly lists which give particulars concerning all deaths certified as due to encephalitis lethargica have been available; these lists have proved very useful as control agents.

5. Semi-official Correspondence.—But, apart from these usual sources of information, a great deal of semi-official correspondence has been carried on with the medical officers of various medical institutions in order to supplement the information in the case of reports concerning patients who, in the course of their illness, were removed for treatment to some institution. In such cases the history of the illness as supplied by the original medical attendant is necessarily incomplete, so that the information concerning later symptoms and the special examinations conducted in the hospital, laboratory or post-mortem room, which has been so kindly furnished by various hospital authorities, has been of great value.

6. **Field Work and Personal Inquiries.**—During the period under review numerous field inquiries have been carried out in districts where the number or the special circumstances of the cases seemed to call for further investigation, and in this connection may be mentioned South Wales (Dowlais and Merthyr Tydfil), Birmingham, Calne (Wilts), Warwick, Bristol and Moreton Hampstead (Devon). The requests for personal consultation over individual cases have been met as far as possible; but, with the general spread of information concerning the disease and the increasing experience now available at most large centres, the need for such clinical consultations has become less and less.

In addition to seeing various cases in London hospitals and in the districts previously mentioned, our clinical experience also embraces visits to Stratford, Cannock, Acton, Earlsfield, Wimbledon, Hounslow, Hanwell, Plymouth, Worthing, Bishop's Stortford, Lewisham, and other places.

7. **Pathological Investigations.**—From the first the collaboration with the Medical Research Council as regards aetiological investigations has been kept up, and the high-water mark of attainment resulting from this co-operation was reached toward the end of 1919 when, with material from one of the now celebrated Derby cases, Professor McIntosh succeeded in transmitting encephalitis lethargica to a Patas monkey.

In June 1920, on the recommendation of the Medical Research Council, the services of Dr. C. Da Fano, Lecturer in Morbid Anatomy and Histology at King's College, were secured in order that expert assistance in post-mortem work should be available when such was thought desirable; Dr. Da Fano also took in hand the detailed histo-pathological examination of such material as had been accumulating. At this time, too, the assistance of the Colonial Office was invoked, with a view to obtaining some post-mortem material from East African cases of human trypanosomiasis for the purpose of control examinations. The nature of the specimens required and the manner in which it was desired they should be collected, labelled and packed, were set out in a memorandum which was forwarded with a covering letter to the Colonial Office in September 1920.

In the meantime, it was considered advisable to supplement the special histological work being carried on by Dr. Da Fano by detailed investigations of the cerebro-spinal fluid obtained from cases of encephalitis lethargica, and by a bacteriological study of the disease involving animal research work.

Moreover, it was felt desirable that the pathologist responsible for this portion of the pathological work should have easy and natural access to clinical facilities in order that control investigations might be carried out in the case of associated nervous diseases. These desires were realised when, towards the end of 1920, Dr. J. Perdrau, Pathologist to Lambeth Infirmary, became associated with Dr. Da Fano, and began to co-operate in

the enquiry generally. At Lambeth Infirmary itself a considerable amount of clinical material has come under review, so that Dr. Perdrau has been able to follow closely and continuously the pathology of cases treated in the Infirmary wards and to avail himself of the facilities for laboratory research also provided by this institution. Unfortunately, the arrangements for experimental research could not be completed before March 1921, by which time there was a marked drop in the incidence of encephalitis lethargica. Meanwhile, by means of various circular memoranda issued by the Medical Department, Medical Officers of Health were kept informed of the arrangements being made for the pathological side of the inquiry. Through such means and by many personal reports, also, the co-operation of all who were in a position to help was invited, with a view to our being kept informed of any opportunities for securing, at first or second hand, material suitable for research work. In London the Medical Superintendents of several Fever Hospitals, Infirmarys and Poor Law Institutions kindly gave special facilities and kept Dr. Perdrau informed of the admission into their wards of any suspected case of encephalitis lethargica. Of cases notified, also, in or near London, one of us was kept specially informed through the Registrar-General's Department, by means of lists sent in advance of the usual weekly publications.

Through these means a certain amount of material has been obtained and forms the subject of Dr. Perdrau's reports in Section IV.; it is most unfortunate, however, that the completion of the arrangements necessary for the experimental research synchronised with a relative disappearance of the disease, so that material from acute, undoubted, cases has, so far, not been easily obtained.

8. **Exchange of Information with Foreign Countries.**—Through the medium of the British Delegate on the Committee of the *Office International d'Hygiène Publique*, we have been able to give and receive information concerning encephalitis lethargica and its epidemic behaviour respectively in this country and abroad, more especially on the continent. To Professor Netter of Paris, Dr. Simon Flexner of New York, Dr. J. H. H. Pirie, South Africa, and Dr. C. B. Rossiter, New Zealand, thanks are due for personal communications regarding the disease in their respective countries.

9. **Literature.**—It has not been possible to keep thoroughly in touch with all the references to encephalitis lethargica which now abound in the home and foreign press. Reports of cases in the British medical journals, however, have often proved useful by supplementing the details already supplied by the schedule reports, while in some instances they have disclosed the occurrence of cases not officially notified. The daily press, too, at one time gave considerable attention to "sleeping sickness" and

reported the occurrence of some suspicious epidemics among animals which might, but for this, have escaped our notice.

A comprehensive bibliography of encephalitis lethargica will be found at the end of this report.

SECTION II.—EPIDEMIOLOGY AND ÆTIOLOGY.

(A) EPIDEMIOLOGY.

1. **Prevalence.**—A disease which, in these islands, affects only 0·02 per 1,000 of the inhabitants can scarcely be considered as prevalent in the strict meaning of the word; and in the comparative table (for 1920) below, it will be seen that encephalitis lethargica is not prominently placed, but shares, with cerebro-spinal fever, poliomyelitis and smallpox, a position roughly midway between the more or less prevalent and the distinctly rare notifiable diseases.

TABLE 2.

Disease.	No. of Cases notified.	Rate per 1,000 of population.
Ophthalmia neonatorum - - -	10,304	10·76 (Rate per 1,000 births).
Scarlet fever - - - - -	119,490	3·19
Diphtheria - - - - -	69,481	1·86
Tuberculosis (pulmonary) - - -	61,655	1·65
„ (other forms) - - - - -	15,851	0·42
Pneumonia - - - - -	38,859	1·04
Erysipelas - - - - -	16,051	0·43
Enteric fever - - - - -	3,109	0·08
Puerperal fever - - - - -	2,898	0·08
Dysentery - - - - -	1,353	0·04
ENCEPHALITIS LETHARGICA - - -	890	0·02
Cerebro-spinal fever - - - - -	583	0·02
Poliomyelitis - - - - -	293	0·01
Smallpox - - - - -	263	0·01
Trench fever - - - - -	39	0·00
Polio-encephalitis - - - - -	36	
Continued fever - - - - -	35	
Malaria (contracted in England) - - -	32	
Relapsing fever - - - - -	7	
Plague - - - - -	3	0·00
Typhus - - - - -	2	

There is reason to think that though labelled differently, the clinical syndrome belonging to cases of what is now known as encephalitis lethargica has been observed occasionally in this country for some time past. It was in the spring of 1918,

however, that attention was first focussed upon cases and collections of cases in which fever, lethargy and ophthalmoplegia were the cardinal symptoms. Since that time the cases have become universally distributed over the country; occasionally they have occurred in local groups, and at certain seasons the number of cases have greatly increased in number. Whatever its antecedents may really be, the disease has now in this country, as in the world generally, assumed epidemic characters, and this would seem to be a new experience.

In 1918 an investigation was made of some 230 cases which had occurred in England and Wales during the first six months of that year. In 1919 the disease was made notifiable as from the first of January, and the cases notified since that date to the end of 1921 are shown in the following table:—

TABLE 3.

Quarter.	1919.	1920.	1921.
First - - - - -	286	264	952
Second - - - - -	70	224	244
Third - - - - -	64	161	144
Fourth - - - - -	121	241	130
Totals - - - - -	541	890	1,470

It will be observed that each year sees a greater number of cases notified; and, allowing for increasing vigilance (though mild cases as yet are rarely notified) and greater experience of the disease, it is reasonable to think that the disease has yearly become more prevalent. The large proportion of cases that are notified in the first quarter of each year is also a noteworthy feature which, with others, is shown more graphically in the chart of seasonal incidence (*q.v.*).

2. **Local incidence in the British Isles: England.**—The first cases of encephalitis lethargica were reported from London and Sheffield simultaneously, and for the two initial quarters of the year 1918 the disease, in England, seemed to be confined chiefly to the eastern counties and the midlands, cases occurring chiefly in and around London and at other large centres; the disease then appeared further afield, and by the end of the year the West of England was involved. Attempts to find an analogy between this course of events on a small scale with the Western trend of the disease through the world generally cannot, however, be fully sustained; thus one of the very earliest cases was reported from Bangor, another was noted in Monmouth

during April 1918, and as early as May 1918 the disease had appeared in Belfast. Not by any steady advance in one direction, therefore, but in a seemingly haphazard way has encephalitis lethargica completed its invasion of this country, and the manner in which the disease here has skipped from place to place suggests in miniature some of its jumps from country to country which have been observed on the continent.

In its incidence the disease has become widespread, so that, in England, cases have now been reported from every administrative county area; the general distribution of cases notified in England and Wales during 1920 is shown in the following table:—

TABLE 4.

Total number of cases after correction.	London.		England, excluding London.				Wales.			Port S.D.
	Boroughs affected.	Cases.	Urban districts affected.	Urban cases.	Rural districts affected.	Rural cases.	Urban districts affected.	Urban cases.	Rural districts affected.	Rural cases.
844	25	133	233	557	85	100	21	41	8	12
										1

It will be noticed that the disease has been reported chiefly from urban districts, which in 1920 supplied 86·8 per cent. of the total number of cases notified. Large cities and industrial centres have been particularly affected; it is doubtful, however, if this is due to anything more than the greater opportunities for producing cases which is provided by comparatively large populations, though, possibly, the better facilities for diagnosing a comparatively new disease possessed by large towns may also contribute. It is, too, in the poorer and more congested parts of towns that the chief incidence of the disease occurs; but, here again, density of population rather than any inferior social conditions is probably the determining factor, and multiple cases of encephalitis lethargica in one household are as rarely found in slums as elsewhere.

Altogether, during the three years 1919, 1920 and 1921, cases have been notified from 520 (45·7 per cent.) of the 1,138 urban, and from 254 (39·0 per cent.) of the 650 rural districts in England and Wales; again, the total number of sanitary districts (774) in which cases have occurred up to the present is composed of 67·2 per cent. Urban Districts and 32·8 per cent. Rural Districts.

The distribution of cases in London is shown in the accompanying table, where it will be seen that the various metropolitan boroughs have been affected in different degrees. The total number of London cases is probably not large enough to furnish any reliable deductions, but on the whole it is the larger populations that have furnished the greatest number of cases; for 1920, the first three boroughs in the order of attack rate per 100,000 of population were Shoreditch, 6·8; Stepney, 6·4; and Wandsworth, 6·2.

TABLE 5.

Boroughs arranged in order of population figures.	Population middle of 1919.	Cases in 1919.	Cases in 1920.	Total
Wandsworth - - -	333,693	1	18	19
Islington - - -	323,034	1	7	8
Lambeth - - -	282,322	1	11	12
Camberwell - - -	273,802	0	3	3
Stepney - - -	232,506	4	16	20
St. Pancras - - -	219,434	2	5	7
Hackney - - -	216,736	4	8	12
Southwark - - -	179,971	1	6	7
Lewisham - - -	167,754	6	6	12
Battersea - - -	159,316	2	7	9
Kennington - - -	157,886	1	5	6
Poplar - - -	153,644	2	4	6
Fulham - - -	152,543	0	1	1
Paddington - - -	143,938	1	1	2
Hammersmith - - -	130,981	1	4	5
Woolwich - - -	136,237	2	3	5
Westminster - - -	127,533	0	4	4
Bermondsey - - -	124,239	1	4	5
Deptford - - -	111,205	0	2	2
Bethnal Green - - -	110,085	3	5	8
Greenwich - - -	98,484	0	3	3
Shoreditch - - -	98,134	0	7	7
St. Marylebone - - -	97,953	0	5	5
Hampstead - - -	88,012	2	4	6
Finsbury - - -	75,291	0	0	0
Chelsea - - -	60,573	0	3	3
Stoke Newington - - -	50,954	0	1	1
Holborn - - -	38,156	0	3	3
Totals - - -	4,344,317	35	146	181

General attack rate for 1920 = 0·03 per 1,000 of population.

Statistics regarding some of the *largest English towns*, other than London, are given in the following two tables. Table 6 shows the incidence in the 12 most populous towns, and Table 7 gives a list of the 12 towns which in 1919 and 1920 produced the greatest number of cases.

TABLE 6.

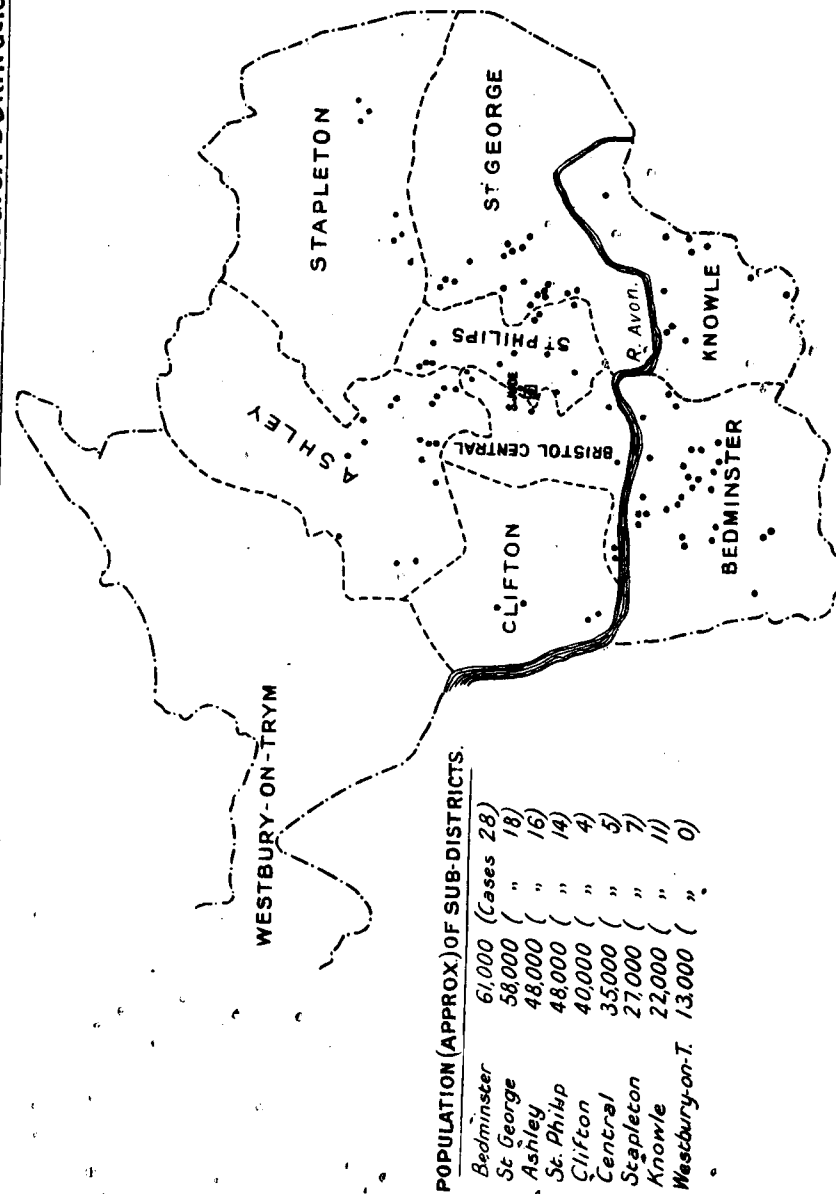
Town.	Population middle of 1919.	Cases notified in 1919.	Cases notified in 1920.	Cases notified in 1921 up to Sept. 30.	Total.	Rate per 100,000 for the three years.
Birmingham	861,585	11	20	24	55	6.3
Manchester -	741,068	11	19	27	57	7.6
Liverpool -	772,665	3	17	29	49	6.3
Sheffield -	473,695	6	16	6	28	5.9
Leeds -	430,834	5	9	13	27	6.2
Bristol -	361,247	18	34	56	108	29.8
West Ham -	287,966	9	13	4	26	9.3
Bradford -	282,714	2	7	12	21	7.4
Kingston-on- Hull -	279,664	2	11	4	17	6.0
Newcastle -	275,099	2	6	13	21	7.6
Nottingham -	257,573	0	6	1	7	2.7
Stoke-on- Trent -	239,316	3	2	6	11	4.5

TABLE 7.

Town.	Population middle of 1919.	Cases notified 1919.	Town.	Population middle of 1919.	Cases notified 1920.
Bristol -	361,247	18	Bristol -	361,247	34
Croydon -	184,239	12	Birmingham	861,585	20
Manchester -	741,068	11	Manchester -	741,068	19
Birmingham -	861,585	11	Liverpool -	772,665	17
Derby -	123,930	11	Sheffield -	473,695	16
Coventry -	132,048	10	Southampton	126,033	14
West Ham -	287,966	9	West Ham -	287,966	13
Plymouth -	181,967	9	Lincoln -	60,581	11
Sheffield -	473,695	6	Kingston-on- Hull -	279,664	11
South Shields	111,502	6	Leeds -	430,834	9
Leeds -	430,834	5	Leicester -	236,059	8
East Ham -	144,451	5	Coventry -	132,048	8

An examination of the two tables above shows that, despite the general rule in the matter as previously proposed, there is not always a strict correlation between population and case incidence. The most striking exception to the general rule is provided by *Bristol*, whose aggregate of 108 cases during the period from 1st January 1919 to 30th September 1921 is nearly double that of Birmingham, though its population is scarcely half that of the Midland town.

MAP OF BRISTOL CITY (AFTER DR. D. S. DAVIES M. O. H.)
SHEWING LOCAL INCIDENCE OF ENCEPHALITIS LETHARGICA DURING 1919, 1920 & 1921 (TO SEPT.)



The case of Bristol, indeed, is apparently unique, for during each of the last three years it has occupied the premier position among the large towns (other than London), both as regards incidence and attack rate. The first case to be reported occurred on 18th December 1918, and others occurred during the first five months of 1919; thereafter the seasonal incidence for Bristol has followed very closely that observed in the country as a whole. The majority of the cases have occurred in the more thickly populated parts of the city, and notably in the St. George, St. Philip and Bedminster sub-districts. During a local investigation made in the autumn of 1920, a curious grouping of cases in the Barton Hill locality was pointed out by Dr. D. S. Davies, the Medical Officer of Health for the City; a large number of the cases occurred among households, one or more of whose inmates were employed at various large factories in the district, but it was found that no particular significance could be attached to these circumstances. Since 1920 the Barton Hill district has remained practically free of cases, but in Bedminster the disease appears to be maintaining its hold; this sub-district, too, contains a curious little partly-hidden street in which three different houses were involved, and where it seemed probable that the disease had proved personally contagious.

The local distribution of cases in Bristol during 1920-1921 is shown on the accompanying map, and the following figures, kindly prepared by Dr. Davies, show the incidence in the various sub-districts of the city during 1921.

TABLE 8.

Sub-district.	Population (approximate).	Number of Cases, Jan. 1—Dec. 23 1921.
Bedminster - - - - -	61,000	15
St. George - - - - -	58,000	10
Ashley - - - - -	48,000	10
St. Philip - - - - -	48,000	5
Clifton - - - - -	40,000	2
Central - - - - -	35,000	3
Stapleton - - - - -	27,000	5
Knowle - - - - -	22,000	6
Westbury-on-Trym - - - - -	13,000	2
Outside city - - - - -	—	2

When forwarding these statistics, Dr. Davies adds: "An interesting point to note is that the St. Philip's Marsh District, which is a thickly-populated working-class district, where we constantly look for scarlet fever, measles, diphtheria or smallpox, has remained during these two years (1920 and 1921) absolutely free from encephalitis lethargica. This district is all on low-lying ground—a great deal of it made from ash-tips, &c.—along the Feeder Canal."

If, now, the epidemiological features displayed by encephalitis lethargica during the last three years in Bristol be compared with those exhibited by the disease in this country as a whole, it will be found that, far from being singular, Bristol is, in this matter, quite orthodox on most points, and its records of encephalitis lethargica reflect fairly accurately the behaviour of the disease generally throughout England and Wales.

Thus :—

TABLE 9.

	Bristol.	England and Wales as a whole.
Prevalence.	Attack rate per 1,000 = 0.0298.	Attack rate per 1,000 = 0.02.
Local distribution.	Chiefly in thickly populated areas.	
Seasonal incidence.	The incidence charts are very similar; the summer rise of 1920, as well as the marked outburst in the winter of 1920-21, are both reflected by the experiences of Bristol.	
Sex.	No difference; a total of 96 cases was exactly divided between males and females.	Both sexes equally affected.
Age.	Most susceptible age 10-30; 24 per cent. of these attacked were between ages of 10 and 20.	Most susceptible age 10-20; 25 per cent. of cases fall into this age period.
Occupation, social status and hygienic conditions.	Of no ætiological significance.	
Instances of infection following recent contact with an acute case.	1 in 100 cases (approx.)	1.2 in 100 cases (approx.).
Mortality	1919 & 1920 combined - 47.7 1921 as far as is known - 29.0	42.3 per cent. 38.0 per cent.

We may, perhaps, be allowed to suggest here that the well-sustained vigilance on the part of the City Health Authorities and the constant co-operation between them and many keen clinical observers in Bristol, have possibly accounted in part for the many cases of encephalitis lethargica notified from that city.

• *Wales.*—A case was reported from Bangor early in 1918, but it is in South Wales that the disease has chiefly declared itself, and particularly in the county of Glamorganshire; the county distribution is seen in the following table :—

TABLE 10.

Wales.

County.	Estimated population middle of 1919.	Notified cases, 1919.	Notified cases, 1920.
Anglesey - - -	47,592	—	—
Brecknockshire - - -	55,147	—	1
Cardiganshire - - -	55,897	—	1
Carmarthenshire - - -	169,746	—	3
Carnarvonshire - - -	111,830	1	1
Denbighshire - - -	141,756	4	—
Flintshire - - -	95,099	1	1
Glamorganshire - - -	795,924	12	26
Merionethshire - - -	39,781	—	1
Monmouthshire - - -	355,216	4	10
Montgomeryshire - - -	48,787	—	—
Pembrokeshire - - -	86,277	3	2
Radnorshire - - -	20,881	—	—
		25	46

It will be noticed that the incidence corresponds very closely with the density of population, and it may be added that a large proportion of the Glamorganshire cases occurred in the Rhymney and Rhondda Valley coal districts; the more mountainous regions of Wales have remained exempt.

• *Scotland.*—It is difficult to say how far encephalitis lethargica prevails in Scotland, since the disease is notifiable in Aberdeen Burgh only; moreover, from a short report kindly supplied by the Scottish Board of Health—in which is given a list of 40 “intimations of encephalitis lethargica” received by the Board between 1st January 1919 and 30th September 1921—it appears that only one case was notified from Aberdeen.

From published reports and such other information as is available, it seems as if the disease, as elsewhere in Britain, is chiefly reported from the larger centres of population, and more especially from *Edinburgh* and *Glasgow*; one authority thinks that, as far as the south of Scotland is concerned, the disease is

as prevalent as it is in England. Clinical reviews of the disease in Scotland have been published by Bramwell and Miller (126), and by Findlay and Shiskin (336), the two latter authors deal especially with encephalitis lethargica in children.

Ireland.—From notes kindly furnished by Sir E. Coey Bigger, of the Local Government Board, Dublin, it appears that records of encephalitis lethargica in Ireland are confined to *Dublin* and *Belfast*. In the former city, three cases occurred in January 1919, and form the subject of a report published by Drs. O'Carroll and Nesbitt (751); another case occurred in November 1919 and was reported by Sir John Lumsden (610), while in 1920 and 1921 only one case in each year apparently came to notice.

In Belfast a case occurred on 21st May 1919, and thereafter the incidence in Ulster is as follows:—

TABLE 11.
Incidence in North of Ireland.

District.	1918.	1919.	1920.	1921.
Belfast - - - - -	1	2	15	4
Newtownards Rural District -	—	—	5	—
Tanderages Urban District -	—	—	2	—
Bangor Urban District - - -	—	—	1	—
Larne Urban District - - -	—	—	1	—
Portrush Urban District - -	—	—	1	—
Newcastle - - - - -	—	—	1	—
Magherafelt Rural District -	—	—	1	—
Cookstown Rural District - -	—	—	1	2
	1	2	28	6

It is said that nearly all the patients from the districts outside Belfast were connected by business with the city or had visited it shortly before their illness. It is in, and around, the chief industrial centre of Ireland, then, that most of the reported cases of encephalitis have occurred.

Islands round Great Britain.—The following table shows the position as regards the disease in the various islands:—

TABLE 12.

Island.	Population.	Number of cases.	Source of information.
Isle of Wight -	79,836	9 (Jan. 1919–April 1921).	Notification returns.
Guernsey - - -	42,997	2	Dr. W. D. Bishop.
Isles of Scilly -	2,097	No cases.	Dr. W. B. Addison.
Isle of Man - -	52,034	No cases.	Dr. R. Marshall.
Anglesey* - - -	50,943	No cases.	Notification returns.

Anglesey and, to a less extent, the Isle of Wight, may be considered epidemiologically as part of the mainland, and the fact that some cases have occurred in the latter while Anglesey remains free is in keeping with the circumstances that their contiguous mainland districts also show, respectively, a comparatively high and low incidence. The non-appearance of the disease in the Isle of Man, which is so popular a resort with large numbers of people from the industrial centres of the North, seems to show that human travel does not help in the dissemination of the virus. This finds some corroboration, too, in the case of Guernsey, another favourite holiday resort both with this country and the Continent: one of the two patients treated in Guernsey was a young recruit, while the other man was sent from Dorset to recuperate from a nervous breakdown. The military case was notified in November 1918, and no other cases occurred until the second was imported in September 1921.

*The Disease in Country Districts.**—When circumstances favoured, intensive investigations of country cases were undertaken with a view to searching more easily for possible mild and abortive cases; such efforts so far, however, have not met with the same success that followed a similar procedure in the case of acute poliomyelitis.

It may be recorded, however, that four of the instances where more than one case occurred in a single household are referable to country districts. Not only so, but in three instances out of the four the dwelling places were isolated and off the beaten track: thus a young collier, occupying with his mother a lonely hillside cottage in South Wales, contracted the disease from his parent and, like her, died. Two well-marked, and two mild cases, occurred in an isolated farm situated high up among some foothills in North Wales, but again this incidence could not be linked up with any suspicious illness in the district; one of the patients in this instance, however, had recently stayed in London (which for him was a very unusual experience) and had come back indisposed. In a third instance, two cases occurred—at long interval—in an out-of-the-way homestead on Dartmoor, and though on this occasion there were two other cases in the neighbouring small town, no connection whatever could be traced between these and the farm cases or between each other.

At present, then, it cannot be said that the circumstances of country cases have helped very much to explain the curious way in which discrete cases of encephalitis lethargica crop up here, there and everywhere.

5. Seasonal incidence.—Our experience of encephalitis lethargica as an epidemic disease is limited at the most to four seasons and shows that hitherto the colder period of the year has formed the setting for the epidemic outbursts of the disease both at home and abroad. It is, perhaps, a little early to regard this

* See also footnote on page 47.

particular seasonal recurrence as a confirmed habit of the disease; there was, for example, in England and Wales a very distinct rise in the incidence during the summer of 1920, which found a parallel in Denmark during the same year, where the cases for May, June and July averaged 30 per month. This summer rise of 1920 will be better appreciated by reference to those charts in which the weekly incidence of cases is represented, not according to notifications, but according to the onset of symptoms; the number of cases began to increase at the beginning of April, and with two slight intermissions rose rapidly until the week ending 5th June, when a total of 33 cases (exceeding by four any previous weekly total for the year) was recorded. Then the incidence fell week by week until it reached a more usual figure by the middle of August. No such summer rise was observed in 1919, but there is a suspicion of the same thing in 1921 when, after an abrupt fall at the end of March, there was a slight recrudescence during April and May.

Generally speaking, however, the incidence begins to rise at the end of the year, reaches its maximum in January and February of the next year, and then declines somewhat sharply: it is during the first quarter of the year that the greatest number of cases are reported. The intervals between the epidemic outbreaks are bridged by a more or less unbroken chain of sporadic cases. The graphs which display the incidence (according to date of onset of symptoms) for the years 1919, 1920 and 1921, regarded separately, show certain differences: thus, that for 1919 is a symmetrical picture which is flanked by peaks of equal height representing the rise in incidence at the beginning and end of the year; in the 1920 graph the valley (seen so well in the previous year) is partially obliterated by the unusual summer rise while the peak on the right overtops by far that of the left.

The contiguous portions of the graphs for 1920 and 1921 illustrate very vividly, and in an unusual degree, the sharp rise in incidence which we have come to associate with the month of December and the large proportion of cases that occur during this and the succeeding two months. The number of cases occurring during the season 1920-21 is unprecedented and, as will be seen, the composite peak representing this marked incidence is precipitous on either side, though marked also with deep steps.* The manner in which the picture for 1921-22 will

* It should perhaps be mentioned that this sudden rise in the number of notifications anticipated the publication, though not the conception, of the Ministry's *Memorandum on Encephalitis Lethargica* (Memo. Med. 45); this pamphlet was distributed to Medical Officers of Health at the end of February 1921, whereas the number of notifications had already begun to fall off by the second week in February. That there was a real, and unusually vivid, "flare-up" of the disease during the early weeks of 1921 is also shown by the experiences abroad, and especially by the returns from Northern Europe. Thus, in Sweden, 978 cases were reported as having occurred during the period January 1st-March 31st, 1921.

Chart I. ENCEPHALITIS LETHARGICA.

SHOWING AN UNUSUAL MIDSUMMER RECRUDESCENCE IN 1920.

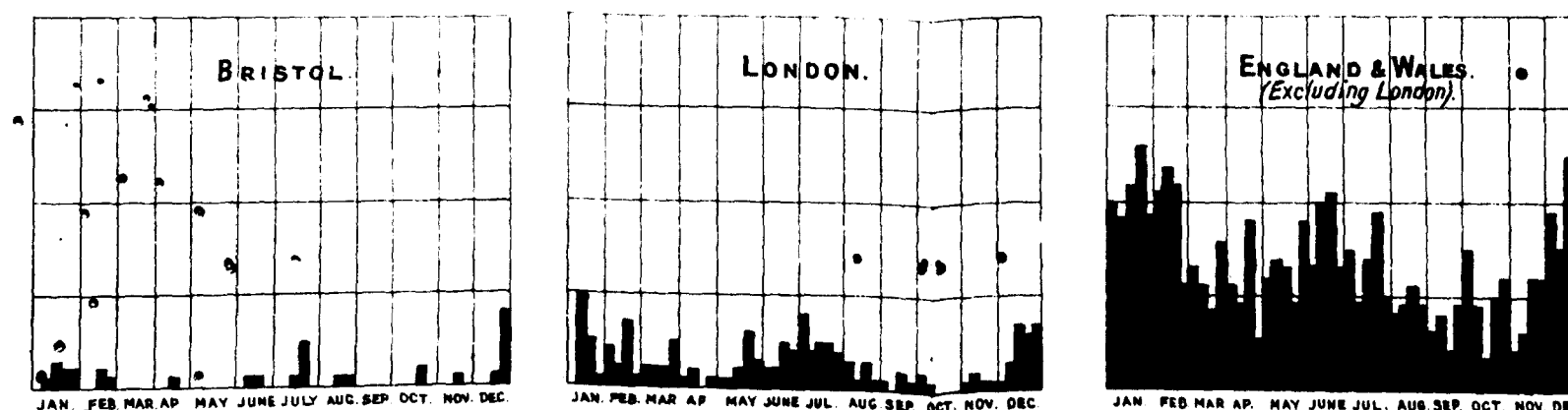
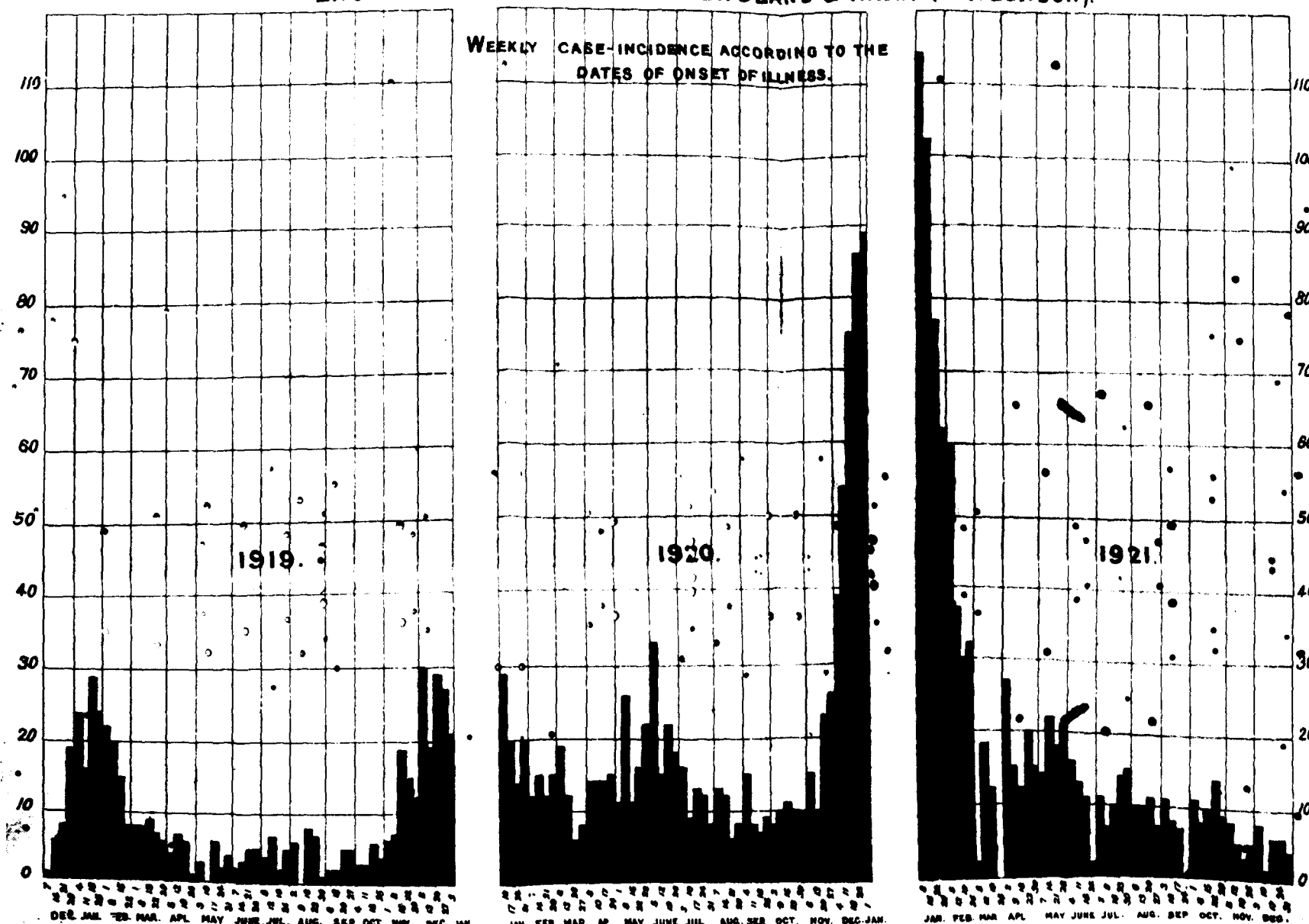


Chart II. ENCEPHALITIS LETHARGICA IN ENGLAND & WALES (INC. LONDON).



be completed is, at the time of writing, a matter for conjecture ; it appears, however, as if the disease had rather spent its epidemic force during the winter of 1920-21 and is now subsiding, for its seasonal recrudescence is overdue.

4. Association with other Human Epidemics : (a) *Influenza*.—

It is not proposed to discuss here the relationship which possibly existed between epidemics of influenza and certain outbreaks of various forms of 'sleeping sickness' which have occurred in the past : the literature on this subject has been well traversed by certain writers, of whom Crookshank (241), Netter (731), Durodie (306), Jorge (492), and others may be specially mentioned, and the first writer in particular has made several most interesting contributions to the historical study of influenza and associated epidemics.

In the consideration of recent experiences of encephalitis lethargica, it is the correlation in time upon which most stress is laid when the similarity, or even identity, of the two maladies is suggested ; but the exact nature of this correlation seems to vary widely under different circumstances. Thus it may be noted that the occurrence of the first cases of this disease in Vienna preceded the pandemic of influenza ; in Canada, on the other hand, the Winnipeg outbreak of encephalitis followed the influenza epidemic after a year's interval. In France, England, Wales and the United States, outbreaks of influenza and encephalitis lethargica during the last three or four years have been more or less coincident, while German observers have noted, in their country, that encephalitis in epidemic form has only occurred shortly before, with or soon after outbreaks of influenza. Switzerland affords a good instance of how the time association between the two diseases varies according to the particular season under review, and the figures below (from the Swiss *Official Health Bulletin*) show how close, in 1920, was the parallelism between the incidence of encephalitis and influenza.

TABLE 13.

Month.	Encephalitis lethargica.	Influenza.
1920.		
January	38 cases	13,023 cases
February	440 "	83,008 "
March	348 "	38,914 "
April	78 "	3,615 "
May	25 "	368 "
June	11 "	208 "
July	12 "	45 "
August	6 "	42 "
September	8 "	101 "
October	4 "	74 "
November	7 "	195 "
December	7 "	186 "

During that portion of the two preceding years, however, when Switzerland was involved in the pandemic of influenza, encephalitis lethargica was comparatively unknown or, as put by Dr. Carrière, "l'encéphalite est restée silencieuse pendant que déferlait la grande vague grippale de 1918-1919." In Spain encephalitis lethargica appeared at the beginning of the winter of 1919-1920, and so coincided with the end of the influenza epidemic. As regards England and Wales, it is not possible to compare accurately the relative incidence curves for encephalitis and influenza, because case notifications apply only to the former disease; but a comparison of death return figures for the 96 great towns, which represent, roughly at any rate, the seasonal incidence of influenza in the country as a whole, with the notification returns for encephalitis, suggest the following observations. The preliminary outbreak of encephalitis in England during the spring of 1918 preceded by some months the serious epidemic invasion of influenza which started in the late autumn of 1918; the greatest weekly number of deaths from influenza (7,557) occurred in the middle of November 1918, and from that date the mortality figures for this disease begin to decline just as the incidence figures for encephalitis begin to rise. The latter figures, in turn, proceed to come down at the time when there begins, early in February 1919, the second sharp rise in the mortality from influenza, which reached its acme about 8th March 1919; soon after this, influenza retires into the background until March and April of 1920. In the meantime, however, a very definite epidemic recrudescence of encephalitis marked the winter of 1919-1920, and this was soon followed by the reappearance of influenza as mentioned above, though the proportions of this outbreak were comparatively small. The next rise in the mortality figure for influenza began about 9th November 1920, and reached its maximum in March 1921; this influenza rise coincided with a marked recrudescence of encephalitis lethargica, but here the parallelism ends. In the first place, this coincidence of onset relates at once to the smallest outbreak of influenza and the greatest outbreak of encephalitis since 1918; and, secondly, the epidemic outburst of the latter disease had waxed and waned before the influenza wave had reached its crest in March. Sporadic cases of encephalitis continued to occur during 1921; but at the time of writing (1st January 1922) there is no indication that encephalitis lethargica is showing any epidemic accompaniment to the increased prevalence of influenza which is now occurring.

Examined in this piecemeal fashion, the events in England regarding the epidemic behaviour of influenza and encephalitis lethargica do not seem to represent a mutuality of any constant nature: at some periods influenza is first in the field and is then followed at varying intervals by encephalitis lethargica, while at other times the latter disease appears first and may even hold the field alone for a time; the fact that the primary outbreak

of encephalitis in the spring of 1918 preceded the big epidemic of influenza in the following autumn and winter should not, perhaps, be unduly emphasised, for in the dazzling glare of a "new disease," the co-existence of a considerable amount of influenza may have been overlooked. On one occasion only did the two diseases begin their epidemic waves coincidentally.

It is, of course, true that the coincident prevalence of two diseases by itself does not necessarily imply a common cause, but it is at least significant that the greatest social upheaval of our times should be marked by the closely-connected appearance of a "new disease" and a pandemic of influenza. In 49 out of 55 Departments of France which were circularised, the opinion was expressed that there was a possibility of a close relationship between encephalitis lethargica and influenza, and in some such guarded manner do many individual observers express their opinions on this subject. Others, however, think definitely that encephalitis lethargica represents a nervous "localisation" or "manifestation" of influenza. Our series of cases, it is true, provide instances in which patients exhibited prodromal symptoms of influenza, while in the acute stages of the disease they were afflicted with symptoms considered typical of encephalitis lethargica, but such cases only formed about 7 per cent. of the total series. Moreover, the stupor and the ophthalmoplegic signs, so characteristic of encephalitis lethargica, are only rarely seen in influenza. The supposition, too, that encephalitis lethargica is but a clinical syndrome which follows in the wake of a previous attack of influenza is not borne out by our investigations, for only in an insignificant proportion of the notified cases is there any history of previous influenza.

In the matter of complications there is a marked difference between the two diseases; for, while in recent influenza epidemics the majority of cases have shown a strong pulmonary bias, respiratory troubles in encephalitis are rare, though they are the most frequently observed of such complications as do occur.

There are differences, also, between the brain lesions that are found in influenzal encephalitis and those that characterise encephalitis lethargica; in the former condition the lesions are more hæmorrhagic and do not especially select the basal ganglia. In none of the post-mortem reports on encephalitis lethargica is the presence of Pfeiffer's bacillus reported, though this organism is often found in the brain tissue and in the meninges of patients dying from influenzal encephalitis.

Certainly, the case reports in our series confirm the opinion now generally held that encephalitis lethargica is a disease unto itself and cannot be considered either as a nervous form, or a complication, of influenza.

Nor have our investigations produced much, if any, evidence that influenza prepares the way or renders the soil suitable for the incidence of encephalitis lethargica. It is difficult, for instance, to reconcile the irregular way in which influenza and

encephalitis lethargica have been associated in this country with the idea that the latter disease is epidemically dependent upon the former; moreover, there is a great disparity between the number of individuals affected by the two diseases respectively and the comparatively small crop of encephalitis cases that followed such a massive and widespread "dose" of influenza as the population received in 1918-19 hardly justifies the assumption that encephalitis lethargica thrives best or exclusively upon ground where influenza has previously flourished. In the case of individuals, too, it has already been noted that in a comparatively few instances only does an attack of encephalitis lethargica follow a previous illness from influenza; with equal rarity, also, is it recorded in our series of reports that cases of the two diseases coincided in the same house.

Lastly, in their respective methods of propagation the two diseases stand widely apart: the infectiousness of influenza is proverbial, and the malady, in its periods of epidemic vigour, may quickly and fearfully encompass whole households, schools, districts, countries; the infectiousness of encephalitis lethargica is scarcely apparent, and we can bear in mind all the instances among our series where case-to-case infection probably took place. The occurrence of more than one case of encephalitis lethargica at a time in home, school or institution is very rarely recorded, and the seemingly capricious way in which the disease crops up in the most unexpected circumstances can be most baffling.

To sum up: such evidence as we have been able to use does not support the alternative propositions that (i) influenza and encephalitis are one and the same disease; (ii) that encephalitis lethargica is a nervous complication of influenza; (iii) that outbreaks of influenza must necessarily precede the epidemic appearance of encephalitis lethargica; (iv) that influenza is a common predisposing cause in cases of encephalitis lethargica.

(b) *Encephalitis Lethargica compared with acute Poliomyelitis and Cerebro-spinal Fever.*—There is some clinical communion in the case of these three diseases which are all concerned with the nervous system and, despite certain marked disqualifications, encephalitis lethargica seems also to have many epidemiological features in common with acute poliomyelitis and cerebro-spinal fever.

The association with acute poliomyelitis, indeed, was at one time considered by many to be so close that this subject formed one of the primary objects of the original inquiry by the Local Government Board and was fully dealt with in the report that followed. Here, before considering the three chief diseases together, we may first draw attention to a case quoted by Boyd (121 b) in which a patient, who suffered in childhood from typical poliomyelitis, subsequently contracted and died from encephalitis lethargica; in view of the theory that one attack

of poliomyelitis confers immunity, such an event as that described by Boyd is very significant. The various points in the epidemiology of encephalitis lethargica, poliomyelitis and cerebro-spinal fever may now be set out in tabular form as below:—

TABLE 14.

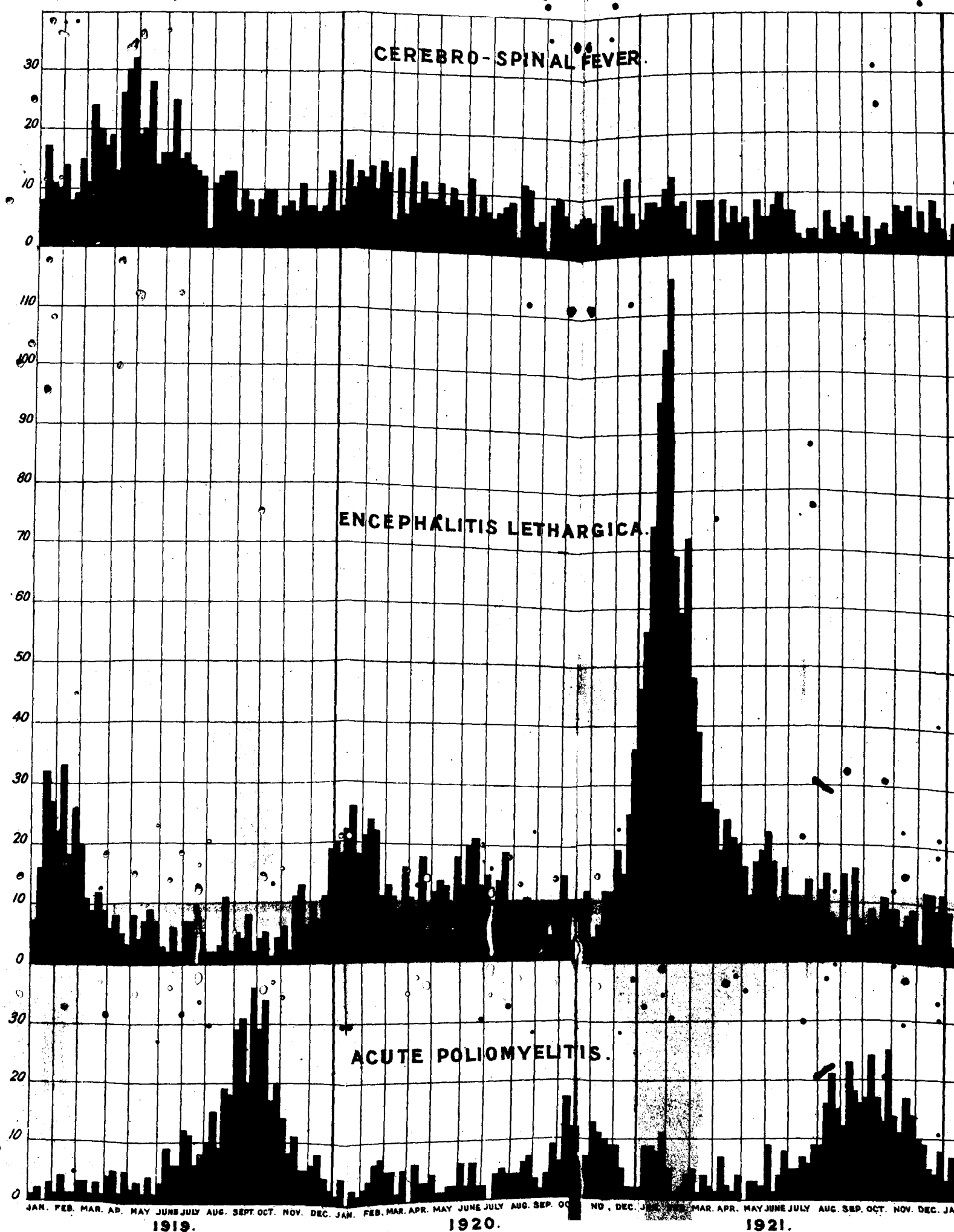
	Encephalitis lethargica.	Acute poliomyelitis.	Cerebro-spinal fever. (among civilians)	Remarks.
Prevalence.	1918 1919 1920 1921 — 0.01 per 1,000 0.02 " " 0.04 " "	0.01 per 1,000 0.02 " " 0.01 " " 0.01 " "	0.02 per 1,000 0.02 " " 0.02 " " 0.01 " "	Population figures according to the 1921 census.
Form	Epidemic and sporadic.	Epidemic and sporadic.	Epidemic and sporadic.	Both Pm. and C-s.f. dispose to localisation during epidemic times.
General distribution.	Widespread	Widespread	Widespread	But cases sparsely distributed.
Local incidence	Industrial centres and urban districts chiefly.	Not so generally urban in its incidence	Urban districts chiefly.	In all three cases sporadic cases occur in isolated country districts not infrequently.
Seasonal incidence. See Chart III, opposite.	December, January and February. January especially.	Late summer and autumn.	Seasonal preference not marked. In 1919, however, the chief incidence was in spring and early summer.	There was a summer rise of E.L. in 1920 and a winter rise of Pm. January 1921.
Sex	Both sexes equally.	Both sexes equally.	Slightly higher incidence in males.	—
Chief age incidence.	(1919 and 1920) 10-20, 35 per cent.	(1919). Under 10, 78.0 per cent.	5-10	E.L. may occur at almost any age; Pm. rarely seen in patients over 10; C-s.f. under certain conditions (e.g., war) becomes prevalent among young adults.
Occupation	No particular occupation figures prominently, but those pursued indoors or in school occur most often in the records.	School	School	During the war C-s.f. was especially prevalent among recently mobilised troops.

Chapt. III. A
ENCEPHALITIS LETHARGICA compared with CEREBRO-SPINAL FEVER and ACUTE POLIOMYELITIS.
SEASONAL INCIDENCE, ACCORDING TO NOTIFICATIONS, DURING 1919, 1920 & 1921.

	Encephalitis lethargica.	Acute poliomyelitis.	Cerebro-spinal fever (among civilians).	Remarks
Social status	All classes equally liable in proportion to their relative numbers.	All classes	Poorer classes most affected.	Adverse material conditions probably have some influence in the case of P.m. and C-s-f., but none as regards E.L.
Hygienic conditions.	Immaterial	Immaterial	Overcrowding is a predisposing factor.	—
Infectivity	Apparently very low.	Slight	Not highly contagious.	—
Method of spread.	?By undetected carriers and through medium of mild undetected cases; rarely case-to-case.	Transmission from person to person including transmission by mild cases and healthy carrier.	Chiefly by carriers and under certain conditions by contact with patients.	No evidence of natural transmission by insect agencies or through fomites. This applies to all three diseases.
Mortality (notified cases).	1919, 48.7 per cent. 1920, 48.0 per cent.	1919, 14.7 per cent. 1920, 10.0 per cent.	1919, 67.0 per cent. 1920, 70.0 per cent.	—

It will be seen that there are many characteristics shared in common by the three diseases; among the features in which they strongly differ should be noted the seasonal incidence, age susceptibility and mortality. Two of the diseases, within the limits of their prevalence, are very fatal, and it may be said that while cerebro-spinal fever is a killing disease and poliomyelitis a crippling one, encephalitis lethargica is both highly mortal and disabling.

(c) *Encephalitis Lethargica and Epidemic Hiccough.*—The occurrence of hiccough as a troublesome symptom of influenza was constantly noted in the recent pandemic, both at home and abroad, and Logre and Henyer (596), among others, suggest that the recrudescence of this symptom in epidemic form during 1920, especially in France, is a further manifestation of "grippe à forme phrénique." In November 1919, however, epidemic hiccough appeared in Winnipeg and neighbouring towns, and its advent coincided more or less with the occurrence of the first case of encephalitis lethargica in that district. The epidemic, which is referred to by Boyd (121 b)—whose paper seems to have escaped general notice in this connection—included a very high number of cases, and doctors were frequently attacked. The condition was, also, observed as a symptom of encephalitis lethargica in a number of cases, "but was never so prominent as to constitute a marked feature of the clinical picture."



Boyd draws attention, also, to some cases in Italy which were reported by Gotti (387), who considered that an outbreak of seven cases near Ravenna was definitely associated with an outbreak of encephalitis lethargica which was taking place in this district at the same time.

In the beginning of 1920, Sicard and Kudelsky (963 a) drew attention to the clonic spasms of the diaphragm which they had noticed in some of their cases of encephalitis lethargica and which, in some instances, were accompanied by hiccough. At the same time, Dufour (293) recorded 11 cases of encephalitis in which hiccough was a prominent symptom. Towards the end of 1920 and at the beginning of 1921, there was in France and in some other parts of the Continent a distinct wave of epidemic hiccough which was closely associated with the greatly increased prevalence of encephalitis lethargica that occurred at that time. The first cases in France were said to have occurred at the beginning of November and the epidemic reached its height at the beginning of December when it became a much discussed subject in the daily press.

Cases of hiccough were now appearing sporadically in England; with certain exceptions to be mentioned later, the affection was not associated with encephalitis lethargica; nor was there any such grouping of cases as was reported, for instance, at Lille, where 40 cases occurred in eight days among 2,000 factory hands.

Many French authorities now considered that there was sufficient evidence to regard epidemic hiccough as one of the myoclonic manifestations of encephalitis lethargica, while Von Economo recorded that a recent epidemic of hiccough in Austria was followed in a few weeks' time by the appearance, in the same district, of myoclonic encephalitis. Finally, Clerc, Foix and Mercier des Rochettes (210) reported a most interesting case of hiccough in a woman of 68, in whom the symptom lasted for six days; on the seventh day the temperature rose, the patient became somnolent and died on the following day. At the post-mortem, no gross changes were found, but histological examination showed the characteristic changes of encephalitis lethargica—not in the brain, but in the cervical region of the cord.

On the other hand, to Sicard and Paraf, writing in December 1920 (965a), the affection appeared as a slight affair, having no connection with encephalitis lethargica; Pontano and Trenti (819) too, as the results of some inoculation experiments on human subjects, and after reviewing the literature, came to the conclusion (in August 1921) that epidemic hiccough is not a form of either epidemic encephalitis or influenza.

As regards epidemic hiccough in England, the most interesting series of cases were investigated by our colleague, the late Dr. Farrar, early in 1921. A clerk in the accounts department of a large factory at Dudley near Birmingham, suffered from severe

hiccupping in December 1920; on 20th December he consulted his medical man (who the next day himself developed an ordinary cold), and on the 22nd he started to hiccup and continued doing so for two days and nights even when asleep. Subsequently two other patients of Dr. L. suffered from the same symptom.

In the meantime a Mr. G. P. of Moseley had dined with his son in Birmingham on 16th December, and from 20th–24th December Mr. G. P. senior suffered from hiccup; while recovering from this affection at Cambridge he was visited by his son on 26th December. On 27th December the son, while playing golf, found that he could not see the ball, and this disability proved to be the first symptom of a definite attack of encephalitis lethargica. While ill his house was constantly visited by a brother, Captain P., who, however, did not see the patient except on 6th January, when he entered the sick room to assist in filling a water mattress. On the same afternoon Captain P. began to hiccup and suffered severely in this way for 36 hours; in other ways he was quite well.

Now Captain P. was employed at the Dudley factory, and a friend of his who was in the habit of seeing him daily at the works had suffered from hiccup from 18th to 21st December. This little local series then involved eight people, seven of whom within a period of two or three weeks suffered from hiccup, while the other developed encephalitis lethargica. All the eight cases appear to have been linked up in an interesting and intimate manner; the infectiveness of this form of hiccupping seems to be illustrated by this series of cases, and that some kind of association existed between the illness of Mr. C. P., junior, and the hiccup from which his father and brother suffered also seems probable.

In another instance, also investigated by Dr. Farrar, a girl of 16, living with her father and mother at Warwick, was overcome by a typical attack of encephalitis lethargica at the time that her father was recovering from 12 days of incessant hiccupping.

A series of 12 cases of hiccup was also investigated at the out-patient department of Charing Cross Hospital by Dr. MacNalty in December 1920, and it is noteworthy that the Casualty medical officer who attended these patients himself became afflicted with the same condition.

Hiccup, as a symptom occurring in cases of encephalitis lethargica, was noted in two cases of MacNalty's, and in five of the present series; it was also mentioned from time to time in the reports received during the earlier months of 1921. These cases will be discussed from the clinical standpoint in Section III., but the main points about the recent occurrence of epidemic hiccup may now be recapitulated here.

(a) A remarkable epidemic of cases of hiccup occurred in Winnipeg during November 1919, in close association with the advent of encephalitis lethargica in this part of Canada;

probably the hiccup outbreak ante-dated the first appearance of encephalitis.

(b) During 1920 it was observed on the continent that hiccup was by no means an uncommon feature in the syndrome of encephalitis lethargica, and more especially in the myoclonic cases.

(c) During the winter of 1920–21, and at a time when there was a marked recrudescence of encephalitis, cases of hiccup became very prevalent for a period both on the continent and in England, and formed, indeed, a concurrent epidemic. Only in a comparatively few instances, as far as English cases are concerned, could any clinical connection between encephalitis lethargica and epidemic hiccup be traced, and the brief-lived though distressing malady in its epidemic behaviour differed in many ways from epidemic encephalitis. As far as can be gathered from the literature, epidemic hiccup, unlike encephalitis lethargica, cropped up first in the West; the infectiveness of individual cases was an obvious characteristic and exemplified by the occurrence of multiple cases in factories, &c., and by the number of medical men affected; the condition was curiously restricted almost entirely to males, and those at a higher age level than is the case in encephalitis.

The striking coincidence of the two epidemics in point of time, however—together with the fact that in certain instances both the encephalitis and the hiccup syndromes have been seen together in limited communities such as factories and families—certainly suggest that the two conditions have something in common; and this inference is strengthened by those instances in which a similar form of hiccup has occurred in the case of a patient actually suffering from encephalitis lethargica. Finally, the circumstances of the patient, who died after an illness characterised by little else than hiccup and fever, and in whose cord were found lesions typical of encephalitis lethargica, would seem to indicate that on occasion encephalitis lethargica can be represented practically by a single symptom due to a lesion in the cord which involves the cervical centre of the phrenic nerve. A less serious involvement than that in this particular instance might account for the more trivial cases of hiccup, and make plausible the suggestion that such cases represent mild forms of encephalitis lethargica. This, however, is to credit the virus of encephalitis lethargica with an extraordinary eclectic capacity in the first instance—if the implication of the phrenic nerve is always a nuclear one—and with an unusually simple and limited set of clinical consequences upon the patient.

5. Encephalitis Lethargica and certain Epidemics among Animals.—During the last two years it has been suggested, both by professional observers and in the lay press, that the usual clinical picture shown by human patients suffering from encephalitis lethargica has, on occasion, been more or less reflected by animals, chiefly dogs and cats, and that the malady among these animals has sometimes assumed epidemic proportions. Thus,

it was casually reported in January 1921 that, at *Alfriston* in *Sussex*, there had been an epidemic of illness among cats which was characterised by great sleepiness and a high mortality; further inquiries showed that this illness had been in existence from August–November of the preceding year, and that young cats were chiefly affected. It was also established that sickness and diarrhoea were prominent symptoms, and that the epidemic stopped with the advent of cold weather. There had been no cases of encephalitis lethargica, however, in the near neighbourhood, and the medical officer of health for the district considered that the cats had been the victims of an intensive anti-rat campaign.

Then, a somewhat alarming report appeared in the papers on 5th February to the effect that "cats are dying wholesale" in north Essex from a mysterious infectious disease. Hundreds "have died in *Saffron Walden* during the last fortnight, and there "are now very few left." It was further stated that the symptoms displayed resembled those of human encephalitis lethargica, and that dogs were not affected. The matter was taken up with the Board of Agriculture and Fisheries, who received from the local veterinary surgeon at *Saffron Walden* a report on the outbreak, in which the malady was described as "some infectious and highly contagious disease of bacterial origin." "Sleepy typhoid" was the name given by the veterinary practitioner in question, but the authorities at the Board of Agriculture and Fisheries were inclined to regard the epidemic as due to a gastric form of distemper. It subsequently transpired that the reports had been considerably exaggerated, and further investigation showed that there were no cases of human encephalitis lethargica that could be in any way associated with this feline outbreak. The cord from one of the fatal cases was examined by Dr. Perdrau, but as five days had elapsed since the cats died, and no other animals were provided, the bacteriological examination was unsatisfactory; in such sections as could be fixed at all, however, Dr. Perdrau found nothing to suggest either encephalitis or poliomyelitis, nor indeed was there evidence of any other lesion.

Meanwhile, at *Hayes* in *Kent*, another outbreak—this time among dogs, cats and ferrets—had attracted the attention of the Press, and this occurrence also was described as "a mysterious outbreak resembling sleeping sickness," in which the affected animals became drowsy, refused food and then crawled into dark corners to sleep and die. These cases were also investigated by the Board of Agriculture and Fisheries, and were considered to be further instances of gastro-distemper, while no sort of correlation between them and cases of encephalitis lethargica could be discovered.

Then, in March, a report was received from *Tacolnstone* in *Norfolk* stating that in one household alone 19 cats had died, or had been destroyed, on account of some strange malady

said to be suggestive of encephalitis lethargica. Here again, however, there was no local incidence of the latter disease.

In *Moreton Hampstead*, *Devon*, where towards the end of February 1921, the circumstances of some familial cases of encephalitis lethargica were being investigated, other instances of epidemic disease among dogs and cats came to notice, and on this occasion there was a coincident aggregation of human encephalitis cases in the neighbourhood.

According to the local veterinary surgeon, Mr. W. F. Sanders, there had been a very bad form of distemper among dogs during the winter of 1920–21 in *Moreton* and the surrounding neighbourhood. The symptoms included paralysis, epileptic fits, deficient vision—sometimes involving blindness, and drowsiness towards the end. Retention of urine was also a common symptom, and there were discharges from the throat and nose and offensive stools.

Previous to this canine complaint there had been an epidemic among cats characterised by groups of cases in which paralysis, vomiting and discharge from the nose and throat were the prominent symptoms. There was no evidence that these cases of animal disease were grouped round the cases of encephalitis lethargica, though it may be mentioned that a dog died of distemper in 1920 at the farm where two cases of encephalitis had occurred. In another part of *Devonshire*, near *Chagford*, the Medical Officer of Health had his attention drawn by an old lady to the case of a cat and her two kittens; all three animals suffered from lethargy and transient squint, and later it was found that the gardener's cat was similarly affected. Knowledge of the previous occurrence of three cases of encephalitis lethargica at *Chagford* had, in this instance, aroused interest in the behaviour of the cats.

Lastly, in a lonely farmhouse at *Abinger*, *Surrey*, a child was attacked in December 1919 with encephalitis lethargica. While a week or so previously three of the horses went down with symptoms of "influenza" consisting of acute catarrh, sore throat, nasal discharge, congestion of the lungs, fever, anorexia, depression and great weakness.

In all the different circumstances mentioned above there is no bacteriological proof that the maladies from which the various animals suffered and died were in any case at all analogous with encephalitis lethargica; nor, with the possible exception of the *Devonshire* cases, can it be said that the clinical signs were at all suggestive of human encephalitis lethargica. Still less reason is there for thinking that the various outbreaks of epidemic diseases among animals had any general ætiological relationship whatever with cases of human encephalitis lethargica. The only suggestive feature in this connection is the fact that these animal epidemics coincided in point of time with a very high general incidence of encephalitis lethargica throughout the country. It is certainly interesting, too, that in the case of both *Saffron*

Walden and Moreton Hampstead veterinary surgeons of long experience stated that in each instance the sickness among dogs and cats was something quite different from what they had been in the habit of treating. Possibly animals, too, have been affected by a new "constitution" peculiar to these times!

As regards the experimental infection of animals MacIntosh and Turnbull (621 and 622) in this country, and Levaditi and his co-workers (570, 571 and 572) in France have found that the rabbit, the guinea-pig, the mouse and, to a less extent, certain species of monkeys, are all susceptible to the virus of encephalitis lethargica.

(B) ÆTIOLOGY (GENERAL).

1. **Race.**—No information was solicited under this heading, for, in the case of such a slightly prevalent disease as encephalitis lethargica, the British Isles do not offer sufficient scope for any profitable inquiry. Moreover, no safe conclusions as to racial susceptibility, or the contrary, can be drawn from the information set out under "Local incidence" (see Sec. II., A. 2), because no comparisons are reliable when the sources of such information, and the methods of its collection, vary so much. In the world at large the disease has been chiefly reported among the peoples of Europe and North America, and from the literature it would appear that the disease is especially prevalent among the Latin races both in Europe and in South America. Little has been heard of the disease in the Far East, though cases have certainly occurred in India, China and Japan. In Africa, encephalitis lethargica has appeared along the northern and western coasts, while in the Transvaal, judging from a small outburst reported by Butt (155), the disease is particularly fatal to natives. In Australia, New Zealand and Tasmania the disease is well recognised, but cases apparently are sparingly distributed and confined to the non-native communities.

2. **Sex.**—There is no discrimination between the sexes considered at all ages, the figures for our series being females 639, males 634. The sex incidence at various ages is shown below:—

TABLE 15.

Age group.	0-1	1-5	5-10	10-15	15-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90
Sex	M. F.	M. F.	M. F.	M. F.	M. F.	M. F.	M. F.	M. F.	M. F.	M. F.	M. F.	M. F.
Number	6 6	39 44	48 47	22 69	89 87	83 108	91 102	103 84	65 56	33 24	4 2	0 1

It will be seen that the scales are held fairly even all through the age periods. During the child-bearing years the female cases slightly predominate, but for the critical age period from 40-50 the figures show no excess of cases among women, but an actual minority.

There is considerable variation in the results of other analyses at home and abroad, and as a rule the preponderance is on the side of the males; in view of the larger number of cases examined, however, an equality of distribution among the sexes, as shown in our figures, is probably the rule.

3. **Age Incidence.**—No age is entirely exempt, but the statistics under review show that the disease, in its usual form, at any rate, is found chiefly among those approaching adult life, and 25 per cent. of the total series of cases occurred among young people between the ages of 10 and 20; the actual figures are set out in the tables below:—

TABLE 16.

AGE INCIDENCE ACCORDING TO DECADES.

Age group.	0-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-9
Number of cases	181	327	191	193	187	121	57	15	1

TABLE 17.

AGE INCIDENCE IN QUINQUENNIAL PERIODS UP TO AGE OF TWENTY.

Age group.	0-1	1-5	5-10	10-15	15-20
Number of cases	12	74	95	151	176

It will be seen from the first table that, after adult life is reached, there is a diminishing liability to attack; a sharp drop occurs in the numbers at the end of the second decade, but the incidence seems fairly evenly distributed over the years 20-40; thereafter the chances of infection rapidly decline as old age approaches.

The second table, which deals more closely with the incidence in the earlier years of life, shows that the liability increases at each quinquennial period until 20 is reached, and the 176 cases occurring in individuals between the ages of 15 and 20 form 18.8 per cent. of the whole series. Twelve infants under 1 year of age were attacked, and the youngest patient in all probability contracted the disease from its mother when but two weeks old. Happ and Mason (427) record an instance of the disease in an infant five weeks old, and Findlay and Shiskin (336) include among their analysis of cases in children in Glasgow the case of a baby aged four weeks.

It should be mentioned that the age period of maximum incidence as shown in our series is distinctly lower than that arrived at in some other analyses, as appears from the tables below; at the same time it will be noticed that in Neale's series exactly half the cases occurred in children of 15 and under.

TABLE 18.

British.		American.		American.		Swiss.		From the literature.	
1,273 cases.		Josephine Neale, 274 cases.		H. F. Smith, 181 cases.		Carrière, 528.		Strauss and Weschler, 864 cases.	
Age group.	Per-centage of cases.	Age group.	Per-centage of cases.	Age group.	Per-centage of cases.	Age group.	Per-centage of cases.	Age group.	Per-centage of cases.
		Mths.							
		3	1.4						
		3-6	1.4	under 1	3.32				
		6-12	2.9						
		Yrs.							
		1-2	7.2						
1-10	14.2	2-5	10.5	1-4	11.6	1-4	2.27	under 5	1.5
		5-10	17.1	5-9	12.5			6-10	4.0
10-20	25.6	10-15	9.1	10-14	8.3	5-14	15.3		
		15-20	5.4	15-19	6.63	15-19	15.1	11-20	15.7
20-30	15.0			20-24	8.3				
		20-30	20.4	25-29	7.18	20-29	19.6	21-30	25.7
30-40	15.1	30-40	12.0	30-34	7.73				
				35-39	6.63	30-39	17.6	31-40	25.0
40-50	14.6	40-50	10.5	40-44	11.05				
				45-49	4.42	40-49	11.3	41-50	16.2
50-60	9.5	over 50	3.2	50-54	3.87				
				55-59	3.31	50-59	7.3	51-60	8.3
60-70	4.4			60-64	2.75				
				65-69	2.21	60-69	5.6	61-70	2.8
70-80	1.1			70-74	—				
				75 & over.	0.55	70 & over.	1.7	over 70	0.7

4. **Occupation.**—Information on this head was available in the case of 1,070 patients, and though the various methods of employment noted are exceedingly numerous, an attempt has been made to classify the many occupations in three groups, as follows:—

TABLE 19.

Occupations involving a considerable time being spent indoors.	Occupations involving a considerable time being spent out-of-doors.	Indeterminate group.
I.	II.	III.
Household duties - 217	Labourers - 54	Artisans and mechanics - 96
School children - 199	Farmers, gardeners, &c. - 31	Professional - 62
Factory hands - 83	Travellers, postmen, messengers and others - 24	No occupation - 23
Shopkeepers - 60	Seamen - 11	Ex-service men - 16
Clerks - 60	Police constables - 5	Various - 6
Domestic servants - 37	Medical men - 4	
Tailoring - 23	Vagabonds - 3	
Miners - 22	Dairymen - 1	
School teachers - 21		
Nurses - 9		
Publicans - 3		
Totals - 734	133	203

The main feature which appears from this point of view is that the disease chiefly attacks those individuals, adults and children, who spend the greater part of the day indoors in pursuit of their duties. It is possible, too, that the figures in column I. are under-estimated, and should be reinforced by some transfers from the "indeterminate" column. It will be noticed that the occupations generally in column I., as it stands, have a distinctly feminine character, in marked contrast with those in columns II. and III.; as stated elsewhere, however, the general incidence of the disease is about equal in the two sexes.

The occupation most commonly recorded is "housekeeping," and this is closely followed by "school," so that housewives and school children between them contribute 38.8 of the cases in which the occupation is recorded. In one respect, the almost identical position at the top of the list of these particular women and children seems significant; for while increased risks of contagion might be held to account for the comparatively large number of school children affected, it cannot apply in the case of the housekeeper left to herself during most of the day. It is not at all unlikely, however, that housekeeping, which during the last few years has become a difficult and disquieting occupation, is in itself a predisposing factor in the case of a disease such as encephalitis lethargica.

Among other reflections suggested by the tables above is the small number of medical men and nurses who have suffered from the disease, which seems to show that contagion must play a very small ætiological part. The fact, too, that only one case was reported among dairy workers is not without significance when considering the question of possible avenues of infection.

5. **Social Conditions.**—Opportunities for obtaining direct information about the social standing and home conditions of the patients have been necessarily restricted; the knowledge concerning the nature of the occupation, however, has given a sufficient clue in many cases, and shows that the disease is no respecter of persons, but attacks the professional workers as well as the manual labourers, those with comfortable homes no less than those who are not so blessed. Moreover, the schedule answers received concerning the hygienic conditions of the patients' homes show that, with a negligible number of exceptions, these conditions are quite satisfactory. Comparatively high though the incidence is among those who work in assemblies or in a restricted space (school children, factory hands, clerks, tailors and others) it does not appear that the disease selects its victim from those living in homes that show poverty and insanitary conditions. If the disease has any predilection at all it is, judging from the homes noticed on our visits, for the thrifty households of the artisan and professional classes.

6. **Other Possible Predisposing Conditions.**—The answers to question 12 on the inquiry schedules as to "any recent illness.

or condition which may have lowered the patient's resisting power, or otherwise predisposed him to the present illness," include conditions both numerous and varied.

A predisposing cause was adduced in the case of 559 or 43.9 per cent. of the accepted cases; but, since in some instances the conditions, chiefly those relating to influenza, were obviously not "recent," these cases are not included. Allowing for this, there remain 488 cases in which the alleged predisposing conditions may be grouped as follows:—

I. Conditions leading to, or symptomatic of, psychic disturbance.

	Cases.
Overwork - - - - -	34
Worry - - - - -	24
Grief - - - - -	11
Neurasthenia - - - - -	11
Military service - - - - -	9
Shock - - - - -	7
"Shell shock" - - - - -	6
"Nervous breakdown" - - - - -	4
Mental strain - - - - -	3
Poverty - - - - -	3
Captivity - - - - -	3
Hysteria - - - - -	2
	— 117

II. Specific infectious diseases.

Influenza - - - - -	114
Measles - - - - -	7
Pneumonia - - - - -	6
Scarlet fever - - - - -	5
Whooping cough - - - - -	5
Diphtheria - - - - -	5
Mumps - - - - -	4
German measles - - - - -	1
Chicken pox - - - - -	1
Acute poliomyelitis - - - - -	1
	— 149

III. General diseases.

Tuberculosis - - - - -	13
"Rheumatism" - - - - -	5
Malaria - - - - -	3
Syphilis - - - - -	1
	— 22

IV. Mental and nervous disorders.

	Cases
Neuritis - - - - -	5
Chorea - - - - -	3
Epilepsy - - - - -	2
Dementia - - - - -	2
Meningitis - - - - -	1
	— 13

V. Circulatory system.

Anaemia - - - - -	10
Cardiac disease - - - - -	4
Cerebral thrombosis - - - - -	1
	— 15

VI. Respiratory system.

Naso-pharyngeal catarrh - - - - -	12
Sore throat - - - - -	7
Pleurisy - - - - -	5
Broncho-pneumonia - - - - -	4
Bronchitis - - - - -	1
Rhinitis - - - - -	1
	— 30

VII. Digestive system.

Gastric catarrh - - - - -	5
Dysentery - - - - -	3
Jaundice - - - - -	2
"Liver trouble" - - - - -	1
	— 11

VIII. Conditions due to physical causes.

Injury to head - - - - -	19
Injury, unclassified - - - - -	12
Heatstroke - - - - -	1
Exposure - - - - -	1
Lightning - - - - -	1
	— 34

IX. Poisons.

Alcohol - - - - -	4
Poison gas - - - - -	4
Septicæmia - - - - -	1
	— 9

X. Conditions peculiar to women.

Pregnancy - - - - -	25
Labour - - - - -	3
Menopause - - - - -	3
Menstrual disorders - - - - -	2
	— 38

XI. Various.					Cases.
General debility	-	-	-	-	16
Headache	-	-	-	-	11
Enlarged tonsils	-	-	-	-	6
Dental troubles	-	-	-	-	9
Otorrhœa	-	-	-	-	4
Life in the tropics	-	-	-	-	3
Epistaxis	-	-	-	-	2
Enlarged thyroid	-	-	-	-	1
Exophthalmic goitre	-	-	-	-	1
Cancer	-	-	-	-	1
Furunculosis	-	-	-	-	1
					55
					488

The conditions enumerated above form a "mixed bag" indeed, and it is safe to assume that, while most or all of these various disorders and diseases may possibly have lowered the patients' resisting powers, only a small minority have any closer connection with encephalitis lethargica than mere coincidence or antecedence in individual cases.

Some consideration, however, may be given to the antecedent conditions mentioned in groups I. and II. References to *overwork*, more especially among professional and business men, and to various circumstances which produced a general nervous instability, are comparatively frequent in the reports, and it is perhaps natural that such circumstances should have been considered as arising out of the war, and its aftermath. Encephalitis lethargica, however, has so far only visited, say, some 2,000 households in England and Wales, while there is hardly a home in the land that has not been affected in greater or lesser degree by the war and its after effects; and, when it is considered that not more than 9 per cent. of the patients were previously affected by disorders which may have been engendered by the war, it will be seen that this great event has had little to do with the appearance of individual cases of encephalitis lethargica. The most that can be said therefore as regards the conditions in group I. is that they figure more often as alleged predisposing causes than any other set of circumstances.

The *specific infectious* diseases as a class evidently do not dispose to encephalitis lethargica; figures of seven and five for measles and scarlet fever respectively acquit these two zymotic diseases of any causal responsibility in this instance, despite the fact that they were unusually rife during at least part of the period under review.

Influenza, on the other hand, cannot be dismissed quite so summarily, and the evidence concerning the possible rôle played by this disease as a clinical antecedent in cases of encephalitis

lethargica may be examined more closely. As regards the patient himself influenza is mentioned as a possible predisposing factor in the following manner:—

TABLE 20.

Time incidence of influenza.	Reported number of cases in which influenza in the patient is mentioned as a possible predisposing cause.	
	1919.	1920.
Just previous to, or as the preliminary symptoms of, the attack of encephalitis.	41	56
3-6 months previously	14	3
6-9 " "	8	5
9-12 " "	5	1
1-2 years	5	20
2-3 " "	1	16
Time not stated	8	0

In the case of those sharing the patient's home the figures are as follows:—

TABLE 21.

Time.	1919.	1920.
At the time of, or just previous to, the patient's illness	51	26
3-6 months previously	6	2
6-9 " "	3	1
9-12 " "	—	—
1-2 years	5	5
2-3 " "	1	6

In view of the prevalence of influenza at various times during the period which embraces the several references to this disease, and as far as any attempts to establish a relevant antecedence to encephalitis are concerned, the figures above seem insignificant and far from convincing; it is further suggested that in a large majority of the cases the influenzal symptoms were in reality the early signs of encephalitis lethargica. It is quite possible, too, that some of the members reported as suffering from "influenza in the house at the time" may have been abortive cases of encephalitis.

No other morbid condition occurs sufficiently often in the reports to call for any remarks, and it may be repeated that in

more than half the number of cases there was a negative history of any kind of predisposing condition; in a few instances stress is laid upon the point that the patient was particularly healthy prior to the attack of encephalitis, as in the case of a young child who had recently obtained two "firsts" at a baby show!

7. Pregnancy and Encephalitis Lethargica.—The remarks concerning this subject are founded upon the examination of 50 case reports in which the patient concerned was either pregnant at the time of her illness or was attacked by encephalitis after the confinement and within the period of normal nursing; a few cases are also included in which the medical attendant considered that a former pregnancy was partially responsible for the attack of encephalitis. It should be mentioned, too, that a wider field than usual has been covered in the collection of these particular case reports; for, in addition to the notification and schedules for 1919 and 1920, those of 1921 up to 30th September have also been surveyed. During the first two years, however, notifications were received concerning 37 cases of encephalitis lethargica associated with pregnancy. The nominal details given in the schedules are not explicit enough to supply information regarding the number of married women of child-bearing age who were attacked by encephalitis; but of 1,273 cases reported during 1919 and 1920, there were 212 women, both married and single, whose ages ranged between 20 and 40.

Dealing with the complete series of 50 cases as set out in Table A (Appendix II.), the average age of the patient when attacked by encephalitis was 29; the youngest woman was aged 17 and the eldest 46. The incidence of the encephalitis is shown in the accompanying table where it will be seen that the majority of cases occurred among women in whom labour was either imminent, actually in progress, or but recently experienced.

TABLE 22.

Near full term.	Month of pregnancy.					During or soon after labour.	After confinement (from 2-13 weeks).	After miscarriage (2 days—14 mths.).	While nursing.	After former pregnancies.	Total.	
	8	7	6	5	4							3
12	6	3	5	2	1	1	4	8	4	2	2	50

The onset of encephalitis was more or less gradual in the majority of cases, but characterised as a rule by signs and symptoms of a definitely nervous character. In three cases the beginnings of the illness were considered to be influenzal and in eight cases neuralgia and localised pain, often severe, were the first symptoms to excite comment. As the disease progressed the common triad of symptoms, viz., fever, lethargy and various

ocular palsies were exhibited in most instances, and were associated not uncommonly with profound psychical disturbances. In over 50 per cent. of the cases, too, the encephalitis was represented by signs of irritation, chiefly localising in character, and consisting of muscular twitchings, tremors of limbs, and coarser movements of a choreic character; in two instances epileptiform seizures occurred. Six of the patients suffered from vomiting which was so intractable in one case that premature induction of labour was necessary, but in the remaining five patients the sickness does not seem to have been of serious import, and in three instances it occurred some time after the confinement.

In 10 per cent. of the cases, a comparatively high proportion, skin eruptions were evident, and these were of an erythematous character in three cases.

In one instance (No. 45) a scarlatiniform eruption was a marked feature in the case of the mother and her child, and both ultimately died.

The mortality rate generally was close on 50 per cent. (24 out of 50) and, of those patients who survived, seven suffered from after-effects, such as lack of control, depression, insomnia, tremors, defective vision, &c.; one patient suffered from a relapse. Of the other 18 cases, six were slowly improving when last seen, while the remaining 12 patients have recovered completely. The highest proportion of deaths occurred among those who were in an advanced state of pregnancy, or had recently been confined, but among the fatal cases was a young girl, overtaxed with the care of her two babies, and also a mother much debilitated from nursing a child for 14 months.

The course of the pregnancy, and the ultimate fate of the mother, are correlated in the table below, which refers to those cases in which the incidence of encephalitis occurred while the patient was pregnant, or during the puerperium.

TABLE 23

Fate of the child or fetus.	Living and healthy child.	Child died shortly after birth.	Still-born.	Mis-carriage or abortion.	Unborn.	Developed encephalitis lethargica.	Signs of congenital syphilis.	Unknown.
	15	4	4	5	8	4	1	6*
Fate of the mother }	R = 10	R = 3	R = 3	R = 2	—	R = 3	—	R = 5
	D = 5	D = 1	D = 1	D = 3	D = 8	D = 1	D = 1	D = 1

It will be seen that, where the pregnancy came to full fruition, the child, in the majority of cases, was healthy when born, and remained so as far as can be discovered; in one case delivery

was effected by Cæsarean section, and the mother also made a good recovery. A comparatively small number (4) of babies died in early infancy, and two out of this number died as the result of encephalitis to which fuller reference has been made elsewhere.

Of the other two fatal cases, one child died on the 10th day and the second, an eight-months child, succumbed two days after birth. Labour was artificially induced in two of the cases where the child was still-born; in one instance the mother's life was saved (intractable vomiting had occasioned intervention), and in the other the woman died. In another case the uterus was emptied with extraneous help at the sixth month, and the patient recovered.

A miscarriage occurred at the seventh month in a patient who suffered severely from involuntary movements during her acute illness, and also during her convalescence from encephalitis. In the other two cases, which both proved fatal, the accident occurred before the onset of acute symptoms of encephalitis, but probably within the prodromal period of the disease.

An instance of early miscarriage or abortion was provided by a woman of 24 whose illness was apyrexial throughout, and from whom a three-months foetus was expelled an hour before the patient's death from coma. In all of the eight cases in which the patients died *enceinte* the pregnancy was well advanced, and in one instance it is recorded that the foetus (six months) was living up to the time of the mother's death.

It might be expected that the supervention of an acute toxæmic disease, in which fever is usually a prominent symptom, and in which the mental and nervous organisation of the patient is profoundly affected, would increase the normal demands of pregnancy upon the mother, with consequent prejudice to the child. This expectation, however, is not altogether borne out by the statistics of our series of cases. The mortality rate in respect of all the cases of encephalitis lethargica which were associated with a pregnancy past or present, is as was stated, approximately 50 per cent.; but, by excluding seven instances in which the pregnancy was a more or less remote antecedent, the figure becomes reduced to 44 per cent., which also represents the general case mortality among all women between the ages of 20 and 40 who contracted encephalitis lethargica during 1919 and 1920. The mortal effect of encephalitis upon a woman then would seem from our figures to be uninfluenced either way by the condition of pregnancy. In an analysis of 864 cases in the United States of America, 24 women were pregnant, and of these only four died; Jorge (492), on the other hand, in reviewing a series of 18 cases, mentioned in the literature of various countries, finds that the mortality rate was 72 per cent.

In two out of our four cases the uterus was prematurely emptied by artificial means with successful results to the mother,

but Jorge again found that in a series of 27 cases this procedure was not beneficial.

As regards premature birth, the reports show no indication that encephalitis lethargica disposes to such an event. Galabin states that the usual proportion of miscarriages to full term deliveries is as 1 to 5, and in the series of cases under review there were five miscarriages as compared with 24 fully completed pregnancies.

8. Infection and Contagion.—There is no evidence from the histories which have been studied or from the general literature on the subject that the virus of encephalitis lethargica is conveyed through the medium of either water, soil or food and the circumstances under which cases of this disease seem to occur also support this conclusion. As regards infection through articles of clothing, &c., there is also no evidence, with the exception of a doubtful case in Warwickshire, where a patient before her attack had been wearing a blouse made for her by a dressmaker who had died of encephalitis lethargica. As a parallel to this case may be quoted an instance reported by Guillain and Lechelle (405) in which the father of a doctor living in Paris was taken ill with encephalitis of a myoclonic type at the end of January 1920, and died nine days later. This patient lived in a town in the centre of France and his son did not leave Paris for his father's late home until the 5th of August following. He remained there 15 days settling affairs and sorting his father's clothes, wearing some of them himself. On 5th September, 16 days after his return to Paris, he himself was also attacked with encephalitis, the acute symptoms of which persisted until 27th November, after which his condition began to ameliorate.

The possibility of infection by inoculation and other ways through the medium of biting insects and house-flies respectively was considered in the ætiological enquiries, but not a scrap of evidence could be found to warrant any further research in this direction; as regards house-flies, too, it may be noted that encephalitis lethargica is most prevalent in winter when these insects have mysteriously disappeared.

Excluding, then, these various agencies that lend themselves to the dissemination of certain other diseases it would seem that the virus of encephalitis lethargica must in some manner be conveyed through the medium of the air and directly from case to case. It must be said at once, however, that there is as yet no solid mass of evidence weighing down the scale in favour of personal contagion as the determining factor in the spread of encephalitis lethargica.

In the first place, the disease, though it has attracted a great deal of attention, is still a comparatively rare one in terms of population, the rate per 1,000 of population in England and Wales during 1920 being 0.2 as compared for instance with figures of 3.19, 1.86, and 1.04 for scarlet fever, diphtheria and pneumonia respectively. Such comparative rareness, together

with a widespread distribution hardly seem compatible with a very high degree of infectiousness. From our statistics, too, it would appear that between 4,000 and 5,000 individuals were in direct contact with cases of encephalitis lethargica during the years 1919 and 1920, but the proportion of these "contacts" who subsequently contracted the disease is almost negligible; there were treated in General Hospitals 206 patients, but only in one recorded instance is it supposed that the disease was passed from one patient to another; cases have appeared in schools, homes, institutions, regiments and ships, but with one or two exceptions no outbreak has occurred. Lastly, reference to the remarks under occupation will show that those in professional attendance upon patients suffering from encephalitis lethargica have very rarely been attacked.

If in amount, however, the evidence concerning the contagiousness of encephalitis lethargica is small, it may be said that some of it, at least, is good. The Derby outbreak of August 1919 which was investigated by MacNalty (629e) stands out pre-eminently as an undoubted example of multiple cases occurring in an institution. The main facts of this outbreak as extracted by MacNalty (629g) from his original report are as follows:—

Date of Onset.

Two patients were attacked on August 14th, 1919.				
Two	"	"	"	15th, "
Four	"	"	"	16th, "
Two	"	"	"	17th, "
One patient was	"	"	"	19th, "
One	"	"	"	27th, "

"No further cases of illness occurred after August 27th. The range of age-incidence was from 15–40 years; the stress of the disease fell on the lower ages (15–18 years). These cases, with one exception, a mild case of the disease, all displayed marked lethargy and double ophthalmoplegia together with other general and nervous manifestations. Of the higher ages, one proved to be a mild case, that is a case in which the general and nervous phenomena were not so pronounced, and three cases, involving members of the staff, showing transient symptoms (so-called abortive cases); five cases including the initial case, proved fatal. The shortest duration of a fatal case was two days in a female child aged 13/4, the longest 49 days in a male, aged 30. . . . The source of the outbreak could not be ascertained. The dates of onset appeared to indicate that the first two cases derived their infection from a common source, as did also the two cases attacked on August 15th, although the hypothesis may be entertained of an unrecognised "abortive" case or carrier among the inmates which introduced the focus of infection into the Home, other cases being subsequently infected by personal contact. This would imply that the

incubation period was of short duration. The last two cases to be attacked were milder in type than the others; this, perhaps, indicated that the virulence of the causal organism had by that diminished or had exhausted itself."

But the most intimate degree of contagion is seen in those cases recorded, both at home and from abroad, where a newly-born infant has contracted the disease from its affected parent and, presumably in some of the cases, by way of the placenta. Four cases of "encephalitis neonatorum" have been reported in this country and both these and the foreign examples may be briefly described here. *Case I.*, which came within the scope of the previous enquiry and was reported by Dr. Wilfred Harris (429a) concerned a mother of 28 in whom labour was induced on 7.4.18 after a sudden onset of encephalitis lethargica on 29.3.18. The baby subsequently exhibited mild signs of encephalitis but eventually recovered, as did the mother.

In *Case II.*, (Dr. Pirie's case) the mother, who lived in a village near Leamington, was confined on 28.10.20, three days after the onset of encephalitis lethargica. The baby weighed 7½ lbs. at birth but an intelligent and experienced nurse who was in attendance did not consider it a normal infant and it was "never strong." The child was fed partly on cow's milk and partly with milk drawn off from the mother. After a week to 10 days it was noticed that the right upper eye-lid drooped and that there was right-sided paralysis of the face. There followed cyanosis of finger tips and lips, sweating, loss of weight, general twitchings, unconsciousness, and death in the fifth week of life. There was no history of tuberculosis, no meningeal cry, no rigidity and no fever was noticed at any time.

In *Case III.*, reported by Dr. R. Montgomery, the mother passed through a normal labour on 12.4.21, and during her confinement was attacked with encephalitis lethargica which manifested itself by fever, diplopia and lethargy with delirium and sphincter paralysis at the end; a scarlatiniform rash was present. An eruption, similar in all respects, appeared on the baby during the 14th day of its existence and was accompanied by fever and feebleness; it remained ill for a week and then died on 4.5.21, sixteen days after the mother.

Case IV. (Dr. D. J. M. Legge's case) concerns a lady who was attacked with encephalitis lethargica in the eighth month of her fourth pregnancy. Conjunctivitis and neuritis were early symptoms and later the patient suffered from lethargy, alternating with delirium; there were present, also, double ptosis, left facial paralysis, tremors, and twitching of left leg and arm. The confinement took place on 6.2.21, when a well-developed male child was born who cried lustily at birth; almost at once, however, the baby became abnormally quiet and lapsed into a condition of lethargy. For two weeks the eyes were constantly shut and ocular examination was therefore difficult; no facial paralysis was

noticed. Soon after the lethargy set in, however, the child became subject to twitchings of the left leg and arm and presented a very similar picture in this respect to that shown by the mother. The child, for the most part, was nursed normally, but at times spoon feeding (mother's milk) was necessary because the small patient could not be aroused to take the breast, or it would relapse into sleep after commencing to suck. There were no other symptoms reported and the child eventually made a good recovery after three months of illness. This case affords another example of the peculiar way in which the disease—with comparative frequency—reproduces in such newly-born infants as are attacked under like circumstances, one or more of the prominent symptoms shown by the mother; in this instance, too, the medical man in attendance considered that the child's illness was congenital.

In the foreign literature notice may be drawn to a Portuguese case described by Novaes and Sousa and referred to by Jorge (492). Here the mother's illness began with myoclonus of the abdominal muscles when she was at full term and at first there were no other symptoms. The woman was admitted to hospital on 18.4.1920 and the contractions were then violent, rapid and accompanied by lancinating pains; there were also slight convulsive movements of the face and of the right leg. On the 19th there was double ptosis, unequal pupils, and increased myoclonus. She was confined quickly and without pain on the same day, but her condition became worse with fever, somnolence and delirium. Coma was complete on the 23rd and death followed next day. The career of the child may be more graphically described in the words of Prof. Jorge: "Le nouveau-né, fortement cyanose, comme celui de Harris, sort du ventre maternel avec des myoclonies pareilles à celles de la mère et aussi de siège abdominal; des papules hémorragiques sont clairsemées sur la face, le cou et la partie supérieure du tronc; l'état général est bon et l'enfant se laisse allaiter très bien. La cyanose se dissipe au bout de 24 heures et aussi l'éruption papuleuse le 3^e jour; les myoclonies s'atténuent et finissent par disparaître cinq jours après la naissance. Santé parfaite et bonne croissance." And he adds: "voilà un cas congénital qui, selon nous, fournit la preuve de la transmission de l'encéphalite épidémique de la mère au fœtus."

Mercier (672) tells a somewhat similar story that concerned a woman of 28 who developed encephalitis lethargica with myoclonic onset and was delivered of a full term living child on the 22nd day of her illness; the mother died in coma on the 40th day of her illness after a notable remission on the 30th day. The child at birth was separated from its mother and fed, first on cow's and then on goat's milk. In the second week it showed myoclonic spasms of the head and upper limbs and reflected generally, but to a less degree, the clinical picture shown by the mother; the child recovered, however, without any treatment.

Santi (432), on the other hand, records an instance in which the disease was comparatively mild in the mother but proved fatal in the case of the child whose brain is reported to have shown typical lesions of encephalitis lethargica.

Reverting to the question of less direct contagion, attention may be drawn to Table B, Appendix II., which tabulates all the cases that have been brought to our notice during the first and the present enquiry in which it seems possible or probable that the patient was infected through contact with another case. Here it will suffice to classify roughly the main conditions surrounding these instances.

TABLE 24.

Conditions under which instances of multiple infection occurred.	Number of instances.
Familial cases*	16
While nursing cases of encephalitis lethargica	3
Institutional	2
Residential proximity	3
Occupational association	2
Social intercourse	2
Child birth	4
Total	32

On the Continent, Netter (731n), in particular, has shown enthusiasm on the question of *contagiosité* but most of the

* The most notable instances of familial infection are reported from Lapland where Kling and Liljenquist (513) of Stockholm, found, in February, 1921, that encephalitis lethargica was very prevalent in certain villages, the morbidity rate varying from 7.1 to 45.0 per cent. Rolleston (1,215) in referring to the report quoted above states: "In some families several members contracted the disease simultaneously, and in two houses almost all the inhabitants were affected. Side by side with severe cases presenting a typical clinical picture were others in whom the symptoms were less pronounced and consisted of catarrh of the upper respiratory tract, insomnia, fever, headache, dilated and sluggish pupils, dissociation of the ocular movements and occasionally facial paresis. Some patients were subject to obstinate hiccup. There were also milder cases still, with the same catarrhal symptoms, but without any signs of cerebral involvement."

This concentration of cases in such a wild and sparsely inhabited district as Lapland is in marked contrast with the usual localisation of the disease in the crowded urban districts of more civilised countries. During the rigours of a Scandinavian winter, however, domiciliary overcrowding and the restricted ventilation in the Laplander's primitive home, are probably concerned with the occurrence of these multiple cases and possibly, as with cerebro-spinal fever, the "carrier rate" rises according to the dangerous degree of overcrowding in any particular compartment. Kling and his co-worker mention that they found no evidence to suggest that either water, milk, bugs, fleas, lice, cats or dogs were concerned in the aetiology of these cases in Lapland; reindeer and mosquitoes are not apparently mentioned in this connection.

American writers in their reports appear to have little evidence in support of contagion.

Carriers.—Striking as is the nature of some of the instances of case-to-case infection, it is evident that the sum total of such instances in our series is hopelessly inadequate to explain the general method by which encephalitis lethargica is contracted and disseminated.

In these days, however, and in situations like the present, suspicion fastens upon the "carrier" and in the present circumstances his postulated existence certainly does appear to explain best how the chain of contact is maintained. The carrier hypothesis becomes strengthened, for example, by comparing the behaviour of encephalitis lethargica with that of two other epidemic maladies which in some ways it closely resembles, viz., acute poliomyelitis and cerebro-spinal fever. The same difficulty in explaining the apparent sporadicity of encephalitis lethargica formerly existed also in the case of these two diseases, and was only removed by the ultimate discovery of the carrier rôle in each instance.

Circumstantial evidence gathered in clinical and epidemiological investigation and crowned by positive bacteriological research showed that in the case of cerebro-spinal fever and poliomyelitis the virus was more commonly present in human individuals than clinical experience alone had suggested. It is extremely probable that the same holds true for encephalitis lethargica though one cannot as yet carry out any routine bacteriological test for carriers as in the case of cerebro-spinal fever; events in this direction are indicative, however, and Levaditi and Harvier (571) have shown that the virus of encephalitis lethargica may exist in the saliva of healthy contacts and by artificial transmission give the disease to rabbits. There are, moreover, here and there among the clinical and field records under review, a few indicative straws, which seem to suggest directions for the quest of carriers, using the word now in its widest sense. To begin with there are circumstances such as those attending the Derby outbreak where "the hypothesis may be entertained of an unrecognised *abortive* case or *carrier*." Then there have been several curious little groupings of cases as those in Bristol, South Wales, Walsall, St. Asaph, Moreton Hampstead and other places where no history of direct contact with previous cases could be established and where a common origin did not seem possible. In the very interesting series of cases at Moreton Hampstead a special problem arose in an isolated upland farm where in April 1919 the eldest son, who very rarely left the homestead, contracted encephalitis lethargica and his was the first case reported from the neighbourhood. The youngest sister in this family lived and worked away from home in at least two large towns, but she had been home for short visits at the end of 1918 and before her brother's illness. Eventually she found employment near London as a nurse, but

her health proving unsatisfactory she returned in January 1921 to Moreton Hampstead to recuperate. She had only been back a fortnight, however, when she became acutely ill and died on February 5th from encephalitis lethargica. It is difficult to avoid the conclusion that there was some correlation between these two cases; but, if the brother infected his sister then he had remained a carrier for 14 months, while if we assume that the more travelled sister brought home the infection from outside during one of her visits at the end of 1918, it must be also assumed that she acted as a carrier who was ultimately prostrated by a clinical manifestation of the disease from which she died.

Then there is the possible "carrier" who is himself definitely ill, but is masquerading under another guise and it seems quite possible that some of the inmates of infected houses who were recorded as suffering from influenza may be included in this category. Probably, however, the cloak beneath which the "carrier" is usually concealed is the "*mild*" or "*abortive*" case which either escapes attention altogether as a subject for any concern and treatment, or, while coming under medical supervision, is not considered in connection with encephalitis by reason of the trivial and, it may be, commonplace group of symptoms. The result in both alternatives is that such cases do not get reported very often, so that any opinion on this matter must be largely conjectural though the hypothesis that mild cases do act as carriers is again supported by analogy with acute poliomyelitis. It must be confessed, too, that the success which followed the plan of making an intensive investigation of poliomyelitis "outbreaks" in rural districts has not so far been repeated as regards encephalitis lethargica.

However, the records of general field work and the valuable contributions made by keen and interested observers have not been altogether barren of result.

A study of the report on the Derby outbreak (629 e), for example, will show that had some of the less striking cases occurred independently and not as component factors of this little epidemic, they would probably not have been reported at all as cases of encephalitis. (This is no new experience, of course, that mild and otherwise atypical cases of infectious maladies are more likely to be recognised during an epidemic phase of the disease than during the quiescent period when sporadic cases are the rule).

The Bristol series of cases, too, which was studied so carefully by the Medical Officer of Health and many practitioners of that city, contains examples of mild and doubtful cases that under less vigilant interest might have passed unnoticed.

Again, the late Dr. W. H. Bowen, while Assistant Medical Officer at Lambeth Infirmary, noticed early in 1921 that there were a number of cases in his P.L. districts (I.-VI.) which shared a peculiar syndrome of symptoms that could not be explained otherwise than by a diagnosis of mild encephalitis lethargica.

The patients were invariably children who nearly all suffered from mild lethargy, fever, throat symptoms and some suggestion of localising nervous lesions; the illness ran a short, and mild course, and ended satisfactorily in each instance. Such cases were prevalent during the early part of February when the general incidence for the country was beginning to decline from its highest point. With regard to other manifestations of encephalitis in the district it may be noted that these mild cases succeeded in point of time the incidence of cases notified from Lambeth Borough and no very striking local correlation could be established between the two sets of cases.

In those circumstances where more than one case has occurred in a family it was frequently found that one of the patients was only slightly affected and, but for the illness of the other inmate, would probably not have been considered a case at all. A good instance of this was investigated at a lonely farmhouse in North Wales where the eldest son was taken ill on January 8th; the case as it progressed was considered to be typically one of encephalitis and was duly notified as such. At the end of March the maidservant was found to be suffering in the same way and her case was also notified: further investigations then showed that not only this maidservant but a small daughter of the house was also overcome with lethargy and other symptoms at the time the son was attacked. The child's attack quickly passed off after 24 hours' continuous sleep, but the maidservant, who on the same occasion slept for two days and who subsequently resumed work, does not seem to have been at all normal in health during the period preceding her definite illness in March.

Convalescents.—Lastly, it is quite possible that the convalescent patient needs more supervision from the point of view of infection than has hitherto been considered necessary, and Netter thinks there are serious reasons for assuming that encephalitis may be disseminated by convalescents. Among such reasons might well be considered the fact that the virus of encephalitis lethargica has been obtained from the brain of a patient who died 15 months after recovering from his attack. (Netter, Cesari and Durand, 734). It may be mentioned, too, somewhat in this connection, that some of the clinical sequelæ are not only associated with severe or well-marked cases, but may supervene upon slight cases and even provide the right diagnosis in cases that at first were wrongly labelled or missed altogether.

The more immediate ways in which the virus of encephalitis lethargica probably reaches the individual and is spread among the community are discussed in the section on Pathology.

From his experience of consulting work in Shropshire, Dr. Willoughby Gardner considers that "a far milder form of encephalitis lethargica is very common indeed," but he goes so far as to think that the higher nervous system is only occasionally

attacked. He lays great stress on a peculiar condition of the throat which he considers is a characteristic symptom in many of the cases seen by him. He notes that this throat condition is generally not a matter for complaint on the part of the patient, so that if not specially looked for it is most likely to be missed.

Epidemiology and Animal Experiments.—This section may be ended with a brief consideration of how far the epidemiological conclusions find support from the aetiological researches which have been carried out by laboratory workers.

Levaditi and Harvier (571) are chiefly connected with the hypothesis that the virus is excreted by the nasal mucous membrane and they have found that while a healthy mucosa will withstand invasion by the virus an injured and inflamed membrane offers no resistance to its passage; in animals whose mucosa has been so prepared infection of the brain readily occurs if the injured parts are anointed with an emulsion of the virus. With regard to this hypothesis it cannot be said that a local reaction in the nasopharynx is by any means a constant clinical feature in patients suffering from encephalitis lethargica, nor is there any significant amount of evidence that patients who have contracted the disease had previously been in contact with a person suffering from nasopharyngeal catarrh. The comparatively small number of instances, too, in which more than one member of a household have been attacked with encephalitis lethargica seems to show that such an easy method of dissemination cannot often be at work. A notable exception was the Derby outbreak in which a majority of the patients in the Home suffered from "sore throat." Nor can it be said that encephalitis lethargica is, in its occurrence, particularly associated with any condition such as measles in which nasal catarrh is a prominent symptom, though it is certainly the case that the disease is chiefly prevalent at that period of the year when "colds" are common. But, again, the statistics do not show that encephalitis lethargica selects its victims from those communities where neglected colds are common and in which every opportunity for personal contagion would seem to exist.

Another assumption first put forward by Netter (734) is that the virus resides in, and is excreted by, the salivary glands, and it is claimed that the disease has been experimentally produced by injections of saliva. Here it may be said that only very rarely in our series of case-reports is there any mention of salivation or inflammation of the salivary glands. The enormous preponderance of single over multiple cases may be again suggested as a reason for thinking that natural infection by means of the saliva is rare; on a few occasions, too, when small groupings of cases did occur there is no record to suggest that the common use of food vessels and other objects was at all responsible for the spread of infection.

Among their more recent researches Levaditi, Harvier and Nicolau (572) have succeeded in recovering the virus of encephalitis lethargica from the hairs of patients who have died from the disease, but again our epidemiological experiences do not lend support to any suggestion that in natural circumstances the virus is spread through the medium of hair.

Lastly, the proposition that in certain cases newly-born infants have been infected by way of the placenta conflicts, it must be admitted, with the experience of those pathologists who have hitherto failed to isolate the virus from the blood; it is interesting to learn, however, that Levaditi, Harvier and Nicolau (572L) have recently succeeded with rabbits in transmitting the virus of the disease from the mother to the foetus.

Those cases of encephalitis neonatorum in which the transmission of the disease from mother to child probably occurred after birth may, perhaps, afford a few isolated instances of direct contagion referable possibly to a salivary source; it is significant, however, that in a majority of these cases the child was nurtured by its mother's milk, but we can find no reference to any research on this particular subject.

SECTION III.—CLINICAL.

A.—TYPES OF THE DISEASE.

"This, if I may say so, is a kind of sleeping sickness, generally prolonged, of which the first symptoms are usually acute headache and nausea; then follows somnolence, often associated with violent delirium, from which the patient is easily awakened and gives a rational account of himself, he can obey orders, stand or walk, but then relapses into somnolence. This delirious sleepiness can run its course rapidly or lead to death in the course of a few weeks; it can last with hourly, daily or even longer remissions for weeks or months unchanged, either as a slight sleepiness or in the form of a deep coma; or an improvement takes place, leaving the patient considerably weakened. In the simplest cases, in which there is sleepiness only, this symptom may quickly pass away so that eventually signs of paralysis may be the main feature of the case. In the first days of the illness certain isolated meningeal symptoms of irritation (rigidity of neck, tenderness on pressing the head and sometimes Kernig's sign) may appear though they are never very pronounced. The illness may proceed with fever of no known type, or it may be afebrile; the intensity of the fever appears to be unaffected by the course and symptoms of the illness

and the depth of the somnolence is quite independent of fever; we have seen a feverish patient who in the moment that she became free of fever was more delirious than before. In addition to the general manifestations there are, as a rule, signs of paralysis in the province of the cranial nerves as in the extremities; the eye muscles especially are frequently attacked. The slight ptosis which can be explained as the physiological eyelid heaviness of sleep, and which by a great effort of the patient can be overcome, passes gradually into a paralytic ptosis, often associated with a partial or total paralysis of the remaining branches of the oculomotor nerve. Paresis of the oculomotor nerves and other cranial nerves may appear and there may be paralysis of the extremities with reflex disturbances. The trouble with the eye muscles can also present itself as a first symptom of the illness, in which case the somnolence appears later in more or less severe form, or it may be completely absent."

In such terms did von Economo, in the early part of the year 1917 and with the experience of eleven cases, give the first description of a "new disease" as observed by him in Vienna and to which he gave the name of Encephalitis Lethargica.

This original conception of the clinical syndrome now known as encephalitis lethargica has, naturally, been broadened with increased experience and the original criteria while still holding good for the classical form of the disease, have had to be modified in many directions; evidence of this need can be seen in the various classifications of syndromes that have been attempted and of the making of "types"—more especially on the part of foreign observers—there seemed at one time to be no end.

As a matter of practice, however, it would probably be hard to find a disease whose symptoms and signs present more difficulties in the way of classification than the one under discussion; our knowledge concerning it is still far from complete and the beacons of bacteriology do not as yet extend all along the road of discovery; moreover, a toxic infection whose special goal appears to be one of the most highly specialised and all-pervading systems in the human body is likely to give rise to an extraordinarily varied symptomatology, which is, indeed, the case. Clinical classifications, as pointed out by several English writers, are difficult to frame and when made are seldom satisfactory; they have, for example, the fundamental drawback that at various stages in the course of an individual case it may be necessary to affix a new type label, witness such a title as *"Un cas d'encéphalite successivement névralgique, psychique choreiforme, myoclonique, léthargique avec séquelles hemimyoclonique après la guérison"* (498). Any attempt to fit into such clinical compartments as certain French authors have devised

the cases reported in our series would be very unprofitable since in most instances the reports do not cover the whole history of the case. The drawbacks to anatomical classifications are not so marked; and, when such are attempted, the close clinical observation which is necessary in order to correlate the outer and visible signs with the site and extent of the lesions producing them helps considerably, not only in a better understanding of each case, but also in the diagnosis of difficult cases.

Material and Classification employed in the present Report.—The total number of cases originally notified as encephalitis lethargica during 1919 and 1920 amounted to 1,549, and this total, owing to the subsequent alteration of diagnosis in many cases, became reduced to 1,431; for the purpose of this analysis a further reduction was made and a certain number of case-reports eliminated by reason of the limited information which they offered. Finally, and after still further eliminations resulting from information received during the enquiry, the number of cases selected for general purposes of analysis is 1,273.

With such an amount of material available we thought it becoming to attempt some form of classification of the cases in question and we have adopted the anatomical one originally used by MacNalty. One reason for using this classification again is to link up the first report with the present one, and, by following the same systemic lines, to offer a comparison of the results arising from the two analyses; also, we know of no better classification for general purposes. We refer gratefully, however, to the inspiration derived from Walshe's particular scheme of classification which we have followed closely in the discussion of the nervous symptoms.

The general classification (MacNalty's) is reproduced here for convenience of reference.

I. Types in which there is general disturbance of the functions of the central nervous system but without localisation.

II. Types where, in addition to general disturbance, there are various localisations in the central nervous system.

- A. Clinical affections of the third pair of cranial nerves.
- B. Affections of the brain-stem and bulb. Local lesions of other cranial nerves.
- C. Affections of the long tracts.
- D. Probable involvement of the cerebellar mechanism.
- E. Affections of the cerebral cortex.
- F. Types indicating some evidence of spinal cord involvement.
- G. Types indicating possibly an affection of peripheral nerves.

III. Mild or so-called abortive cases.

TYPE I.

In Type I. have been placed 78 cases or 6.7 per cent. of the total series and in the majority of such cases the general nervous symptoms were of a "negative" character and consisted mainly of lethargy and asthenia. In some patients the general constitutional symptoms were severe and the mortality in this class (Type I.) was as high as 40 per cent.; on the other hand some cases might almost go into Type III. but for the fact that the lethargy was prolonged and the asthenia severe. In patients whose convalescence was slow, mental dullness was the usual after-effect reported, but in five instances eye palsies or other localising signs were mentioned in the after-history summaries, showing that a certain proportion of cases, at first considered

*The classification above will often be referred to in this report (and more especially in tables) by means of the numerals, "I," "II." and "III." The plus and minus signs which frequently accompany these numerals in the classification of a particular case, are further intended to indicate—not the anatomical sub-division in which the case might be placed—but the clinical complexion of its nervous symptoms. The plus sign (+), indicates shortly that symptoms of *irritation* were present; in cases coming under Group I. these symptoms would be referable to the higher centres alone, while in cases belonging to Groups II. and III., the sign would indicate the presence of localising symptoms of an irritative nature with, or without, emotional excitement. The minus sign (−), on the other hand, bears a similar significance as regards *paralytic symptoms*; in relation to cases in Group I., again, the symptoms of paralysis would refer only to the higher centres to a paralysis of the emotions, while for cases included in Groups II. and III., the minus sign would indicate the occurrence of localising paralysis with, in most cases, also, an involvement of the higher centres. Thus, the symbol "I. + −" means that the case in question, while belonging to Group I., was characterised more particularly by symptoms of both an irritative and paralytic character, say delirium and somnolence respectively. A case noted as "II. − − +" would be one coming under Group II., and in which localising signs both paralytic and irritative (e.g., ophthalmoplegia, facial paralysis, paresis of legs and tremors) were present, the former being most in evidence as shown by the duplication of the minus sign. In such a case the higher centres, too, would in all probability be involved in one or both directions for, as may be noted here, lethargy was present in 95 per cent. of the cases in our series; delirium also (as representing irritative symptoms of a higher order) was very commonly present.

It should be further explained that the terms "positive" and "negative," which will frequently occur in this report, are used in the sense in which they are employed by Walshe (1,065, b), who attaches to these phrases the significance intended originally by Hughlings Jackson. Thus—quoting from Walshe—"positive" symptoms being expressions of exalted function either from irritation or from loss of higher control, and 'negative' symptoms expressing depression or loss of function from destruction of nervous structures or otherwise." In this special sense a squint, for example, would be classed as a "negative" symptom though it might be just as positively a feature of the clinical syndrome, as, say, such a "positive" symptom as tremor.

Where they are used in this report the signs "+ " and "− " may be taken as synonymous respectively with the expressions "positive" and "negative."

as coming into Type I., became subsequently disqualified owing to the late appearance of localising signs.

TYPE II.

The great majority of cases, amounting to 90 per cent. of the whole series, are classed under Type II. The localising signs may be conveniently divided into (a) those of a "negative" nature and would include the various muscular palsies and other forms of diminished action and (b) "positive" manifestations of exalted or disordered activity and of irritation, such as tremors, rigidity and pain. These various localising signs are considered individually at a later stage; here it may be said that while the various "negative" signs have special reference to the cranial nerves and particularly to the IIIrd and VIIth the "positive" signs are not so restricted in their reference but indicate a more extensive and a more capricious action on the part of the virus.

Group A: Clinical affections of the third pair of Cranial Nerves.—Many a case in this large grouping fits in well with the classical description, quoted at the beginning of this section, in which fever, lethargy and ophthalmoplegia, so commonly present in this type as a notable trinity of symptoms, are fully recognised. The group, however, is not exclusive in this respect, and, while in many cases the paralytic symptoms are confined to the muscles of the eye, in an equal number of cases other muscles are affected and particularly those of the face. There may, also, be much variety in the manner of onset, in the degree of mental excitement or depression and in the general toxæmic symptoms. In the great majority of cases "positive" symptoms, either general, localising, or both, were present, and the mortality ranges round 50 per cent., thus:—

TABLE 25.
TYPE II., GROUP A.

Clinical type (see footnote p. 55).	Number of cases.	Percentage of total number in group.	Case mortality rate (cases in 1920).
IIA, + —	725	69	49 per cent.
IIA, —	213	20	47 "
IIA, ++ —	79	7	49 "
IIA, + — —	28	2	32 "

Group B: Affections of the Brain-stem and Bulb.—This group shares with the preceding one a large number of cases, but here facial paralysis may be regarded as the salient localising sign, just as oculomotor palsies are the predominating feature of cases in Group A. The eye and the face are very often affected in the same patient, but in a third of the facial palsy

cases there were no symptoms of IIIrd nerve involvement. In addition to the facial, other cranial nerves may be implicated in cases belonging to this group and such instances are referred to later; here it may be stated that the present series of reports furnishes instances of each cranial nerve, except the first, being affected.

Group C: Affections of the Long Tracts.—Using the presence of tremors and a positive Babinski sign as criteria it may be said that the rubro-spinal and the pyramidal tracts were involved in a considerable number of cases, but in none to the exclusion of other parts of the nervous system; a positive Babinski was recorded in some 47 cases, while tremors were reported in 250 patients.

Group D: Ataxic Type.—There is mention of ataxia in the case of some 24 patients, chiefly in respect of the legs, but it is doubtful whether the condition was due to cerebellar causes in all of these instances; in some cases it appeared probable that the difficulties in walking were due to a paresis of the legs, together with, possibly, some associated diplopia and giddiness. In three patients, however, the gait strongly suggested cerebellar ataxy; one man had, in the early stages of a long illness, very great difficulty in walking, chiefly on account of a tendency to fall forwards and a feeling as if he were intoxicated: there were two other somewhat similar cases. Three patients displayed considerable asynergia in all the limbs, and in the case of a school-master who had "marked ataxy of arms and legs of cerebellar type" there was also an articulative ataxia. Some three or four cases in which there was also severe vertigo should possibly be placed in this group as instances of cerebellar ataxia.

Group E: Affections of Cerebral Cortex.—A greater proportion of cases than formerly now find inclusion in this group and by reason of motor manifestations alone the cerebral cortex in some 40 cases of the present series may be considered to have been affected; convulsions, hemiplegia and Jacksonian epilepsy were the chief signs noted. Possibly some of the defects in speech and memory may have been also due, in certain cases, to cortical lesions.

Group F: Cases indicating some evidence of Spinal Cord involvement.—In this group are tentatively placed some 14 cases in which were present a certain number of the signs significant of lower motor neuron paralysis. As noted elsewhere, there were over a hundred cases in which the knee-jerk was lost for a time, but that sign, by itself, has naturally not been taken as a passport to this group. In a few instances, however, the abolition of this reflex was associated with a paralysis or paresis of the legs; in three adult patients there was a flaccid palsy of the limbs with lost deep reflexes and general nervous symptoms, and in two cases marked wasting of leg and arm muscles is recorded. It does not appear that any one class of case is

peculiarly liable to cord involvement and in those cases where such occurred there were present both general symptoms and the localising signs of a higher order; we suspect that the cord may be affected more often than the schedule reports seem to show.

In the literature the subject has received notice from Bassoe (59e), who describes two cases in which cord symptoms predominated, and from Gerlach (377), Pardee (777), Stiefler (994) and others. Gerlach recalls the fact that von Economo first pointed out the possibility of the cord being involved and himself cites two cases in which there was marked involvement of the spinal cord; in both instances it was the lower dorsal and upper lumbar regions that were particularly affected.

Group G : Cases in which the Peripheral Nerves were possibly affected—(Polyneuritic Type).—There would seem to be a clinical warrant for including some 22 cases in this group, but whether the nerve pains were always due to a primary neuritis, or not, is open to question and possibly some of these cases should rather be placed in the preceding group. In some three or four instances the nerve roots certainly appeared to be affected; in one patient there was severe "pins and needles" sensation along one side of the spinal column; two patients suffered from intercostal neuralgia and in one of them herpes zoster subsequently appeared.

In 10 cases the upper limbs were affected and were subject to such conditions as neuralgia, numbness and tingling, anæsthesia, &c.; no instance of wrist-drop is recorded, but in one case there was palsy of the fingers of one hand with anæsthesia. In the remaining cases the lower limbs were affected in similar ways and foot-drop was mentioned twice. In some instances there was a symmetrical implication of nerves suggesting the peripheral neuritis of toxic origin and in the case of a schoolboy the neuritis observed in both fore-arms was at first thought to be post-diphtheritic.

In all the cases of this group general nervous symptoms were present, including some degree of lethargy, and in the majority there were also present some of the more usual localising signs.*

TYPE III. (Mild Cases).

The schedule reports, based on notified cases, probably afford no exact indication of the extent to which the disease may occur in a mild or abortive form. From the present series of 1,273 cases we have picked out 31 as instances of mild or abortive forms and this gives a percentage only very slightly in excess of that found in the first series of cases. The expression "mild" has been

* In a fatal case of one month's duration, reported in 1922, the illness began with polyneuritic symptoms, which were at first attributed to "rheumatism"; no other signs appeared until a week or so before death, when wild delirium and maniacal symptoms appeared, to be followed, later, by a deepening lethargy, dysphagia and complete incontinence; the temperature rose to 106° F. on the last day.

applied to instances in which some of the earlier symptoms of a well-marked case are present in slight degree, in which the course of the disease is comparatively brief (2-3 weeks), and where the patient quickly resumes his work without feeling any after effects. Such cases are usually recognised only at times when the disease is prevalent and the best instances are seen where different members of an institution or a household are involved in a special outbreak; in this connection the Derby outbreak and some smaller groupings of cases mentioned in Table B, Appendix II., may be specially mentioned. (See also remarks under Sec. II., B. 8.)

Apart from information derived through purely formal channels there is also evidence that the existence of mild and of atypical cases is being recognised in various quarters; the guise under which the disease is suspected, however, seems to vary with the observer.

Thus Dr. Willoughby Gardner of Shrewsbury, in a private communication, describes a set of cases occurring, he thinks, very commonly in Shropshire and characterised by the following signs and symptoms:—

"1. Fever, sometimes sudden, sometimes gradual or even insidious in its onset, of varying type, uncertain duration and prone to recrudescence.

"2. Sore-throat of a peculiar character: there is slight swelling but very marked congestion of the fauces and pharynx; the colour is generally a deep crimson, but sometimes is almost vermilion. There is comparatively little swelling of the tonsils, but whitish patches of exudation are to be seen on tonsils, fauces and pharynx. These patches sometimes look very like diphtheritic membrane and occasionally can even be stripped off, but in none of the cases has the specific organism of diphtheria been found. These throats tend soon to become dry, and are often associated with a red, bare, swollen and dryish tongue.

"3. Gastro-intestinal disturbance, generally vomiting and diarrhoea.

"4. Broncho-pneumonia, with sharp crepitations at the base of the lungs.

"5. Often an erythematous rash on the body and legs, chiefly in the form of red patches, but varying greatly and of short duration. This is by no means always noticed, possibly because of its transient nature and because it is not looked for.

"6. Marked prostration and slow recovery."

It will be noticed that in this list of symptoms there is no reference to any implication of the nervous system, and that cases presenting such a clinical picture can hardly be considered as mild. Dr. Gardner supposes, however, that "we have a very widespread disease in our midst, which is itself new in character, which is markedly infectious and therefore tends to be fairly

common, and which occasionally attacks the higher nervous system and especially the mid-brain, producing the symptoms to which we have given the name encephalitis lethargica. I think it is quite possible that a far milder form of this disease is very common—indeed, and that it is characterised by slight sore-throat and fever and some mild gastro-intestinal symptoms." Dr. Gardner cites cases in which the throat symptoms he described were either followed or accompanied by the nervous signs of encephalitis, and he quotes instances where in the same hospital ward there was a series of mild cases of sore-throat occurring at a time when well-marked cases of encephalitis lethargica were under treatment. An interesting point in connection with the peculiar throat conditions observed by Dr. Gardner is that the patient makes no complaint of any faucial pain or discomfort.

Then Dr. Montague Dixon, writing in March 1921, says, "I am under the impression that there are quite a number of encephalitis lethargica cases overlooked," and he suggests that cases of illness marked by the following symptoms should be regarded with suspicion:—

- (1) Sudden onset generally with severe vomiting.
- (2) Headache.
- (3) Diplopia (without perceptible squint or nystagmus).
- (4) Pain in front of chest or upper abdomen.
- (5) Catarrhal symptoms (cough and rhonchi).
- (6) Insomnia at night, with less restlessness and apparent sleep by day.
- (7) Mental irritability.

A rather different set of circumstances was brought to our notice by the late Dr. W. H. Bowen, who, in the course of his duties as a Poor Law Medical Officer in the Lambeth Districts I.-VI., noted an affection among children which he considered was a mild form of encephalitis lethargica. The ages of the patients varied between 12 months and 12 years and some 20-30 cases were observed during February and March, 1921, i.e. towards the end of a period which was marked by an unprecedented rise in the number of notifications of encephalitis lethargica.

The general symptoms common to most of the cases were slight fever, asthenia, constipation and headache, while some faucial catarrh was commonly seen. Drowsiness, together with delirium, were characteristic features and in some cases there were diplopia, tremors and paresis of limbs. No physical signs in the chest or abdomen were noticed and all the cases ran a short course to recovery.

The clinical notes of five cases will be found in Appendix I., cases K.-O.

Ambulatory Cases.—There are eight cases mentioned which may be considered as belonging to this category; some of them refer to patients who had disregarded the symptoms of an insidious onset until the onset of diplopia had caused them to

seek medical advice. Or, there may be a lull between the onset and the declared disease during which the patient partially resumes his work until he becomes seriously ill. In a few other mild cases the patient is not incapacitated sufficiently to keep him in bed or even long away from work. In Appendix I. (Case C.) will be found extracts from the diary of a professional man who continued his daily journeys to London for a week though handicapped severely during that time with diplopia and an increasing lethargy.

B.—INCUBATION PERIOD.

The data available for estimating the length of the incubation period are still few, because undoubted examples of case-to-case infection are scattered very sparingly through the records; nevertheless the reports of cases notified up to date do afford perhaps sufficient material upon which to form a provisional hypothesis. In the Derby outbreak the incubation period was short if it be granted that in this outbreak infection was derived from established cases through personal contact. Some of the inmates who were first attacked, however, probably became infected through a common source, though as MacNalty (629g) suggests an unrecognised "abortive" case or "carrier" among the inmates may have introduced the focus of infection into the home. It may be recalled that after the occurrence of two cases on August 14th, the subsequent events were as follows:—

TABLE 26.

August -	15th	16th	17th	19th	27th
Cases	2	4	2	1	1

It will be seen that on the basis of a case-to-case infection the incubation period in all but the last case ranged between 24 and 48 hours; the last inmate to be attacked, however, succumbed to the disease eight days after the penultimate case occurred and 13 days after the two first. Possibly the short incubation period of one to two days signified the intensity of the virus during the first few days of what was undoubtedly—as shown by the high mortality rate and short duration of illness, in the fatal cases—a very intensive outbreak; the longer interval that elapsed between the last two cases may possibly be explained also by a reduction in the potency of the virus to a more usual degree.

Some of the features in the Derby outbreak were reflected in a small way by the experiences of a doctor's family in Lincolnshire. Here a healthy baby, nine weeks old, suddenly became ill on 14th February with loss of colour, profound unconsciousness, laryngeal stridor and a cessation of respiration. Attacks similar

to this recurred every 20 minutes for a space of 36 hours and the infant was only kept alive with oxygen and artificial respiration. After two days there was apparent recovery, but the crisis appeared again and this time the infant experienced two typical infantile convulsions of the clonic type. In the meantime the father was not feeling well, being feverish and very drowsy; he suffered also from sore-throat and intense headache and took to his bed on the 16th, but was about again in 48 hours. Then a boy of eight, who had been suffering from slight laryngitis, was attacked on the 17th, when he was found asleep on the floor. The onset in his case was rapid and marked by complete loss of appetite, furred tongue, slight fever, and vomiting which recurred often. He had become very lethargic and for 24 hours could not be roused by any means; the pupils were dilated (in the sister's case they were "pin-point") and insensitive to light and there was a marked tendency to retention of urine. The fever early abated and the pulse rate was low. The baby is said to have had a "relapse" in April, but all these patients have since completely recovered. This little domestic epidemic had some of the explosive suddenness of the Derby outbreak and, if case-to-case infection was at work, the incubation period was again very short, 24-48 hours. In the particular circumstances of this household it is improbable that all three patients were infected at a common source; it seems also unlikely that the baby—who had not mixed with other children—brought in the infection from outside. The onus falls much more naturally upon the father, who was particularly liable to the risk of infection by reason of his work, and who had, indeed, six weeks previously, seen in consultation a doubtful case of encephalitis lethargica; it is quite possible therefore that he became infected first and transmitted the virus to his children before himself succumbing to a clinical attack. This implies that he was infectious during his incubation period and that while his own attack was an abortive one, the virus in the case of the children found a soil in which it displayed exceptional virulence. It is significant that the father suffered from sore-throat and possibly the throat was infectious before it was subjectively sore.

Of the other examples taken from Table B., Appendix II., mention may be made of the following:—

Instance No. 4.—An elderly lady, Mrs. H., was admitted into a Plymouth nursing home on 17th December, 1918, suffering from encephalitis lethargica which began twelve days previously. There were no other cases of encephalitis in the home until, on 26th December, 1918, the nurse who had been in nightly attendance on Mrs. H. became ill with symptoms similar to those of her patient. There was no suggestion that patient and nurse had been affected by any common circumstances outside the hospital previous to Mrs. H.'s admission; as the nurse became ill also while still in attendance upon her patient, it is not easy to say accurately when she became infected, but, assuming that

it was very soon after she took charge of her patient, the period of incubation would then be nine days.

Instance No. 6.—A woman residing in Bristol visited her father (who lived outside the city) on the day—5th February, 1919—that he was removed to hospital suffering from encephalitis. On 8th February, the daughter complained of headache and tiredness, but returned to work on the 10th and 11th. A febrile attack started on the 12th, she was removed to hospital on the 17th and died on the 20th. Here the incubation period would be 3-4 days if the headache and tiredness were part of the disease, or seven days if this indisposition was irrelevant. (*Vide* Bibl. I., 1006).

Instance No. 5.—Here, a boy aged 13 and his sister aged 12, who shared the same bed in an impoverished and insanitary home and who were attending the same school, became ill on 24th January, 1919, and 7th February, 1919, respectively. The boy was removed to hospital on 31st January, 1919, i.e., eight days before his sister showed symptoms, and, if he infected the girl, her incubation period would vary between 14 and 8 days: it is quite possible, however, that they were both infected from the same source, though not necessarily at the same time.

This element of doubt also exists in *Instances 9, 11, 18 and 28* where in each case the two persons affected were relatives living under the same roof. Moreover, if case-to-case infection is taken for granted it is still impossible—in these particular instances—to say at what period of contact the infection was passed on; the difference in days between the dates of onset in the four instances respectively were 17, 4, 18 and 13.

Instance No. 16 concerns a married woman, at Lymington, who developed a mild attack of encephalitis lethargica on 15th February, 1920. A sister, also married, had previously come to Lymington from Bournemouth on a visit, was taken ill on 13th January and moved back on 28th January to Boscombe Infirmary, where she died of encephalitis lethargica on 3rd February, 1920. The two sisters, though not sharing the same house, had been often in contact, and the incubation period of the surviving sister's illness would be somewhere between 18 and 33 days.

More direct perhaps is the evidence provided by those newly born infants (*Instances 22, 30 and 31*) whose attacks of encephalitis lethargica were maternal in origin. One such child, born while the mother was seriously ill with encephalitis, showed similar symptoms at the 10th day or so of life; another developed the disease after 14 days, when a scarlatiniform rash and other symptoms, similar to those exhibited by the mother, appeared. In the third instance there was a much shorter interval between the birth of the child and the beginning of its illness suggesting placental infection: the infant "cried lustily at birth, but from then onwards was abnormally quiet—sleeping most of the time." The eyes remained closed for a fortnight or so, and during this time there were marked twitchings in the left arm and left leg;

these movements reflected those of the mother's illness in character and localisation: both mother and child eventually recovered.

From such data as have been set out, then, it seems that the incubation period may vary within fairly wide limits; from one or two days to two weeks and even more. Boyd (121b) quotes three cases in all of which the incubation period appeared to be two weeks. Of one he writes, "a rig-driver, who lived in a small town where there was no encephalitis, drove a doctor thirty miles into the country to see a case of the disease, and stayed for three hours in the house. Two weeks later he developed lethargic encephalitis."

C.—ONSET.

The protean character of encephalitis lethargica, which with increasing experience has become more and more appreciated, is exemplified by the number of ways in which, as our reports show, the disease may first make itself manifest. These various modes of onset, however, may be broadly considered under two headings: the first group comprises those cases in which the onset was *sudden* without apparently any premonitory signs, while the second and larger group comprises those cases in which there was a more or less *gradual* and insidious approach to the actual invasion.

1. **Cases with acute onset.**—In the early series of cases investigated by MacNalty, a sudden onset was very exceptionally recorded, but among the cases notified during 1919 and 1920, there are an appreciable number of instances (about 6 per cent.) of acute onset without any history of premonitory signs. In the Winnipeg outbreak, Boyd (121b) considered that a sudden onset was the rule, and, though this is further than most observers are prepared to go, it is clear from the literature that cases with an abrupt inception are now observed more frequently than hitherto.

The sexes were equally involved in these acute cases and the mortality rate was only slightly in excess of that for the whole series. The age incidence as regards acute onset is shown in the following table:—

TABLE 27.
Cases with acute onset.

Age period.	Number of cases.
Under 1	1
1-5 years	3
5-10	4
10-15	4
15-20	8
20-30	5
30-40	19
40-50	16
50-60	9
60-70	6
70-80	1
	76

The greatest number of cases occurred during the mature age period 30-50, and it is noteworthy that in 20 instances the illness began with an abrupt loss of consciousness, which in some cases was strongly suggestive of an apoplectic seizure. Thus a woman of 38 suddenly sank into unconsciousness after a hard day's washing and became deeply comatose while at stool; she eventually recovered after exhibiting other signs of encephalitis. Another woman of 46 lost consciousness while at dinner and subsequently developed fleeting palsies of the right hand and foot. In a third case, that of a doctor's wife, the attack of unconsciousness was equally abrupt and accompanied by fever and ptosis; in all these instances the patient eventually made a good recovery, but in others a fatal result ensued. References to these apoplectiform cases have appeared in the English journals, notably by Purves Stewart (993) and Box (120), while Branfwell (128) includes such cases as forming one of his four general types of encephalitis lethargica. It is obvious that this method of onset is one that has to be carefully kept in mind when forming a diagnosis.

These cases beginning with unconsciousness, however, are not confined to patients of over 30; thus, a youth of 21 became suddenly unconscious while in a train; another, aged 16, collapsed in an unconscious state while working in a factory and died in five days, when multiple hæmorrhages were found in the brain: the first of these cases was diagnosed in the Royal Infirmary, Manchester, and the second in Cardiff Infirmary. Closely resembling the cases which have just been described are those in which an attack of faintness, sometimes accompanied by vomiting, was the first indication that anything was amiss, and such cases, together with those resembling apoplectic attacks, constitute about 38 per cent. of the instances of sudden onset. In the remainder of the cases the onset was particularly associated with some one or more of the symptoms that form the preliminary outlines of the clinical picture in the gradually developing cases. Thus giddiness and vomiting, with or without a previous headache, were the initial signs in a fair proportion of these cases, a good instance being afforded by the experience of a house decorator who, while looking at a shop window, became suddenly giddy and vomited so that he collapsed and had to be taken home. Vomiting was by no means a symptom restricted to young subjects, but rather the reverse. With almost equal frequency are recorded cases in which a patient, apparently in good health, is suddenly made aware of some defect in vision; when this defect takes the form of diplopia the consequences are sometimes startling, as in the case of the land girl who, while returning with her team of horses, suddenly became diplopic and fancied she was in charge of a drove of animals.

Our series also provides instances of individuals who suddenly became the victims of strong emotional or psychological disturbances. In a woman of 45 the illness was ushered in with a fit resembling

an outburst of hysteria, though the patient was described as an "always robust, quiet living woman with the strength of a man"; a clerk also, aged 37, suddenly behaved hysterically and passed into a condition of catalepsy, this being the second occasion within a year that she had so behaved: both the patients died. A boy of 16 while engaged in gardening at the Gordon Boys' Home suddenly left off his work, began to dance and showed signs of dementia; the early stages of his illness were also marked by much delirium, and exaltation, but he made a good recovery. The disease in a child of nine months started with a convulsion. On the other hand, in two or three instances, it was a sudden paresis or even paralysis of some skeletal muscles that first attracted attention. An interesting example of such a case is reported from Scotland where a girl "had a sudden seizure characterised by paresis of right foot while playing golf on the 27th May and was driven home in a cab"; she died on 24th June. Among some cases investigated in South Wales was that of a man who walked up to his doctor's surgery to ascertain the reason why the muscles on one side of his abdomen were "all on the twitch."

In another series of cases the precursory symptoms were those of a severe general infection and included rigors, sudden elevation of temperature with delirium.

2. Cases with more or less gradual onset.—In the great majority of patients the illness began in a gradual and often indefinite manner, in some cases with symptoms of a general systemic infection, but perhaps more often with signs that pointed definitely to involvement of the nervous system. In the first class of cases a general malaise including headache, loss of appetite, constipation, languor and disinclination for work, are frequently mentioned and in many such cases the prodromata are definitely described as those of influenza.

Headache.—Pain referred to the head is mentioned more often than any other symptom and—including those cases labelled as influenzal in onset—is reported in just over 50 per cent. of the cases in which initial symptoms are described; this percentage is considerably lower than that resulting from the analysis of the first series of cases.

The symptom is dealt with here as one of the manifestations of a general infection; the fact, however, that the headache is not uncommonly mentioned as being very severe and as representing alone the prodromal stage would suggest that sometimes it has more specific indications. In a certain number of cases the headache, appearing at first as a solitary symptom to be joined later by symptoms of a general malaise, preceded the fully developed disease by some weeks, while in 11 instances it was regarded as a predisposing condition of some months' duration. In the majority of records the exact cranial localisation

of the pain is not specified, but such information as is available is tabulated below:—

TABLE 28.

Headache: Character and Localisation.

—	Unclassified.	Frontal.	Occipital.	Vertical.
Headache - - -	403	27	22	8
Severe headache - -	42	5	4	1
Very severe headache -	36	6	4	—

Although the reports are seldom very explicit on the point, it would seem from some of our own observations that in many cases the headache remains a constant symptom during the declared illness as long as the patient is conscious and capable of complaining. In about 40 cases the headache was associated with retraction of the head or with cervical rigidity and in many of these cases Kernig's sign was present.

Vomiting.—The next most frequent symptom observed was vomiting; it was present in 17 per cent. of cases, which is a higher proportion than that which obtained in the first series. Here again it is not always clear as to whether the vomiting was due to direct action of the toxin on the cerebral centre as part of the general infective process or whether it was a form of cerebral vomiting due to reflex action on the centre. In a certain number of cases the act bore no relation to the taking of food, but occurred suddenly and without nausea; in the majority of instances, however, the vomiting was not of this cerebral type, though children by no means figured prominently in these cases. With diarrhoea vomiting was only associated on 17 occasions, but in the case of a medical practitioner (see Appendix I, Case B.), these two symptoms definitely formed the starting point of his illness.

Constipation.—Although this condition is mentioned as an initial symptom in 63 cases, it is probable that in some of these instances it should more correctly be referred to the symptomatology of the declared disease, where it will be more fully discussed in this report.

Pains.—In 128 instances the prodromata included pains distinct from those connected with the head and from neuralgia and in many cases these were referred to as a general aching or "pains all over"; in 37 out of the 128 cases the discomfort was definitely localised in the abdomen and for the rest the back, shoulders and slopes of the neck were most commonly implicated. The pains in the class of cases here under reference were as a rule dull, indefinite and myalgic in character, but with no other

particular distinction; with the general pains *stiffness* was often associated.

Asthenia.—Under this heading may be grouped the 76 cases in which “prostration,” “feeling of weakness,” “legs giving way” and a general lowering of muscular power were noted; the general weakness in these instances is very possibly aggravated by the anorexia which seems to be more marked in the initial stages of the disease than later on, when, according to many observers, the patient, so long as he is capable of swallowing, takes plenty of nourishment.

Pyrexia.—Fever is mentioned 106 times among the initial symptoms and it certainly may be an early symptom, associated with the general malaise; it seems more natural, however, to regard it as a sign that the disease has definitely declared itself and it will be discussed later in this connection. With it will also be postponed references to *anorexia*, *condition of tongue*, &c.

Sore-throat.—The actual references to sore-throat as an initial sign are few and far between and the reports therefore do not bear out the conclusions reached by some observers as to the importance of this condition. It is quite possible, however, that local signs in the throat existed more often than the figures would indicate, for in these cases where the clinical course has been closely observed, sore-throat has been reported much more frequently—as in the case of the Derby outbreak. If, too, the striking faucial condition which is described by one experienced clinician is, as he says, not at all appreciated by the patient himself, it seems likely that the sore-throat often escapes attention in a disease characterised so often by much more striking signs.

Bronchial Symptoms.—In 2.5 per cent. of the cases a bronchial catarrh with cough was noted, but it is doubtful whether these respiratory symptoms have anything more than an accidental place in the early symptomatology of encephalitis.

Conjunctivitis again is only mentioned in 18 cases, while in MacNalty's series it seems to have been a very common initial symptom and one recommended as a help in the diagnosis. A certain number of instances of conjunctivitis may be concealed in the general description of the onset as “like that of influenza,” but even so the comparative disappearance of this easily observed condition from the group of initial symptoms as first drawn up is somewhat striking.

Lethargy.—Chief among the initial symptoms suggesting specific infection is lethargy which, in greater or less degree, was observed in some 440 cases (38 per cent.); in a considerable number of instances, too, where the history was carefully investigated, some degree of lethargy was actually the first warning that all was not well. Very commonly this state of things was appreciated by the patient's family or employer before he himself made any complaint or considered that his state was anything

more than a passing indisposition. Thus a wife would state that her husband sat over the fire brooding and taking no interest in the children “and other domestic affairs”; that he seemed “dazed-like” and irresponsive to any attempts at interesting him. Employers would complain that a good workman had apparently begun to get slack and was no longer “on the spot” as he used to be; or it would be noted of a child that she seemed unusually stupid at school and no longer wanted to play with other children. In connection with these objective phenomena, it may be mentioned that occasionally an unwonted pose of features is an early sign, as in the case of a medical man whose changed expression of face was first pointed out to him by his patients. In other cases the constant desire to sleep was fully appreciated by patients who were “always trying to keep awake,” and of some of whom it is reported that they sought medicine for the attainment of this object. In one case medical advice was invoked by a wife because her husband would always fall asleep when riding with her on the top of a bus and, while she feared he might fall when descending from the vehicle, she was also disturbed by the suspicion that their fellow passengers attributed the somnolence to alcohol. The intermittent type of somnolence which was characteristic of this patient was also exemplified in the case of two others, whose condition was suspected from the evidence of interrupted tobacco smoking; in one instance the man's trousers were riddled with holes, the result of constant impact with live tobacco ash, and in the other the smoker's fingers were charred. An important feature indeed of these early manifestations of somnolence is the dramatic suddenness and inconsequence with which they occur; thus a man was found asleep standing at a tap with his ablutions but half performed, and it is commonly reported that a patient “fell asleep” while eating his dinner or in the middle of a conversation. In very many cases the somnolence is at first associated with headache, pain over and behind the eyes and general hebetude; then, with the appearance of other characteristic signs, it gradually passes into the stupor of the declared disease.

Instances of sudden loss of consciousness followed by stupor and coma have been previously discussed, but it may be mentioned here that such cases did not invariably prove fatal, but from the depths of unconsciousness the patient emerged and, as it were, retraced his steps through stupor and lethargy to a normal state. In two children living in the same house, the illness suddenly began with deep sleep which continued for 24 and 48 hours respectively, and from which both patients then emerged apparently normal.

Mental State and Disposition.—In marked contrast with these patients in whom slight somnolence and mental torpor gradually deepened into lethargy and stupor were those who at first showed signs of irritability and restlessness. “Annoyed over trifles,” “loses his temper,” “irritable and uncertain,” “teases other

children" are expressions used to show the change of character that was noted on many occasions, and some of these cases were associated with insomnia, which is mentioned 42 times as an early symptom. The insomnia was accompanied by delirium in most cases and occasionally the latter state was marked by an urgent desire to leave the house and resume work; thus one man was found trying to communicate with his employers over the telephone and the character of his efforts led the listeners to assume rightly that the man was ill; in another instance an elderly man, who had been unwell for a day or so, got up in the night and tried to resume his work in a sewer. Reference has been made previously to a case that began suddenly with symptoms of mania and more than one patient has been considered at first as a "mental case" and put under restraint. Very occasionally the character is temporarily changed for the better, as in the patient who became effusively grateful; in another case (from the literature) a suddenly acquired devotional habit led to further observations being made. In all, a history of some early change of character was obtained in 122 instances. As pointed out by MacNalty, not a few cases of encephalitis lethargica are characterised by an emotional introduction and the present series presents many examples; cases beginning with an hysterical outburst have already received mention and there were others in which crying, shouting, laughter and other signs of depression or exaltation occurred.

Speech.—In a few cases a change in the manner of speaking has attracted notice during the early stages, and in one patient a difficulty in articulation was noticed three weeks before any other prodromal symptom.

Defective Vision.—Not a few cases of encephalitis lethargica have first been diagnosed in the consulting room of the ophthalmic surgeon, and of all the premonitory warnings of this disease, none perhaps is so significant as a sudden and unexplained inability to see properly, which at once alarms the patient and causes him to seek advice. The most usual explanation of "eyes going funny" is probably *diplopia*, which is mentioned specifically among the initial symptoms in 170 cases; "defective vision" occurs in 61 cases and squint is recorded in 31: "defective vision" probably includes a large number of cases of *diplopia* so that this symptom occurred early in nearly 20 per cent. of the cases, which is the proportion observed in the first series. The double vision in this stage most generally appears without any obvious squint, but is often accompanied by headache and other initial symptoms, particularly dizziness. The condition in most cases is probably binocular, as in the example of a man who when cycling to and from his work found that he could control his movements by bandaging up one of his eyes. Although the condition may continue well on into the illness and be subsequently associated with a paralytic strabismus, it perhaps more frequently clears up before the graver symptoms supervene.

Among other early ocular symptoms *squint*, *ptosis*, *photophobia* and *nystagmus* are all mentioned, but they are recorded far less frequently than *diplopia* and are generally observed in a more advanced stage of the disease. "Staring eyes" and "eyes looked queer" are expressions that occur in some of the histories and probably refer to some form of strabismus or they may be related to pupillary reactions.

Vertigo.—As mentioned previously, some of the most striking of the cases with acute onset begin with an attack of dizziness and this symptom is mentioned 106 times, generally in company with headache and often with vomiting and *diplopia*; in a few cases it was also associated with deafness and buzzing in the ears. It does not seem clear whether the condition is toxic in origin or due to definite involvement of the vestibular portion of the 8th nerve; in many cases it is probably closely concerned with the *diplopia*, though not directly due to this ocular defect.

Inco-ordination.—The instances in which lack of co-ordination is mentioned as an early symptom refer chiefly to the legs and in many cases this symptom was associated with vertigo and deficient vision and may possibly have been cerebellar in origin; in other patients the ataxy would seem to have been a manifestation of general asthenia.

Involuntary Movements.—In 4.4 per cent. of the cases, involuntary movements were noted; these were generally described as twitchings and most often affected the facial muscles.

Myoclonus is mentioned five times among the record of initial symptoms and reference has already been made to the curious unilateral spasm of the abdominal muscles. Tremors are also represented and usually involved the upper limbs; "chorea" is mentioned as an early symptom in four cases, while in 17 instances a fit occurred. In a girl of 16 the illness began with an attack of Jacksonian epilepsy affecting the right arm.

Neuralgia and Neuritis.—In quite an appreciable number of cases acute neuralgia, from the patient's point of view, overshadowed the other prodromata. In the majority of instances it was associated with nerves arising from the upper region of the cord, or with cranial nerves, so that the arms, shoulder girdle and face were the regions most commonly affected.

In nearly all the cases the pain is described as severe and usually "shooting" in character, while in some instances there was a progressive involvement of adjoining regions. One patient first complained of "pins and needles" all over the body and in another similar sensations were limited to the spinal region; in three cases there was local formication in the hand and in one patient there was "pins and needles" in the legs with numbness in the thighs and arms.

Facial palsy was noted as an early symptom in sixteen cases; in two instances the condition was manifested by dribbling of saliva and in some others was preceded by facial twitchings.

The relative frequency with which the various conditions described above were recorded in the schedules under the heading "initial symptoms" is shown in the accompanying graph.

There remain various other symptoms which were occasionally reported, all of which, however, occurred in less than 1 per cent. of the cases; these are tabulated below:—

TABLE 29.
Infrequent Initial Symptoms.

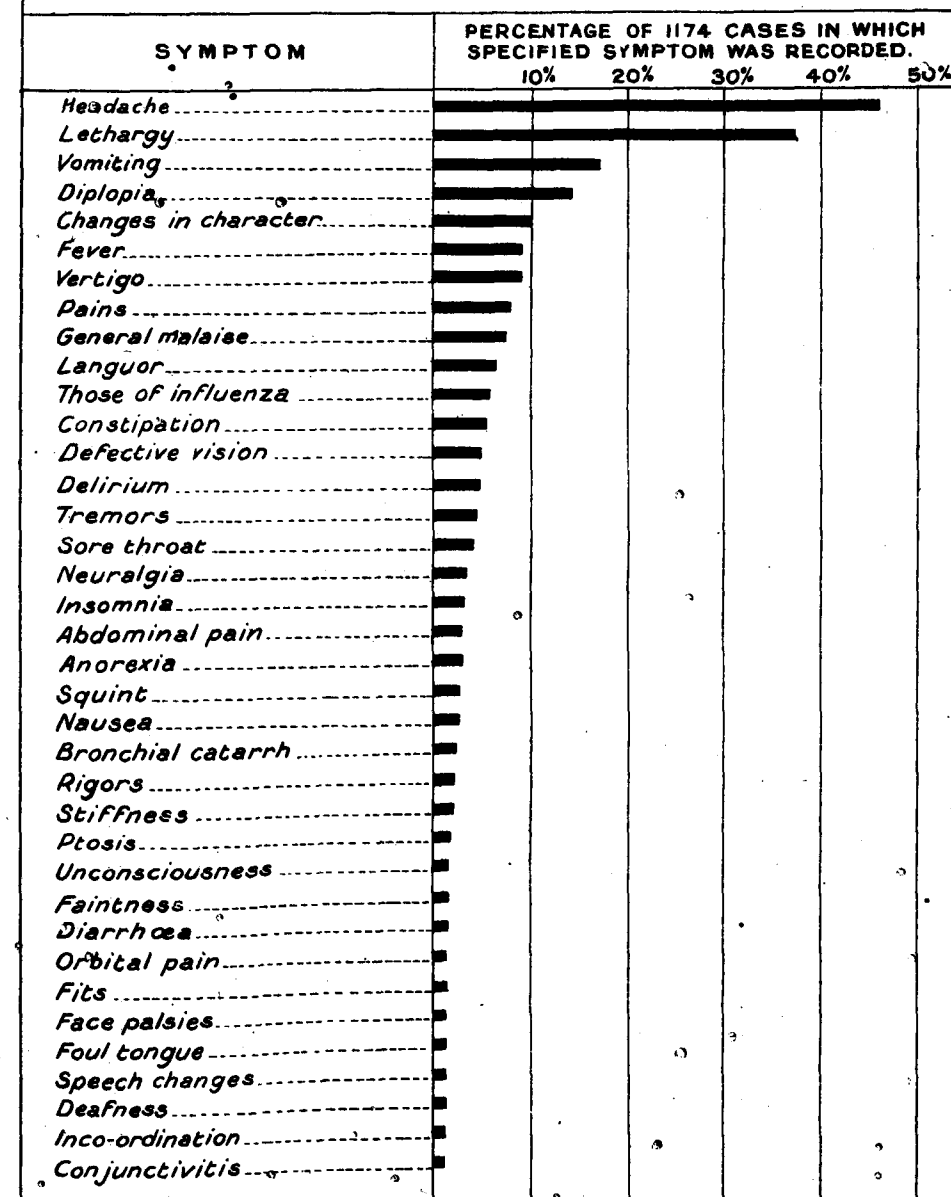
Symptom.	No. of times mentioned.	Symptom.	No. of times mentioned.
Gastric attacks - - -	11	Retention of urine - - -	4
Photophobia - - -	10	Palpitations - - -	4
Amnesia - - -	9	Dyspnoea - - -	4
Bilious attacks - - -	9	Yawning - - -	3
Earache - - -	7	Herpes - - -	3
Joint pains - - -	7	Rash - - -	2
Palsy or paresis of arms - - -	6	Swollen glands of neck - - -	2
Mania - - -	6	Dysphagia - - -	2
Epistaxis - - -	6	Incontinence of urine - - -	2
Sweats - - -	6	Aphonia - - -	1
Thirst - - -	5	Jaundice - - -	1
Nystagmus - - -	5	Hiccough - - -	1
Pleurodynia - - -	5	"Heliotrope rash" - - -	1
Myoclonus - - -	5		
Disorders of sensation - - -	5		

The infrequency of *gastro-enteritis* as a prodromal symptom suggests a comparison with epidemic poliomyelitis where gastric symptoms are comparatively common in the early stages of that affection. In one case of encephalitis Dr. Nankivell very strongly suspected that the alimentary tract formed the original focus of infection. The early evidence of *loss of power in the arms* in some cases may be compared with the comparative frequency with which the upper limbs also are subject to neuralgia. The importance of bearing in mind the fact that *mania* may, if only rarely, usher in an attack of encephalitis needs no emphasis and the figures above probably underestimate the number of cases in which profound mental disturbances at first confused the diagnosis. None of the other infrequent symptoms call for special comment here and some of them probably were only casual incidents.

The onset of encephalitis lethargica, then, may be so sudden and marked by symptoms of such a character that the true diagnosis at first is not entertained; in another and larger class of cases the beginning of the illness is so insidious and so indeterminate that no diagnosis of any kind is for some time attempted. It should perhaps be emphasised here that the division between "initial symptoms" and the later manifestations of the disease, which has been made in the enquiry schedules and in this report,

Chart IV.

COMPARATIVE FREQUENCY OF INITIAL SYMPTOMS.



is only artificial and by no means always in accord with the natural history of the disease. In a large number of cases the prodromal stage merges imperceptibly into that of the declared disease while in a fewer number the invasion is explosive in its intensity and without any premonitory manifestations. On the other hand, the reports show that not uncommonly there is a distinct interval of time—during which the patient may resume work—between the prodromal period and the fully developed stage, which in these cases may set in quite abruptly.

As regards the duration of the prodromal period the reports do not yield much precise information; the average time, deduced from a study of 36 cases with gradual onset in which the period is definitely stated in figures, works out as just under a month. This is a much longer period than that suggested by MacNalty who, using data from 146 cases with varying onset, concluded that the “prodromal period therefore most commonly ranges from the first to the seventh day, but may be as protracted as three weeks.” It seems probable, however, that increasing clinical experience has resulted in the tracing further back of the actual beginning in many individual cases of encephalitis lethargica and that the usual limits of variation should be extended from, say, a few hours to three weeks.

D.—SYMPTOMS AND SIGNS IN THE DECLARED DISEASE.

1. **General symptoms of Infection.**—(a) *Fever.*—Concerning fever the schedule reports are not always very full and in the case of such a symptom, requiring close and continued observation, it is not unnatural that this should be so, especially where patients were treated at home amid the stress of much other professional work. A certain amount of information, however, can be abstracted from the answers and is here set out.

In 1,264 case reports, fever was mentioned as present, at some stage or other of the disease, in 905 cases (71·5 per cent.); it was reported as absent on 182 occasions (14·3 per cent.); in 177 instances no answer was returned. The proportion of cases in which, according to the schedules, there was no fever is certainly too high, because from some “follow-up” inquiries it was found that pyrexia was noted during the patient’s stay in hospital, although previously to his admission none had been observed; it is also probable that some patients were not seen by their medical attendant until the fever had subsided. At the same time there were an appreciable number of patients, observed closely both at home and in hospital, whose illness undoubtedly was afebrile throughout. Well-authenticated instances of apyrexial cases also occur in the literature; thus in a clinical review of some seven cases, Walshe (1965, *b.*) mentioned that in three of them there was no fever.

From the schedule reports it would appear that where fever was absent other symptoms of a general infective nature were also missing, and the patients were early attacked by definite nervous manifestations which in turn were usually of a “negative”

nature (lethargy, paralysis, &c.); absence of temperature, too, was reported in cases of both short and long duration, and was not necessarily of good portent, since the mortality among such patients was 27 per cent. In a few instances (16) the temperature was subnormal during the greater part of the illness.

It is difficult from the reports to correlate the temperature accurately with the various periods and phases of the illness, but "in the early stages"—a phrase constantly occurring—roughly indicates the time of chief incidence. The appearance of fever usually marks the end of the prodromal period and the beginning of the more characteristic phases of the disease but, as stated in a previous section, fever may appear at an earlier stage and form part of the prodromal picture. In most cases a rise of temperature seems to be associated more with the general than with the nervous symptoms, and indeed in certain instances the fever declined as the lethargy deepened. There is, however, a good deal of apparent caprice about the behaviour of the temperature; thus cases are reported in which the fever was remittent (not common) or intermittent (common); of long and short duration; where resolution was by lysis (most common), or by crisis (rarely); and where, as death approached, it rose rapidly or gradually sank to or below normal. In many reports it is stated that there was a slight evening rise in temperature.

The duration of the fever may vary within wide limits, but as a general rule it is comparatively short. In many instances pyrexia lasted for a few days only, while in one case it was prolonged for 83 days; 14 days was the average time derived from an analysis of 137 cases in which the necessary details were available.

The maximum temperature was recorded in some 600 reports and the results are tabulated below:—

TABLE 30.

99°–100° F.	-	-	-	-	-	33 cases.
100°–101° F.	-	-	-	-	-	115 "
101°–102° F.	-	-	-	-	-	133 "
102°–103° F.	-	-	-	-	-	129 "
103°–104° F.	-	-	-	-	-	111 "
104°–105° F.	-	-	-	-	-	51 "
105°–106° F.	-	-	-	-	-	20 "
106°–107° F.	-	-	-	-	-	6 "
107°–108° F.	-	-	-	-	-	7 "
108°–109° F.	-	-	-	-	-	2 "
110° F.	-	-	-	-	-	1 "

The most usual range of temperature is thus from 101° to 102° F. as recorded in the first report, but within the limits 100° and 103·5° may be included the great majority of the cases now under reference.

Hyperpyrexia is a comparatively rare and nearly always fatal complication; in many cases—but not in all—it signified the concurrence of some other febrile complaint, such as malaria or

the late supervention of some inflammatory process, usually a broncho-pneumonia. Many of the temperatures ranging between 103·5° F. and 105° F. were seen in those cases where there was a previous history of influenza, or in which the onset resembled that of influenza.

In nearly all instances of very high fever the temperature rose sharply within the last 24 hours of life (in one case the temperature registered 105° F. after death),* and no patient survived a temperature of 106° F. or over. Usually, too, the cases in which hyperpyrexia occurred were of short duration, but in two patients, whose temperature reached 107° F. before the end, the illness lasted for five and six weeks respectively. The incidents in the case where the temperature reached the unusual figure of 110° F. are as follows:—The patient was a joiner, aged 44, living in Sunderland; he had influenza in 1918 and in June, 1920, he suffered from shock, the result of an accident. On 9th August, 1920, he complained of general malaise, felt cold and shivered, and was depressed by a sense of impending calamity; there were twitchings of left face and limbs. Lethargy was observed on the first day and the patient rapidly became stuporose, though not actually unconscious; there was double ptosis and incontinence of faeces and urine. The temperature ranged between 100 and 101 until the last day, when it rose to 110° F.; an erythema appeared on the arms just before death. The man died in Sunderland Royal Infirmary on 13th August, after an illness of four days. At the post-mortem examination, the liver, spleen and kidneys showed signs of recent inflammation, and the kidneys were also found to be in a condition of advanced chronic nephritis. "There was marked congestion of the meninges, but no meningitis. Inflammation and necrosis of the nerve cells and fatty degeneration of neuroglia. Diplococci in all respect like Rosenow's present."†

Another case in which a temperature of 108·6° F. was recorded is fully reported in Appendix I, Case A.

(b) *Circulatory System*.—As far as can be learned from the scattered references in the schedule reports there is nothing peculiar about the heart's action as observed in cases of encephalitis lethargica. The pulse rate as a rule appears to follow closely

* In the notes of a case (Notification No. 2939/22) observed in hospital during March 1922, it is recorded that the temperature was 106° F. just before death and 109° F. 15 minutes after this event.

† This case, however, is surpassed in the literature where Gelpke (374) describes the illness of a man of 29, who after partial recovery from an attack of influenza, was sent to hospital on account of extreme debility. Ten days after admission, his temperature rose to 111·6° F., and he exhibited symptoms of encephalitis, including rigor, epistaxis, delirium, diplopia, palpitations and rigidity of muscles in arms and legs. There is no mention of any bronchial complications and it is stated that there was no acceleration of pulse, no albuminuria and no dryness of the tongue. More interesting still, is the statement that the thermometer registered 111·6° F. on several occasions during a thirty days' period of hyperpyrexia and delirium,—and that the patient eventually recovered.

the variation of the temperature and so for the greater part of an average illness it varies little from the patient's normal; in one or two of the 10 instances where *tachycardia* was specially mentioned, however, there is a possibility that the acceleration was due to paralysis of the vagi. Five of the patients who suffered from tachycardia died. *Bradycardia* was reported in six patients, of whom five died. Most of the allusions to the state of the heart itself are concerned with chronic conditions, but in two consecutive cases at Bristol cardiac dilatation was considered as possibly due to the virus of encephalitis. The blood was examined in 22 cases and as a rule was found to be normal; in a few instances leucocytosis was noted, and in one patient a short streptococcus was found in the blood.

Widal's test was applied in 52 instances, and in two a partial reaction was obtained which was thought to be due to anti-typhoid inoculations made while the patients were in the army. There is evidence that the test proved useful in some of the asthenic cases which simulated typhoid fever. In three out of 48 patients tested a positive *Wassermann reaction* (blood) was obtained, but this did not influence the diagnosis of encephalitis lethargica in these cases; in others the test was the means of revising the diagnosis.

(c) *Respiratory System*.—Apart from those few cases in which nasal catarrh or other affections of the upper respiratory tract were recorded as prodromata, there is little mention of respiratory symptoms occurring during the course of the disease; in a few cases, however, broncho-pneumonia occurred as a late and often fatal complication. The respirations were comparatively rapid where the pulse rate and temperature were high and in three instances early dyspnoea was noted; Cheyne-Stokes breathing is mentioned seven times and in two patients it was not a fatal omen.

(d) *Digestive System*.—In the present series of reports the condition of the tongue is only referred to definitely in some 16 cases, but possibly this reticence is due to a feeling that the subject is too trivial for mention. In many cases, certainly, it is heavily furred and dry and sometimes associated with foul breath. A marked feature of the disease is *constipation*, but here, again, and perhaps for the same reason, this symptom is not mentioned nearly so often as it was in many previous clinical reviews; possibly there is, indeed, a diminished tendency to constipation, which would account also for a healthier state of the tongue. The total number of cases of the present series in which constipation was reported is 262 (12.7 per cent.), while in the first series of cases it was considered as "being present in every case almost without exception." There is still, however, plenty of evidence that the condition when present is most refractory, though we have no parallel to a case described by Bassoe (59) where the administration of nine drops of croton oil together with enemata proved unsuccessful. *Vomiting* has been

discussed previously as one of the most frequent of the prodromata, and it is chiefly on the early stages, as one of the signs of a general toxæmia, that this symptom—often associated with *nausea*—is found. Cases are recorded, however, where vomiting, probably of cerebral origin, occurred after the disease was well established. There is no clinical evidence that the liver is specially implicated, though in one or two instances inflammatory changes were discovered at the autopsy.

Salivary Glands.—In a very few cases only was excessive salivation noted, though dribbling as a result of certain facial palsies is not uncommon. Both parotid glands were enlarged in a patient of 47, who died after a short illness of eight days and whose brain showed changes typical of encephalitis lethargica. This very rare involvement of the salivary glands in our series is interesting in view of the opinion of Netter and others in France that these glands are often intimately affected in the disease.

(e) *The Urine*.—In 33 cases only was any report on the urine furnished and in half of this number it was said to be normal; in the majority of the remaining cases albumen was present in small quantities. Two patients developed glycosuria—without albumen—and one of these was the subject of hiccough. Blood was present in two cases and was caused by the action of urotropin. No cases of polyuria were mentioned.

(f) *The Skin*.—It would appear from the reports that encephalitis lethargica is not associated with any characteristic rash and with this statement the findings of MacNalty and the experience of foreign observers are in keeping.

In 92 cases of the present series some form of eruption was present though the eruption was not, in every case, precisely described; something of the appearance and nature of these rashes, however, may be gathered from the list below:

TABLE 31.

Rashes resembling:—	Inflammations, etc.	Indefinite.
Scarlet fever - - 5	Herpes labialis - 11	Rashes - - - 2
Measles - - - 4	„ frontalis - 1	Spots - - - 3
Typhoid fever - 2	„ costalis - 1	Red spots - - 1
—	Bullæ - - - 2	Flea-bite type - 1
Desquamating	Vesicular rashes - 4	Duskiness - - 1
rashes - - 4	Sudamina - - 6	Enema rash - - 1
	Papular rashes - 7	Petechiæ - - -
	Papulo-macular	Red blotches -
	rashes - - 2	Leucoderma with
	Papular erythema - 3	erythema - - 1
	Erythema - - 16	
	Urticaria - - 4	
	Furunculosis - 1	
	Acne - - - 1	
	Hæmorrhagic rash 1	
	Purpuric rash - 1	

A certain number of the conditions named above were probably nothing more than casual co-incidences; it is possible, too, that, in a disease where constipation is such a common and troublesome feature, a larger number of rashes may be attributed to the giving of enemata than the figures indicate. As in the previous series of cases, erythema is the eruption most frequently observed. From the point of view of diagnosis it may be noticed that the eruption in certain cases simulated some of those of the common exanthemata and in the case of scarlatiniform rashes particularly there has been some hesitation over the diagnosis in one or two instances.

Allusion may be made here to a case (in 1921) in which an infant contracted encephalitis lethargica from its mother and exhibited also a scarlatiniform eruption which was similar in character and distribution with that shown by the mother.

The relatively small number of cases in which herpes labialis occurred may be compared with the much more frequent appearance of this condition in cerebro-spinal fever; further, the figures for herpes generally may be kept in mind with reference to the experimental work recently carried out in connection with the peculiar potentialities of herpetic fluid, and which will come up for consideration in Section IV.

In two cases there was noticed the "heliotrope cyanosis" described and figured in the *Report on the Pandemic of Influenza 1918-19*, Ministry of Health, No. 4. In both cases the patients were males aged 38 and 27 respectively and both died after the temperature had reached 105° F. The elder man was ill for nine days in March 1919 with lethargy and "negative" nervous signs, including eye palsies; the other patient lived for 17 days, August 7th-24th, 1920, and in his case there were signs of irritation, both general and local, eventually superseded by deepening stupor and coma at the last: in neither of these cases could a diagnosis of influenza be excluded.

Speaking generally the rashes observed were mild and evanescent; rather more than half the subjects of rashes died, but in no case does the fatal event appear to have had any connection with the eruption.

No particular group of symptoms was common to the cases in which a rash appeared; generally there was fever, but in a few instances the malady was afebrile; sore-throat was associated with one of the cases of scarlatiniform rashes and in one of the patients labial neuralgia preceded the appearance of herpes on the lips. The eruptions appeared both early and late in the illness and were indeterminate in their distribution; thus in the case of the erythemata the whole body, the neck and chest, the arms and other parts were variously affected and in one instance the rash was limited to a single finger.

Although skin eruptions are rarely seen in cases of encephalitis lethargica, and no one form of such is in any way characteristic, it is important to know the variety of rashes

that may occur in this disease and which sometimes resemble closely those of the acute infectious exanthemata.

2. General Nervous Symptoms and Mental State.—(a) "Negative" symptoms: *Lethargy, Stupor, Coma.*—The leading characteristic of the disease is implicit in its name and, as pointed out previously, lethargy in greater or less degree was very frequently present as an initial symptom. In a large proportion of cases the preliminary lethargy deepens gradually into the stupor or clouded consciousness of the declared disease and at the same time the general intelligence becomes more and more blunted. Patients who may have shown a lack of interest in their surroundings now become apathetic or bored and from being merely inattentive perhaps they are now described as "stupid," "obtuse," "obfuscated"; there is no power of initiative and no desire to persevere along any line of thought or action suggested by others. The patient lies motionless on his back in the peaceful posture of a trance or deep sleep; there is as yet, however, no absolute unconsciousness nor is the patient necessarily asleep. Through the auditory sense in particular he can be aroused—and in many cases "easily aroused"—and will then answer questions and accede to simple requests in a slow and deliberate manner, to relapse once more into depths of seeming slumber. Or, without any external provocation (and as if, so to speak, he himself had switched on the light and dispelled his oblivion), the patient may quietly and abruptly return to full consciousness and enter into intelligent conversation with those about him. There is considerable variation in the depth of stupor to which the patient may sink; thus in an account of his experiences a professional man writes, "while in the stupor I felt nothing; no pain nor any sensation. It was absolutely dreamless, heavy sleep." On the other hand, there are some grounds for thinking that the muscular inertia of a patient in stupor is not always a reflection of intellectual inactivity, and there are records of patients who afterwards stated that while in a state of stupor their "minds were working" and that they were sometimes conscious of their environment though unable to assert themselves.

The occurrence of definite lucid intervals during the periods of stupor and even coma is by no means uncommon, but even though the return to consciousness after a spell of semi-coma may be complete the prognosis is not necessarily improved and the patient may sink back once more and not recover.

As the patient's sensory isolation becomes more and more complete the stupor merges into coma and this transition is often marked by loss of control over the sphincters and sometimes also by dysphagia. Coma is the most common final phase in cases that prove fatal, but it would seem from the records that only during the last day or so of life are the majority of patients absolutely unconscious.

In other circumstances the patient emerges from his stupor or coma and may then show signs of depression with possibly loss of memory; in most cases there is marked weakness.

Some degree of lethargy ranging from slight drowsiness to complete coma was noted in 1,084, or nearly 90 per cent., of the reports in which clinical details were given; in 526 cases the lethargy developed into stupor and in 292 instances the condition progressed still further to the stage of coma: of the patients who became comatose 86 per cent. died. In a certain number of instances the length of the period during which the patient was lethargic, stuporose or comatose is given, thus:—

Lethargy: in 55 cases the average duration was 20 days.

Stupor: in 21 cases the average duration was 19 days.

Coma: in 26 cases the average duration was 8 days.

The cases, however, are too few to make the average figures of much value and there are evidently wide limits to the length of time that a patient may remain lethargic. In mild or abortive cases the drowsiness, hardly amounting to lethargy, may be a fleeting symptom of two or three days duration, in other instances lethargy may persist for weeks throughout a long illness and be still present when the patient has otherwise recovered.

In 14 of our series of cases lethargy was absent and death occurred in five of these instances; cases have also been reported in the French literature where no lethargy was present during the course of a fatal attack.*

Stupor is more limited in duration and though patients may—usually with one or more intermissions—remain in this condition for six weeks or more, the usual time is much shorter. Complete coma, whether appearing early in the illness or as a terminal condition, is the shortest phase of all.

It may be emphasised here, however, that coma is not necessarily a fatal development in this disease, and in certain instances patients have recovered after being unconscious for two or three weeks.

The general intelligence, as stated above, deteriorates in many cases with the tightening grip of lethargy, but in a large number of patients there is no such mental deterioration. Thus in some 74 cases it was reported that the patient was mentally clear and other instances of the same thing are afforded by such expressions as “mentally normal,” “intelligence unaffected,” “mind as usual,” “quite sensible” and others.

In the case of two patients it is specially mentioned that the mind which had previously been clouded cleared just before death, and Hanley (424) speaks of a patient who made all arrangements for her own funeral a day before she died.

* In a communication dated 29th October, 1921, Dr. Charles Porter, Medical Officer of Health, Johannesburg, stated that there had been a recrudescence of encephalitis lethargica in the Johannesburg district during September and October, 1921. The disease on this occasion seems to have shown atypical characters and in particular there was an absence of lethargy among the symptoms. In the case of 20 patients, specially examined by a neurologist, the clinical syndrome included headache, diplopia, meningitis, paresis of limbs, and mental obfuscation; 40 per cent. of the graver cases proved fatal.

(b) “Positive” Conditions: Irritability, Restlessness, Insomnia, Delirium, Emotional Outbursts, Hallucinations, Mania.—Excluding a certain number of mild cases which are known to us but which were unnotified, there are 13 instances recorded in the schedule reports where there was no drowsiness or lethargy shown by the patient at any time during his illness. In one such case the clinical picture was completely “positive” with such features as headache, insomnia and delirium, together with myoclonic contractions of arms and legs and a complete absence of muscular palsies. More usually in these non-lethargic cases, both “positive” and “negative” localising signs were present; thus, a labourer, was attacked with abdominal pain and vomiting; he became feverish and delirious and in addition to tremors developed double ptosis and squint; there was never any lethargy throughout the 17 days’ illness and at the post-mortem examination lesions typical of encephalitis lethargica were found.

In a far larger proportion of cases the clinical picture is a composite one made up of both “positive” and “negative” features. Very commonly it is reported that the patient gets little or no sleep by night but is overcome by lethargy or somnolence during the day; in such cases, too, the sleeplessness is often associated with restlessness and delirium.

Insomnia may be a very distressing feature of encephalitis lethargica and it was mentioned as a symptom in some 70 cases of our series; most commonly it begins early in the illness either as a condition precedent to lethargy or, in combination with delirium, occurring at night in patients who are lethargic by day. It may also delay the convalescence following a period of stupor and more frequently it remains for some time as a serious handicap to patients who have returned to work.*

Delirium was associated with lethargy in 329 cases; it occurs chiefly in the early part of the illness and may cease after a few nights in those cases where the general nervous symptoms have a paralytic or “negative” bias. Where the general clinical picture is more “positive” in its features delirium may persist for a longer time or it may reappear at intervals during the illness as part of a general exhibition of irritability; in five of the cases reported during 1920 delirium persisted throughout the entire course of the illness. In a few cases the delirium was caused by, or associated with, a high temperature; but, on the other hand, in 50 per cent. of the hyperpyrexial cases there was no delirium. About 60 per cent. of the patients who became delirious at some time during their illness exhibited also positive focal signs such as involuntary movements, rigidity and the like. In its degree the mental excitement may vary from a

* In discussing “ardent fever and phrenitis which occurred early in spring after the cold had set in,” Hippocrates mentions that his patients “were constantly affected with coma in which they got no sleep, or with insomnolency, attended with pains.” Epidemics, Bk., III., Sec. 3, § 6, Adams’ translation.

mere and occasional muttering to maniacal outbursts, but usually it consists of physical restlessness and occupational soliloquies.* Occasionally the thoughts and words would be accompanied by mimetic actions; thus a young schoolmistress who by day was deeply stuporose would as a nightly routine go through the movements of washing her face and hands and "doing up her hair" with the aid of imaginary hairpins abstracted from her mouth: this case also illustrates the very characteristic way in which the delirium of encephalitis lethargica alternates with the contrary condition of lethargy. Gradually these early manifestations of irritation may become overshadowed by a deepening lethargy so that the general nervous symptoms become mainly negative in character; or, like the oft-recurring "motif" in a musical composition, one may look for a reappearance and reassertion of "positive" signs such as predominated in the early stages. Thus the emotional patient may give way to screaming or indulge in hilarious laughter and with this mental instability there may be much physical restlessness requiring special nursing precautions; in some cases, indeed, the patient may be maniacal. The sense-perceptions may be awry as in the case of a patient who tried to clean an imaginary set of teeth in his neck and who also while having his legs bandaged chided the nurse for her silliness in so treating, as he thought, the legs of a piano. In other cases again the attitude of the patient is suggestive of that displayed by a sufferer from meningitis, and the desire to be left alone is shown by marked irritability and resentment when attempts are made to elicit some answer or action.

These various signs of irritation appear and reappear as the patient emerges for a time from his stupor, but some of them may also be manifested while he is apparently in a semi-conscious state. In grave cases the patient, worn out it may be with the irritative action of the virus, finally sinks into unconsciousness and dies from coma. Or this state of combined exaltation and depression of function may pass away and leave the patient in a state of mental confusion amounting in some cases to disorientation and with a faulty memory especially for recent events; it is characteristic, too, of these "positive" symptoms that some of them may remain for some time as troublesome sequelæ.

3. Localising nervous symptoms: (a) "*Negative*" *Symptoms: Paralyzes*.—Just as the classical type of encephalitis lethargica is characterised by a general paralysis of the emotions, so, too, certain muscular palsies of focal origin have come to be considered almost pathognomonic of the disease and in a very large pro-

* The cerebral excitement seen in certain cases of encephalitis lethargica may be compared with that resulting from such organic poisons, as alcohol and belladonna; the foreign literature (434) affords an instance where the mental confusion and other symptoms of encephalitis lethargica also suggested acute veronal poisoning.

portion of cases the lethargy of the mind finds a counterpart in muscular inaction; there is a parallelism also between the many grades of lethargy varying from slight sleepiness to deep coma and the different degrees of muscular weakness ranging from a very slight and partial loss of power to complete paralysis. Moreover, the muscles which are particularly picked out are those concerned with the eye and as pointed out by MacNalty there are anatomical grounds—consolidated by histopathological study—upon which the close and common association of lethargy with ophthalmoplegia can be often explained. A very characteristic attribute of the ocular and other palsies as seen in encephalitis lethargica, however, is their transient nature which suggests that sometimes the lesion does not affect the nuclear cells of the implicated nerves, but rather their fibres, which Boyd (211b) thinks may be temporarily compressed by inflammatory oedema or dilated blood vessels.

In addition to the ephemeral nature of the palsies generally, especially of those concerned with the eye, there is, as a rule, much irregularity in distribution as well as asymmetry; there may be paralysis in one arm and a paresis of the leg on the opposite side; both sides of the face are far less frequently affected than one side only or if both sides are affected they are so to an unequal degree.

The paralyzes are usually progressive and differ in this way from those occurring in acute poliomyelitis, where muscles may quickly become completely powerless; they are rarely permanently complete and, as the schedule reports abundantly show, the loss of power is often a paresis rather than a paralysis; the general rule is for the paresis or paralysis to clear up, quickly, leaving no atrophy unless from disuse. There may be a certain amount of flaccidity at first, but in protracted cases the paralysis is generally associated with rigidity.

To repeat: the palsies of encephalitis lethargica, and especially those connected with the cranial nerves, are progressive and transient in character, and the way in which the recovery of one cranial nerve is so often followed by the implication of another is highly characteristic of this disease.

The relative frequency with which the various muscle groups may be affected in encephalitis lethargica corresponds roughly with the "levels" of emergence of the nerves supplying them. Thus ophthalmoplegias are the most frequent and distinctive paralyzes of the disease,—next in order come those of the facial muscles, then those of the arms and finally those of the lower limbs. The various paralyzes will now be considered more particularly.

Cranial Nerves: 1st Nerve.—No abnormalities in the sense of smell are mentioned in the reports and we can find no references in the literature to any cases where the first nerve was involved. We should observe, however, that this special sense was as a rule not scientifically tested.

IInd Nerve.—Optic neuritis, though pre-eminently associated with irritative lesions, is discussed here for purposes of completion and convenience. The *fundi* were examined in 234 cases and in 80 per cent. of these it was reported that no changes were seen; in the other 20 per cent. the following conditions were reported:—

TABLE 31.

Condition of fundus oculi as reported.	Number of cases.
Optic neuritis - - - - -	11
R. early papillitis inner border of disc - - - - -	1
L. marked papillitis and a few hæmorrhages at inner edge - - - - -	
Choked disc - - - - -	2
Discs swollen, margins blurred, vessels buried - - - - -	1
Vessels contracted - - - - -	1
Edema - - - - -	4
Slight swelling - - - - -	1
Disc pale, edema - - - - -	1
Discs pale, white - - - - -	2
Discs blurred - - - - -	2
Inner edge blurred - - - - -	1
Congested disc - - - - -	7
Injected disc - - - - -	4
Slight hyperæmia - - - - -	1
Dilated vessels - - - - -	5
Dilated veins - - - - -	1
Disc cupped, vein large - - - - -	1
Patches of degeneration - - - - -	1

It will be seen that optic neuritis including papillitis is mentioned as present in 12 cases; in two or three others the condition would seem to have been well marked, while in the majority of the remaining cases the several signs which were observed form part of the ophthalmoscopic picture of optic neuritis. This means that in 5.5 per cent. of the cases in which the fundi were examined optic neuritis was present, while in 19 per cent. of the cases there were present one or more signs portending the same.

These figures are undoubtedly high and imply a much greater frequency of retinal changes than is usually supposed to be the case, so that the schedules responsible for them have been repeatedly overhauled with a view to reconciling a diagnosis of encephalitis lethargica with the occurrence of a condition hitherto thought to be very rare. It is unfortunate that in most of the fatal cases no post-mortem examination was made so that a certain amount of doubt may be legitimately expressed as regards some of the cases; on the other hand, to dispute the diagnosis in most of the instances would be to disregard cases about which clinically there seemed, in other respects, no reasonable doubt. Special questionnaires were sent to some of those responsible for the reports in which optic neuritis is mentioned, but in the replies,

almost without exception, the original data and diagnosis are upheld; moreover, in many of the cases the reports received the additional imprimatur of consultants or hospital authorities.

The subject is an important one, for it so happens that the greatest diagnostic difficulties of encephalitis lethargica lie with intra-cranial tumours and tuberculous meningitis,—two conditions in which one of the classical signs is optic neuritis. MacNalty (629a) in discussing the diagnosis of encephalitis lethargica, considers that “a negative sign of value is the absence of optic neuritis” and suggests that this involvement of the II. nerve is in the nature of an accident and due to hæmorrhagic infarction into the brain substance which raises the intra-cranial tension. The question is one that we think might well receive further and specialised attention from those able to follow closely the clinical course of the disease; in the meantime a table of such references to optic neuritis as we have been able to find in the literature (British and American only) is set out below:—

TABLE 32.

Some references in the literature to the state of the fundi oculi.

Authors.	Number in bibliography.	Number of cases reported.	Number of cases showing fundus changes.	Percentage (approx.)	Conditions observed.
Buzzard, E. F. -	158	20	2	10.0	(a) Slight swelling and blurring of upper and inner edge. (b) Discs, swollen, veins engorged.
Findlay and Shiskin, C.	336	24	1	4.0	Swelling of disc with tortuosity of veins and obscuration of vessels in places.
Findlay, L. -	335	4	1	25.0	Slight redness and swelling of both discs.
McClure, W. St. C.	617	17	6	35.0	Congestion of retinal veins, 4 cases. Commencing optic neuritis, 2 cases.
Symonds, C. P. -	1007a	4	4	—	Optic neuritis in four patients admitted within 2 months to hospital in Boston. U.S.A. The author has informed us that in one case the diagnosis of encephalitis has been since abandoned.
Watson, G. A. -	1069	1	1	—	Optic neuritis of medium intensity; lesions involved chiefly the cerebral cortex.
Dr. A. B. Soltau (communicated).	—	24	2	8.0	Optic neuritis.
O'Carroll, J. and Nesbitt, G.	751	4	1	25.0	White patch near right disc.
Boyd, W. -	121b	75	2	2.6	Optic atrophy found as a sequel.
Grinker, J. -	394	1	1	—	Optic neuritis began 3rd day of disease, well marked within a week and intense for some days; scars formed.

Authors.	Number in bibliography.	Number of cases reported.	Number of cases showing fundus changes.	Percentage (approx.)	Conditions observed.
Happ, W. M. and Mason, V. F.	427	81	2	2.4	(a) Optic neuritis L disc; R normal. (b) Optic neuritis more marked on R.
Alexander and Allen, H. E.	206	100	7	7.0	(a) Choked disc, 4 cases. (b) Retinal veins engorged, 3 cases.
Spaeth, E. B.	981	5	1	20.0	"True optic neuritis."
Hoffman, R. V.	458	4	3	75.0	(a) Some oedema. (b) R. disc oedematous. (c) Moderate oedema.
Brasher, C. W. and Caldwell, J. R.	129	2	1	50.0	Pale disc, vessels engorged.
Ezekiel, G. A.	328	2	1	50.0	Slight pallor of discs.
Rossiter, C. B.	901	10	1	10.0	Both discs pale in temporal segment.
Holden, W. A.	463	100	5	5.0	Blurred discs, 4 cases. True papilloedema, 1 case.
Reverchon and Worms.	861	25	1	4.0	"Choked disc."
Chail, D.	681	—	3	—	(a) "Choked disc," 1 case. (b) Papillary congestion, 2 cases.
Woods, Hiram	1,097	7	1	14.0	Edge of disc blurred, veins enlarged.
Waardenburg	1,233A	—	2	—	(a) Scotoma (retrobulbar neuritis). (b) Peripheral narrowing of visual field.

IIIrd, IVth and VIth Nerves.—These three nerves are conveniently discussed together because they are often involved together in producing one of the most striking and most frequent symptoms of the disease, viz., *ophthalmoplegia*.

Exercising its strange selective powers in this case upon the oculo-motor nuclei in the floor of the Sylvian aqueduct, the virus of encephalitis lethargica may produce an acute nuclear ophthalmoplegia, both internal and external: the manifestations of such ophthalmoplegia are chiefly connected with the IIIrd nerve whose muscular responsibilities outweigh considerably those of the IVth and VIth* combined.

* Boyd (121b) considers that VIth nerve paralysees—often fleeting in character—are due to "involvement of the nerve fibres in their long course through the pons," and states that in several of his cases it was possible "to demonstrate a greatly dilated and inflamed vessel lying between the fasciculi of the nerve."

An analysis of 1,152 individual reports shows that in 78 per cent. of the patients the external ocular muscles were affected in various ways specified below:—

	Cases.
Ptosis alone	245
Squint alone	118
Ptosis and squint combined	109
Nystagmus alone	56
Diplopia alone	50
Ptosis and diplopia combined	46
Ptosis and nystagmus combined	46
Ptosis, squint and nystagmus combined	43
Ptosis, squint and diplopia combined	34
Squint and diplopia combined	34
Squint and nystagmus combined	22
Ptosis, diplopia and nystagmus combined	18
Ptosis, squint, diplopia and nystagmus combined	17
Diplopia and nystagmus combined	11
Squint, diplopia and nystagmus	6

Ptosis is the most common form of IIIrd nerve paralysis and was reported, alone or in combination with other palsies, in nearly half the cases; its most usual accompaniment was squint. In 38 per cent. of the cases the ptosis was double and in 15 per cent. unilateral; information is lacking concerning the remainder, but it is the exception for this particular paralysis to be limited to one side and very commonly one upper lid begins to droop as the first affected is recovering. All degrees of weakness may be observed, but where both lids are simultaneously and equally affected the condition is probably due to general lethargy and lack of tone rather than to paresis of the IIIrd nerve; photophobia, too, may cause the lids to be kept shut. In one case, reported by Hanley (424), the patient could not open his eyes without help, although closure was easily effected; the behaviour of a little girl suggested that of some dolls, in that her ptosis disappeared when she sat up. Ptosis may occur quite early in the disease and when transient it is sometimes ignored; a patient will volunteer a history of diplopia but may require prompting as to a slight previous ptosis which was thought to be caused by a cold.

Squint was present in about 34 per cent. of the total cases; the strabismus was internal in 61 patients and probably external in the majority of the others; in three patients it was supposed that the IVth nerve was specially implicated.

In a few cases squint was noticed among the initial symptoms but it is observed more usually when the disease is well established; the frequency with which patients early complain of blurred vision and diplopia, however, show that there is often some weakness and inco-ordination in the eyeball muscles before, if ever, a definite squint is obvious.

Diplopia is a characteristic and important early symptom in encephalitis lethargica and occurred in about 18 per cent. of the cases; it was associated with squint in 91 patients out of 222.

Blurred Vision.—Among the early symptoms of the disease there are 61 references to "blurring of eyesight," "dimness of vision" and similar visual disabilities, most of which probably are due to errors in accommodation. Inability to read the newspaper is quoted in a few instances as the first sign of the disease, and it may be mentioned here that these defects not infrequently remain during the illness and necessitate glasses when the patient returns to work.

Pupils.—In their appearance and in their reactions the pupils vary considerably not only in different cases but at different times in the same patient, thus:—"at first equal and reactive, then fixed and unequal"; "both pupils dilated, then both contracted but R. most, then both equally so"; "reactions varied daily." Comments were made in 187 cases and are tabulated below; in the Derby outbreak very marked dilatation was noticed in most of the patients.

TABLE 33.
Condition of Pupils.

Equal	Un-equal.	Ectopia pupillæ.	Dilated.		Contracted.		Re-actions normal.	Re-actions sluggish	All reactions lost.		Argyll* Robertson		Light + Accom.
			Both	One	Both	One			Both	One	Both	One	
33	41	1	47	15	35	4	25	23	12	2	18	1	13

* In 8 cases the condition was described as doubtful.

Nystagmus.—The involuntary movements of the eyeballs may be considered here though it does not seem certain that these "positive" manifestations are always directly associated with the oculo-motor nerves now under discussion. Nystagmus appears later in the disease than most of the other ocular derangements and in many cases would seem to be one of the signs of a general irritative phase; on the other hand it might possibly represent the tremulousness that follows when muscles lately paralysed again begin to function. The symptom is mentioned 215 times so that it occurred in about 18 per cent. of the cases; in 25 instances it is specified as lateral, in 4 vertical, and in 12 mixed. Very rarely are the oscillations described in detail, but such evidence as there is shows that the movements are generally coarse and irregular; in three instances the nystagmus was described as "morse code." It was noted in a previous

table of ocular symptoms that nystagmus, like diplopia, occurs alone much less frequently than do ptosis or squint; the association with squint, however, in 88 cases suggests again that the condition described as nystagmus may, in many cases, be due to the unbridled action of some of the ocular muscles and that, as MacNalty considers, it is a question of "ocular inco-ordination rather than a true nystagmus."

Vth Nerve.—Disorders of sensation are comparatively rare in encephalitis lethargica, so that the trigeminal nerve is not often implicated. In three cases it was mentioned that the cornea was anæsthetic and in another three patients the second division of the nerve may have been responsible for a loss of taste. (In a few instances there was facial neuralgia and some six or so patients suffered from spasm of the masseter muscles.)

VIIth Nerve.—Next to the oculo-motor nerve there is none which is more frequently affected than the facial and in nearly 40 per cent. of the cases some degree of facial paralysis was noted. The weakness, as in the case of the ocular muscles, may be very slight and hardly appreciable; a slight flattening of the nasolabial fold or a general smoothing of the features may be all that is seen. There is often a progressive tendency, however, so that a paresis becomes a true paralysis, but it is rare for both sides of the face to be equally involved. According to the records it is the lower part of the face that is usually affected; in the great majority of cases facial palsies are associated with some degree of ophthalmoplegia and other paralyses. The table below summarises the information derived from the reports.

TABLE 34.
Paresis and Paralysis of Face Muscles.

Paresis.						Paralysis.					
One side.	With eye palsies.	With other palsies.	Both sides.	With eye palsies.	With other palsies.	One side.	With eye palsies.	With other palsies.	Both sides.	With eye palsies.	With other palsies.
49	34	16	134	107	38	161	133	63	49	41	22

VIIIth Nerve.—Deafness was recorded in eight cases and in one of these there was paralysis also of the VIIth and XIIth nerves. Tinnitus is mentioned seven times and was generally associated with severe headache and sometimes with vertigo; it usually occurs early, but is not apparently such a prominent feature of the prodromal period as some writers aver.

Vertigo has been discussed previously; here it may be doubted whether the vestibular fibres of the VIIIth nerve are often

responsible for the common occurrence of this symptom in encephalitis lethargica.

IXth Nerve.—*Dysphagia* occurred in 60 cases, but it is so often reported as a late symptom, coming on while the patient was comatose, that it may be doubted whether the glosso-pharyngeal is often primarily involved; the fact that 75 per cent. of the patients who suffered from dysphagia died suggests that the difficulty in swallowing in such cases may be due to more general causes. Since, however, in some cases there is on the part of conscious patients a difficulty in swallowing solids, while fluids are taken fairly readily, it would seem as if the ninth nerve is sometimes at fault either through its sensory or motor fibres, or through both.

In the majority of cases where dysphagia occurred other paralyses were present as follows :—

TABLE 35.

	Cases.
With III. nerve (ocular) palsies - - -	19
„ III. and VII. nerve palsies - - -	24
„ VII. and X. nerve palsies - - -	2
„ III., VII. and XII. nerve palsies - - -	1
„ XII. nerve palsies - - -	1
„ III. and non-cranial palsies - - -	2
„ VII. and non-cranial palsies - - -	1
„ III., VII. non-cranial palsies - - -	2
„ III., VII., X., XII. and non-cranial palsies - - -	1
Alone - - -	7

60

Xth Nerve.—*Paralysis of the soft palate* is mentioned 12 times; on 10 occasions there was an associated ophthalmoplegia, the facial nerve was also affected in five cases and the IXth and XIIth nerves once each.

Aphonia is reported 18 times, but it is not certain that the condition in these cases was due to paralysis of the recurrent laryngeal nerves.

There are, also, no definite indications as to how often the visceral branches of the vagus nerve were involved; but *tachycardia* occurring near the end of the illness is mentioned some 10 times and in three or four cases the slowness and irregularity of the respiration may also have been due to involvement of both vagi.

In the case of a patient with symptoms of bulbar paralysis, in whom the IIIth, VIIth, IXth, Xth and XIIth nerves were involved and who also developed hemiplegia, vomiting may have had a pneumogastric origin.

XIth Nerve.—There are no direct references given to the spinal accessory nerve, but paralysis of the “neck muscles” is mentioned in three cases and among these the *sternomastoid* and *trapezius* were possibly involved.

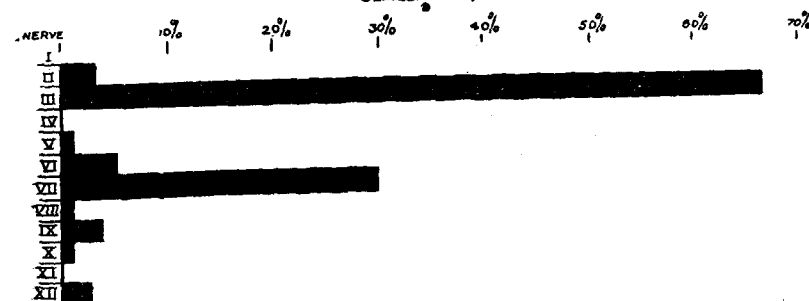
XIIth Nerve.—The hypoglossal nerve was affected in 30 cases; in 21 patients the *paralysis of the tongue* was unilateral, and in 9 patients both sides were involved; the associated palsies are shown below :—

TABLE 36.

	Cases.
With III. nerve paralysis - - -	4
„ III. and VII. nerve paralysis - - -	7
„ III., VII. and VIII. paralysis - - -	3
„ III., VII. and IX. paralysis - - -	1
„ III., VII., IX., X. and hemiplegia - - -	1
„ III., VII., X. and hemiplegia - - -	1
„ III., VII. and paresis of leg and arms - - -	2
„ III. and IX. - - -	1
„ III. and paralysis of legs - - -	1
„ VII. - - -	2
„ VII. and VIII. - - -	1
„ VII. and palsy of L. arm - - -	1
„ IX. - - -	1

The comparative frequency with which the cranial nerves were affected by both paralyzing and irritative lesions is shown below in graphic form; the results, pictured as percentages of a total series of 1,273 cases, are approximate only and generally represent possible maxima.

CHART V.



Other Motor Paralyses.—The motor palsies affecting the trunk and limbs resemble in many respects the cranial nerve palsies just described, and in the great majority of cases they are of the upper motor neuron type; the general tendency is for the paralyses to clear up and residual palsies are very rare compared with the case of acute poliomyelitis. Complete recovery of muscular power, however, may be long delayed and signs of irritation, such as tremors, may persist for some time in muscles that have been affected during the illness.

In the majority of cases motor paralyses of the limbs and trunk were associated with cranial nerve palsies. “Great

asthenia” and “general paresis” are terms frequently used in the reports and in some cases probably denote a general lack of muscular tone and power which are not dependent altogether upon the toxæmia; paresis of the legs is comparatively frequent. In four cases, all of which proved fatal, there was said to be a general paralysis: in a little girl of 11 there was at first a paralysis from the waist downwards, then the left hand became palsied and subsequently the “paralysis was complete.” *Hemiplegia* occurred in nine patients and the importance of recognising this condition as a possible feature of encephalitis lethargica has been pointed out in the literature by Buzzard (158) among other writers.

Paraplegias and *monoplegias* occur more frequently as will be seen in the table below:—

TABLE 37.
Motor Paralyzes.

Type of paralysis or paresis.	Unilateral	Bilateral.	Total.	Asso- ciated with cranial nerve palsies.
General paresis - - -	—	—	42	36
Asthenia - - - - -	—	—	73	62
General paralysis - - -	—	—	4	4
Hemiplegia - - - - -	9	—	9	9
Hemiparesis - - - - -	14	—	14	12
Paralysis arms and legs -	20	10	32	25
	<i>i.e.,</i> arm and leg of one side.	<i>i.e., all</i> members.	mixed in two cases.	
Paresis arms and legs - -	15	14	30	24
	<i>i.e.,</i> arm and leg of one side.	<i>i.e., all</i> members.	mixed in one case.	
Paralysis of arms - - -	19	7	26	22
Paresis of arms - - - -	19	5	24	18
Paralysis of legs - - -	5	16	21	13
Paresis of legs - - - -	8	37	45	39
Paralysis or paresis of hands -	6	2	8	6
” ” ” feet - - - -	1	2	3	2
Paresis of shoulder and arms -	—	—	1	1
Paresis of shoulder and pectoral muscles.	—	—	3	1
Paralysis of chest muscles -	1	1	2	2
Paralysis of trunk and arms -	1	2	3	3
Paralysis of trunk - - - -	2	1	3	3
Paresis of abdominal muscles	—	—	2	—

Sensory Paralyzes.—Anæsthesia is a rare phenomenon in encephalitis lethargica and the schedule reports mention but

four cases, in two of which the loss of sensation was associated with a peripheral neuritis.

Thus, a young miner, shortly before his death, was noticed to be constantly biting his finger nails in consequence of neuralgic pains, and later his hand became anæsthetic; a woman of 21, who had been hysterical during her illness, developed palsy and anæsthesia in the ulnar side of one hand together with trophic ulcers; in the case of another young woman of neurotic tendencies, whose illness began with symptoms of insanity, there was “pins and needles” in the legs and then numbness in legs and forearms: in the last two patients, both of whom recovered, there was a strong neurotic bias.

(b) “Positive” localising Signs: *Involuntary Movements.*—Almost the last thing suggested by a patient suffering from the classical type of encephalitis lethargica is movement. Of late, however, there has accumulated much clinical evidence that the toxins of this disease have, in addition to their paralysing powers, a considerable capacity for producing irritating effects as shown by the various involuntary movements that are now so frequently observed. Moreover, the many different kinds of movements—almost baffling in their variety—which are described in the reports support Walshe’s contention that, in its exciting or irritating effect, the virus of encephalitis lethargica has none of the selective affinity so characteristic of its paralytic action; it would seem rather that every part of the nervous system may be attacked, from the lowest motor neuron up to the very hierarchy of the nervous centres.

MacNalty, it may be remembered, explains the association of paralyzes and involuntary movements somewhat as follows. In the localised types of encephalitis lethargica there is a selective action of the virus in or about the region of the IIIrd cranial nerve nuclei; this selective action appears to be so intense that often there is a spread of the action to other parts of the nervous system. Such extension is denoted by irritative effects. A comparison may be made with acute poliomyelitis where the virus of that disease is often observed to assail, as it were, various parts of the nervous system (as indicated by transient pareses) before “settling down” in a localised portion to produce paralytic effects.

Twitchings (myokymia) are frequently mentioned as one of the early signs, and when occurring in the facial muscles, as they often do, these fine movements soon attract attention; the fact also that they appear early in the disease serves at once to distinguish them from the flickering of fibres seen in muscular atrophy. In some cases, however, these and other tremulous movements occur, and may long remain, in muscles whose nerves are recovering from paralysis.

Fibrillary or fascicular twitchings were mentioned in some 143 cases and were distributed as follows:—

TABLE 38.

Various muscles of the face	-	-	-	43
" " upper limbs	-	-	-	20
" " trunk	-	-	-	6
" " leg	-	-	-	5
" " limbs	-	-	-	10
No localisation stated	-	-	-	29

The facial muscles are chiefly affected and particularly those near the mouth, while in the limbs the muscles of the shoulder-girdle were most often involved.

In one patient the fibrillary twitchings always ensued after exposure of the skin to air and in two cases it is mentioned that the movements continued during sleep, though probably this was the general rule. It may be noted that twitchings of abdominal muscles were mentioned only four times, but possibly they were not often sought for in this situation.

Myoclonic Movements.—In January 1920, Sicard and Kudelski (1920) described four cases of "encéphalite aiguë myoclonique" which were mainly characterised by lancinating neuralgic pains and by explosive rhythmical contractions like those produced by electric shocks; somnolence and ocular signs were absent, but the speech was staccato and the patients were constantly delirious until they became comatose and died. Netter, who was present at the meeting when these cases were discussed, considered that the patients in question were subjects of encephalitis lethargica. Thereafter many similar cases were reported, in France particularly, and in Germany, Switzerland, the United States and Britain: the association of myoclonus with encephalitis lethargica had become so firmly established on the continent that "l'encéphalite myoclonique" became a popular "type" of the disease, though it is doubtful whether in all the cases included in this type the contractions conformed to Friedreich's original description of myoclonus. Among the earliest cases of myoclonic encephalitis to be described in this country were those reported by Ellis in the *Lancet* of 17th July 1920 (313); in these cases insomnia and mild delirium at first took the place of lethargy, while the abdominal muscles were subjected to "forceful, short, rapid, explosive and rhythmical contractions"; eventually coma supervened and proved fatal in each case.

There is evidence, as pointed out by the author of these English cases and by Crookshank amongst others, that the condition had been observed in England—if not often reported—for some time previously and 4 out of the 24 instances of myoclonus supplied by the schedules were reported during 1919; a brief summary of a few cases reported during 1920 may be placed here.

Case 1157.—A schoolboy, aged 11, was attacked on 11th September 1920, with severe headache and vomiting, quickly followed by symptoms of acute mania and by rhythmical muscular contractions in the arms which eventually spread to the trunk and legs. He became speechless and lay in a condition of coma-vigil; there was ptosis on the right side and incontinence of urine, but no other paralytic symptoms; the cerebro-spinal fluid was clear, but the pressure was increased. By February 1921 the boy had completely recovered.

Case 1138.—A factory hand, aged 32, was suddenly seized with pain and myoclonic contractions in the muscles of the abdomen accompanied at first by insomnia and delirium; diplopia was also an early symptom and later there was lethargy deepening to coma. Blood examination showed a well-marked leucocytosis; this was not observed in the cerebro-spinal fluid which was also sterile, but under increased pressure. Death occurred after 12 days and at the post-mortem some injection of the pia and one or two patches of sub-pial hæmorrhage were found.

Case 1160.—A glassworker, aged 16, suddenly became ill with epistaxis, headache and restlessness; there was great thirst, the pulse was weak, erratic and fast (average 110) but fever was absent. The patient was lethargic by day, sleepless and delirious at night; pupils dilated and sluggish, double ptosis and lateral nystagmus. The right side of the body was affected with continuous myoclonic spasms together with fibrillary twitchings of individual muscles and delayed sensation; epileptiform fits also occurred and necessitated the use of chloroform which incidentally diminished, but did not suppress, the myoclonus. The knee jerks, at first exaggerated, were finally lost; examination of the blood showed a polymorphonuclear leucocytosis and in the cerebro-spinal fluid there was a relative lymphocytosis, but no increased pressure. The patient died after two weeks illness, and a naked-eye examination of the brain revealed congestion in the Rolandic areas which was more intense in the left side.

Case 1165.—A hospital nurse, aged 27, complained of spasmodic contractions of her abdominal muscles, and at first these movements were considered hysterical; the temperature rose, however, a double ptosis developed and the patient became lethargic by day, though wildly delirious at night. The illness was prolonged, and six months after the onset the patient was still troubled with spasms of the face and leg muscles.

From these and from other reports it appears that (i) myoclonus is a grave and often fatal symptom of encephalitis; (ii) it occurs in cases which have a marked "positive" bias and where lethargy, when present, is slight and broken by insomnia, delirium and psychic ebullitions; (iii) the abdominal muscles are particularly and often primarily affected, but the limb muscles may also be involved; (iv) a blood leucocytosis is common and the pressure of the cerebro-spinal fluid is increased; (v) coma generally precedes

death. The contraction frequency was noted in two patients, in one of whom the masseters twitched at the rate of 100-120 times per minute; in the second case, where the right half of the body was concerned, there were 45 contractions per minute.

Tremors.—Involuntary rhythmic contractions affecting synergic groups of muscles, and often marked by joint movements, were recorded in 20 per cent. of the total number of cases and occurred early in about one-fifth of the cases involved. In the majority of cases the localisation is unspecified, but tremors affecting the head, face, eyelids, mouth, tongue and limbs are recorded; the face and arm are the sites most frequently mentioned. The movements are variously described as "coarse" or "fine" and "marked" or "slight"; fine tremors resembling those of paralysis agitans occurred in a few patients, and in one of these a peculiar chewing motion of the mouth and jaws is described. Seven instances of *intention tremor* are quoted, and in the case of one patient it is mentioned that the tremors persisted during sleep: in a girl of 17, a weaver, the legs were subject to tremors for the space of three-quarters of an hour three times a day. Tremor may persist for a considerable time both during and after the acute illness, and in describing his own case, a medical man refers to "paretic crises" which periodically arise and in which there is a troublesome stiffness and weakness of the right arm combined with tremor and excessive salivation.

Cerebral Fits.—Clonic convulsions without loss of consciousness are seemingly rare and only two cases of *Jacksonian epilepsy* are recorded. A girl of 16, most of whose illness was marked by profound stupor, suffered from several "fits" that began in the right arm and extended up the neck to the right side of the face; this patient died. In another young subject the convulsions occurred in the leg and there was a positive Kernig; lethargy was moderate and a double ptosis and nystagmus were present. In this case, however, there was some cause for doubt, since the cerebro-spinal fluid which was under increased pressure gave a positive Wassermann reaction in 0.5 c.c. although the blood was negative in this respect; the patient recovered.

Epileptiform convulsions, as such, are recorded in four instances, but possibly in this class also should be included many of the general convulsive seizures briefly recorded as "fits." In a boy of nine the fits occurred during the third week of the illness and were followed by catalepsy; in a rapidly fatal case the epileptiform seizures were preceded by choreic movements, while in a patient, to whom reference has already been made, the fits were so frequent and distressing in character as to call for the use of chloroform.

General convulsions are reported in about 2.8 per cent. of the total cases; such fits occurred most frequently at the beginning of the illness—in three cases a fit was the herald symptom—less often they marked a recrudescence of the general symptoms and in one or two instances they took place just before death.

More than half the patients affected were young children, in some of whom probably the convulsion was toxic rather than organic in origin. Thus, a baby nine weeks old became alarmingly ill, with sudden loss of colour, profound unconsciousness and cessation of respiration; it was also overcome by two typical infantile convulsions. Recovery took place without the sequence of any focal palsies and indeed without any inherent proof that the illness was due to encephalitis lethargica; however, the occurrence in the same house and at the same time of two cases of the disease makes it most probable that this baby, hitherto strong and healthy, was also overwhelmed by the same virus, though in a manner more dramatic and with results more acutely toxæmic.

Choreiform movements.—We follow Walshe (1065b) in reserving for the more generalised choreic movements seen in encephalitis lethargica the highest of the three physiological levels of the nervous system, and by regarding these movements, in many cases, as motor manifestations complementary to the various psychic and emotional phenomena.

Choreic movements are mentioned in 21 cases of our series and as a rule they were exhibited by young subjects; in no case was there any history of a previous attack of chorea, and none of the patients apparently had suffered from rheumatic fever or valvular disease of the heart.

In four instances, it may be noted, choreic movements occurred during the onset of illness and the following case may be specially cited. A young factory worker while suffering from menorrhagia suddenly became hysterical and within 48 hours was seized with a "typical chorea" which lasted for three days, during which time there was nystagmus and fever; on the cessation of the choreic movements the nystagmus ceased and the fever abated, but in the meantime ptosis and squint had developed, the patient became lethargic and after some terminal delirium the patient died unconscious on the 17th day. A report stated that the findings at the post-mortem "were consonant with a diagnosis of encephalitis lethargica."

The early and close association between the emotional outburst and the choreic movements, together with the limited duration of the latter and the rapidly fatal course of the illness, strongly suggest that in this case the psycho-motor excitement was due to an irritative action on the highest centres. In more than half of the other patients who suffered from choreic movements there were also such irritative signs as insomnia, delirium, restlessness and a general psychological unrest. In the remainder of the cases, however, choreic movements appeared in patients who showed no emotional excitement or restlessness and whose symptoms were mainly paralytic both generally and focally; here the 'chorea' appeared late in the course of the disease and remained for some time.

Cerebellar Fits.—Although the reports of one or two of the cases suggest that the cerebellum was involved there is no record of cerebellar fits.

Hysterical Fits.—We are aware of a few instances in which a diagnosis of encephalitis was withdrawn in favour of hysteria, but the reports supply plenty of evidence that encephalitis lethargica may readily graft itself on hysterical stocks. In some cases, as in that described above, hysterical manifestations form an introduction to the attack of encephalitis; in other cases—to resume the analogy—there are hysterical outbursts from the old stock after the encephalitis has become established. But various psycho-motor disturbances which may be strongly suggestive of functional disorder, if considered without their context, are frequently reported in patients not previously considered hysterical; emphasis therefore may be again laid on the importance of recognising how often in cases of this disease “a strong functional element is present.”

Hypertonic Conditions.—Stiffness is mentioned in a few cases as one of the early symptoms which attracted the patient's notice; it was usually associated with indefinite pains and other symptoms of a general malaise. In a larger number of cases there was definite rigidity which was either a matter of complaint on the part of the patient or was proved to exist by passive movements; in some 4 per cent. of the patients there was neck rigidity not infrequently combined with head retraction and tenderness and nearly always accompanied by headache. This stiffness about the head and neck, though seen generally in patients with other “positive” symptoms, was by no means always accompanied by further signs of meningeal irritation; thus in nine patients only did neck rigidity and a positive Kernig coincide. The characteristic loss of facial expression which may occur early in some cases, and sooner or later in most stuporose patients, is probably due in part to rigidity or increased plastic tone; this mask-like appearance was so marked in a few instances that it was described as Parkinsonian.

Rigidity of the limbs was reported in about 5 per cent. of the cases and occurred during the later stages of the illness when the patient's lethargy had deepened into stupor or semi-coma. *Katatonie rigidity* (i.e., rigidity brought about by passive movements) is only rarely mentioned in the reports and the patient who was most severely affected in this way made a good recovery.

Spasmodic Conditions.—Apart from the increased tone and stiffness of the muscles in general, there is a spasticity about the groups of paralysed muscles in encephalitis lethargica as distinct from the flaccidity seen in the lower motor neuron lesions of acute poliomyelitis.

Athetosis (mobile spasm) finds mention in six schedule reports, but the part affected is not always clearly indicated; in two cases, however, the hand was involved. The condition arose late in the illness (which in no case proved fatal) and will be referred to again when the sequelæ are discussed: the

“*accoucheur's hand*” is only mentioned on a single occasion. Seven cases of *trismus* are recorded and *blepharospasm* was observed in two patients. *Cramps* in the muscles are occasionally mentioned, and in one case the tonic spasms were so severe and widespread that they were described as tetanic.

Pain.—With the exception of headache, so frequently reported, there does not appear to be any local pain particularly associated with encephalitis lethargica. A general aching referred to the back and limbs is frequently mentioned among the general symptoms, but during the course of the disease, and at such times as the patient is “taking notice,” there is seldom any complaint of discomfort; indeed, it is not uncommon for the sick man to express his sense of physical well being (*euphoria*). A comparatively large proportion of the various pains which are mentioned in the reports come under three headings. *Pain in the neck* and extending down over the shoulders is not uncommonly present and may be combined with the symptoms of irritation in the region of the shoulder girdle. Then *abdominal pain* or discomfort is not infrequently mentioned and in some cases is associated with clonic contractions of the abdominal muscles; the very obstinate constipation of encephalitis lethargica probably accounts, too, for much of the discomfort. Lastly, there are various *neuralgias* in which nerves at many different levels are apparently involved. Thus, in five instances, there was facial neuralgia; in one of these the Vth nerve was very severely affected, pain being experienced in the face, tongue and pharynx. The upper limb figures frequently, and in one case the pain, which started above the left clavicle and extended to the fingers and then the arm, is described as agonising; the ulnar and musculo-cutaneous nerves are specially mentioned in a few cases. Certain radiating pains resembling those connected with cord tumours are interesting and the reports provide three or four examples of these excruciations; in two such cases the roots of the lumbar nerves on one side were implicated. *Hyperæsthesia* is mentioned only three times and *paræsthesias* such as the “pins and needles” sensation appear to have been equally rare: it is doubtful, however, whether the schedule reports are very conclusive concerning disorders of sensation and no particular attention was drawn to the subject in the questionnaire; moreover the co-operation of the patient in the necessary tests is often not possible.

Sensory changes, it may be added, are often significant of functional influences.

4. *Appearance of Patient: Facies.*—There are not a few instances recorded where, in the early stages of the disease, some slight modification in facial expression was noticed by the patient's associates before he himself was aware of being in any way ill. In some cases it is stated that the patient had “looked queer for some days”; in others an observant parent would note that a child's “eyes looked funny”; in the case of a medical

practitioner, who continued at work during the prodromal period of what proved to be a severe attack, the general expression of the face was noticed by his patients to be altering. Such changes in the appearance, occurring in subjects who do not at first appear to be ill, may be significant as premonitory signs of the disease, reflecting some psychical disturbance or indicating that the muscles of the eye or face are being affected. As the disease progresses the face becomes more and more characteristic; the features are impassive and show no play of emotion, the creases become "ironed out" and the face assumes the appearance of a mask which in severe cases strongly suggests the Parkinsonian type of features.

In some cases there is an associated and bilateral paresis of the face which would account in large measure for the mask-like appearance; other signs of the face muscles being involved are dribbling and a tremulousness of the lower lip, though such happenings modify somewhat the general appearance of lifelessness.

In many cases, however, it would seem that the mask is caused by rigidity of the facial muscles, a condition which is shared by the body generally: the mask-like look is, of course, heightened by the lidded eyes—the result of deep slumber or of double ptosis—while the profound asthenia seen in so many patients contributes to the general impassivity.

In bed, the patient lies on his back with legs outstretched and arms by the side or folded over the chest and this position may be maintained for days.

Often there is great helplessness owing to the rigidity, but no murmur escapes the patient as a rule and, except for the possibility of delirious bouts at night, he remains quiet and immobile, "lying like a log" as so many reporters express it. Such an attitude contrasts strongly with that of the meningitis patient who lies curled up on his side, restless and resentful, and inclined to moan or cry out; there may be on the part of the encephalitis patient, however, a strong desire to be left alone, though in many cases he will, if strongly urged, carry out simple instructions after the manner of an automaton.

Katatonic rigidity of a limb was mentioned in ten cases, and twelve patients became cataleptic. In severe cases the closed eyes and expressionless face, the hands crossed on the chest, the fully extended legs and the general immobility suggest very strongly the appearance of an effigy both in aspect and in posture.

5. Speech and Articulation—Answers were given under this heading in about three-quarters of the total number of reports. In 25 per cent. of the answers it was stated that there were no speech changes and in the other 75 per cent. of cases the changes noted were as follows:—

TABLE 39.

Description of change in speech.	Number of cases in which the change was noted.
"Slow"	235
"Slurred," "slurring"	72
"Lost," "absent," "speechless"	62
"Thick"	55
"Indistinct"	47
"Difficulty in articulation"	40
"Hesitating"	39
"Unintelligible," "incoherent"	27
"Aphonia"	18
"Aphasia"	17
"Blurred"	17
"Nasal"	15
"Feeble," "weak," "faint"	15
"Long latent interval"	15
"Difficult"	14
"Monotonous"	13
"Sluggish"	9
"Drawling"	9
"Muttering"	8
"Staccato"	8
"Syllabic"	7
"Mumbling"	6
"Inclined to scanning"	6
"Toneless"	4
"Muffled"	4
"Husky"	4
"Low"	4
"Tremulous"	3
"Dull"	3
"Slow at first, then quick"	3
"Difficulty in finding words"	3
"Motor aphasia"	2
"Stammering"	2
"Bulbar paralysis type"	1
"Whining"	1
"Difficulty in forming words"	1
"Disjointed"	1
"Heavy"	1
"Auditory aphasia"	1
"Using wrong consonants"	1
"Whispering"	1

Examination of the above analysis shows a remarkable confirmation of MacNalty's description of the manner of speaking

in which he says, "In the type most commonly heard sentences are uttered *slowly*; words are *slurred* into one another, like the speech of a general paralytic and uttered *hesitatingly*" (629a). From the brief description given in the schedule reports only three instances of MacNalty's rarer type could be recognised, but possibly some of the cases classed as "unintelligible" should come into this category.

The psychical process of speech was completely arrested in 62 instances; *aphasia* without qualification is mentioned 17 times, while "motor aphasia" and "sensory aphasia" figure twice and once respectively. In the great majority of cases this loss of the power of speech was associated with deep stupor or with coma and was probably due to the patient's higher intellectual functions being in abeyance rather than to any definite affection of the cortical speech centres. Most of the cases in which speech was completely lost proved fatal, and in those that recovered the return to verbal fluency was protracted; many of the more trivial speech defects are those that might occur in any acute disease in which asthenia and delirium are prominent symptoms; in some cases the patient is deliberately silent.

A girl of 19, after recovery, found herself unable to spell as well as before.

Handwriting.—In three instances difficulties in writing or in composition attracted attention early in the prodromal stage: one lady lost the power of letter writing owing to words failing her; another was "found to be writing gibberish," and in a third case the handwriting became shaky and much smaller than usual.

Articulation.—*Dysarthria* was definitely noted in 41 cases and to this number should probably be added some of the instances in which the speech or voice is described as "difficult," "unintelligible," "blurred," "nasal," and so forth. In many cases the disordered articulation was correlated with a paresis or paralysis of the face, lips, tongue or palate and in one patient the speech was specified as "bulbar in type." In two instances *stammering* supervened. The repeated mention of "slurring" suggests that in many cases the cortico-nuclear neurons are involved as in alcoholic intoxication and in general paralysis of the insane. Cerebellar influence was possibly responsible in some 12 cases for the articulative ataxia as shown by a scanning or staccato speech.

6. Reflexes and Special Signs.—Explicit information regarding the superficial, deep and organic reflexes was given in just over 500 instances and is analysed in the tables below:—

TABLE 40.
Superficial Reflexes.

Nature of reflex.	Number of recorded observations.	Normal reaction.	Reflex obtained.	Reflex not obtained.	Diminished reaction.	Exaggerated reaction.
Superficial reflexes as a whole	21	16	—	3	—	2
Cremasteric	1	1	—	—	—	—
Corneal	2	—	—	2	—	—
Abdominal	25	5	—	10	3	7
Plantar flexion	21	14	—	2	0	5
Plantar extension (Babinski's sign)	78 (+ 21)	—	47	31 (+ 21)	—	—

TABLE 41.
Deep Reflexes.

Nature of reflex.	Number of recorded observations.	Normal reaction.	Reaction not obtained.	Diminished reaction.	Exaggerated reaction.	Various reactions.
Deep reflexes as a whole	26	22	2	—	2	—
Knee jerks	213	46	58	• 26	60	23 { absent or diminished, then normal (5). absent or diminished, then exaggerated (4). normal or diminished, then absent (7). exaggerated, then absent (7).
Ankle clonus	28	Present. 11	17	—	—	—

TABLE 42.
Organic Reflexes.

Number of recorded observations	-	-	-	404
Action of anal and vesical sphincters reported as				
normal	-	-	-	29
Retention of urine	-	-	-	77
Incontinence of urine	-	-	-	119
Incontinence of urine and faeces	-	-	-	160
Retention and then incontinence of urine	-	-	-	8
Retention of urine and incontinence of faeces	-	-	-	2
Incontinence of faeces	-	-	-	9

TABLE 43.

Special Signs.

Sign.	Number of recorded tests.	Positive reaction.	No reaction.
Babinski's - -	99	47	31 (Flexor 21)
Brudzinski's - -	6	0	6
Drodzenaki's - -	1	0	1
Kernig's - - -	123	74	49
Romberg's - - -	3	1	2
Oppenheim's - -	2	2	—

Superficial Reflexes.—References to the skin reflexes as a whole are few with the exception of those concerning the plantar reaction. A positive *Babinski* was obtained in roughly 47 per cent. of the cases in which there is a record of this reaction having been specially investigated. Generally, the sign is well-marked and present either in both limbs or unilaterally; in the case of a patient of 63, who suffered from neuritis of both legs and in whom there were exaggerated knee-jerks and ankle-clonus, the plantar reflex remained extensor for two months. In some instances, however, Babinski's sign was ephemeral in character—"present one day, gone the next"—suggesting Boyd's explanation (121*b*) that the phenomenon in these cases was probably due, not to destructive lesions of the nerve centres, but to some temporary affection of the related nerve tracts.

Deep Reflexes.—The state of the *knee-jerks*, as representing the tendon reflexes, finds mention in some 240 cases. According to the statements this reflex was more often abnormal than otherwise, but it is probable that it was examined more frequently than the records reveal and that in the majority of cases the reflex was normal. There is apparently no sort of constancy about the abnormal reactions and this is not surprising in a disease whose virus has such a large field of action; only occasionally, too, do the schedule reports give more than the state of the reflexes at one particular phase of the illness so that any deductions must be necessarily inconclusive on that account.

In some cases the response was exaggerated but on only a few occasions was this exaggeration associated with *ankle clonus*; diminution or absence of jerks in one or both limbs was more frequently noted than exaggeration, and "lost near the end" is a frequent note in connection with the reports on fatal cases. The most noteworthy feature of the knee-jerk is, perhaps, its variability according to the stage of the disease and the general inclination of the nervous symptoms towards a "positive"

or "negative" bias; in some cases, too, an overshadowing emotional state is reflected by an exaggerated response for some time during convalescence.

Organic Reflexes.—The references in this respect among the reports are confined to the retention or incontinence of urine and faeces, and the remarks made under this heading in a previous report (588*a*) so nearly represent what emerges from the present enquiry that they may be quoted here. "*Retention of urine* may occur early or midway in the disease. It may be partial or complete, and last one day or for a week or more. When it has existed for an appreciable time it usually gives place to reflex incontinence; often such a condition is probably due to retention with overflow incontinence. *Incontinence of urine* apart from retention usually appears late in the course of the disease. It is associated sometimes with lack of control of the anal sphincter."

The last statement only needs modification in the light of further experience and as will be seen in Table 42 incontinence of urine is more often associated with incontinence of faeces than otherwise. It may be emphasised, too, that not only does the loss of sphincter control occur late in the course of the disease, but also that it is generally associated with a deepening and fatal coma. Of the 279 cases in which single or double incontinence was noted, 67 per cent. proved fatal.

Kernig's sign was reported as present in 74 cases, though in 11 of these there was some doubt about the reaction; in many instances the sign was transient and corresponded with a passing phase of irritation. It was observed, indeed, most frequently in cases where both "positive" and "negative" symptoms were present, while in 10 instances where the sign was present the symptoms reported were chiefly "negative"; thus the reaction was recorded as present in a child of ten whose chief symptoms were ophthalmoplegia and coma with absence of knee-jerks and in whom photophobia was the only sign that could be considered "positive." Other irritative signs which were associated with a positive Kernig were headache (36 times); head retraction (4 times); stiffness and pain in neck (9 times), general rigidity (19 times) and tremors (30 times).

A positive Kernig, like the presence of optic neuritis, is sometimes regarded as incompatible with a diagnosis of encephalitis lethargica; there is probably less reason for hesitation, however, in the case of the first mentioned phenomenon than in the case of the second, since both the meninges and the sacral nerve roots may be implicated in encephalitis lethargica. In the more recent literature, too, the sign is constantly mentioned as present; thus Schwartz (947) reports a positive Kernig in eight out of 10 consecutive patients treated at the Harlem Hospital, New York.

Babinski's sign has been referred to under superficial reflexes.

Special Signs in Encephalitis Lethargica.—In the American literature of 1920 certain special signs are described which are

thought by the several authors to be useful in the diagnosis of encephalitis lethargica. Thus, *Reilly* (852), writing in March 1920, describes a "Hitherto undescribed sign in diagnosis of lethargic encephalitis" which consists of a "rhythmic convulsive twitching of the muscles of the abdomen in the neighbourhood of the eighth and ninth ribs. It often simulates the muscular movement of hiccough, except that it is one-sided." The sign was noted in both mild and severe cases.

Cadwalader (162) calls attention to "the diagnostic importance of the occurrence of bilateral sympathetic ophthalmoplegia in cases of lethargic encephalitis" which he explains as follows: The ophthalmoplegia consists of contraction of the pupil, narrowing of the palpebral fissure and slight retraction of the eyeball. These symptoms are due to a lesion in the tract containing oculopupillary fibres and when such a lesion is "diffuse or inflammatory" there is a bilateral involvement of the sympathetic nerves of the eyes. The author adds, however, that the condition "is not likely to attract attention and is overlooked" because of its bilateral nature.

Sainton and Cornet (924) refer to a "frontal sign" often present in some forms of encephalitis lethargica and which consists in a slow contraction of the frontal muscles when the upper eyelids are raised.

Thorp (1020) describes as a sign of encephalitis lethargica a curious behaviour of the pupils when these are partially shaded by the finger of the observer. The shaded portion of the pupil contracts sluggishly and may assume various shapes according to the position of the shadow and the length of time it is in operation. "The disappearance of the sign appears to be of favourable import, and the sign appears most easily elicited during the drowsy stage."

Eccentricity of pupils was noted in one of the cases of our series, but here the condition was not apparently induced.*

7. Trophic Symptoms.—There is no mention in the reports of any trophic disturbances in the muscles, nor is there any evidence in the after histories that wasting and degenerative changes have been observed.

The skin was rarely involved; *bed sores* were reported in seven instances and *bullæ* in three, while one patient suffered from trophic ulcers of the fingers.

8. Vegetative Nervous System.—It is not clear from the reports how far the cervical sympathetic may have contributed to the oculo-pupillary changes so often seen, and only in the following

* Professor Jean Piltz of Cracow, writing in October 1920, (810), claims that he was the first observer to note, in cases of encephalitis lethargica, the occurrence of "amoeboid movements" and irregularity of the pupils; he states further that he has observed the same phenomena in a child suffering from tuberculous meningitis and also in a newly-born baby instrumentally delivered. No claim is made by this observer, however, that he experimentally produced these pupil changes as described by Thorp.

three cases of *exophthalmos* does the implication of this part of the nervous system seem assured. In a woman of 36 there was slight prominence of both eyes; the pulse was consistently rapid, and the patient was "fussy and troublesome." There was moderate lethargy with temporary ptosis and squint, bilateral facial palsy and arthritis in the right hip; the patient became weak and wasted, but eventually recovered. The other two patients lived in the same town: one a woman of 53³ suffered chiefly from ophthalmoplegia, right facial palsy and lethargy. The third patient, a man of 47, who was frequently excited and otherwise emotional, developed a very distinct double exophthalmos, the right pupil being dilated and vision blurred: there was facial paralysis, at first unilateral, and then double, together with great weakness and loss of flesh. All three patients recovered and of none is it recorded that the thyroid was enlarged.

Sweating is mentioned in 26 cases and was occasionally severe; it usually occurred early, but in a few instances it was a late symptom associated with hyperpyrexia; though usually general in distribution the sweats in a few cases were confined to certain areas notably the face and the hands. In two patients there was associated *face-flushing* which is also recorded on six other occasions.

E.—COURSE OF THE DISEASE.

1. Duration.—The conduct of the disease varies considerably in different cases and some of the criteria used in other infectious diseases are not available here: the temperature, for example, is no sure guide, for in many cases there is no apparent correlation between the rise and fall of fever and the incidence of nervous symptoms; in a certain number of instances, too, there is an absence of fever throughout the illness. Rashes again have very little significance since they are rarely observed and when present are so variable in character and time of appearance. Then, the transient character of the motor palsies, which is one of the most significant features of the common paralytic types, makes it difficult to set up any time limits, for with the rapid clearing of one set of palsies another set may be developing elsewhere; or the hopes raised by the disappearance of paralytic symptoms that an improvement has set in may be frustrated by the onset of irritative signs. The degree and duration of the stupor provide, perhaps, the best means of estimating roughly the duration and course of the illness as a whole.

Of both mild and fatal attacks it may be said generally that the disease usually runs a comparatively short course and the patient in each case respectively recovers or dies within a period of three to four weeks; in the case of patients who do not die as a result of a moderate or severe attack the length of the illness varies within wider limits.

The following table shows the position as regards 414 fatal cases in which the necessary data were available :—

TABLE 44.
Deaths.

Week of illness in which death occurred.	Number of fatal cases.	Week of illness in which death occurred.	Number of fatal cases.
1st week	75	13th week	1
2nd "	138	14th "	1
3rd "	91	15th "	1
4th "	39	16th "	1
5th "	18	17th "	1
6th "	13	18th "	0
7th "	7	19th "	0
8th "	5	20th "	3
9th "	6	21st "	0
10th "	5	22nd "	1
11th "	3	23rd "	1
12th "	3	30th "	1

It will be seen that a very large proportion, 51·2 per cent., of the fatal cases end within two weeks, while 82·8 per cent. of all the patients who succumbed died within a month.

Relapses, or the supervention of some complication, account for some of the instances where the disease lasted for more than 4-6 weeks before proving fatal.

It is not easy from the reports to estimate at all accurately the length of illness in the non-fatal cases; still more difficult is it to determine the point at which the patient can be said to have fully recovered from his illness. From data yielded by some 250 schedule reports average figures for the duration of the illness at various age periods have been worked out below :—

TABLE 45.
Recovery.

Age period.	Average length of illness.	Age period.	Average length of illness.
0-1 year.	4-5 weeks.	30-40 year.	8-9 weeks.
1-5 "	5-6 "	40-50 "	9-10 "
5-10 "	6-7 "	50-60 "	9-10 "
10-15 "	11-12 "	60-70 "	9-10 "
15-20 "	7-8 "	70-80 "	8-9 "
20-30 "	5-6 "		

For purposes of the above table the "length of illness" was considered to be the period which started with the definite onset of the disease and ended with the reappearance of the patient downstairs, still weak, but with no active symptoms. Two months, perhaps, may be given as the time during which a good

proportion of the patients are away from work on account of their illness, but it will be noticed that patients between the ages of 10-15, who are relatively very susceptible to attack, are also much longer ill than patients of any other age period.

In another large proportion of cases the illness is more prolonged and convalescence becomes a tedious and dispiriting experience; various late effects, to be mentioned presently, may follow closely in the wake of the acute symptoms and delay for months the patient's complete recovery. These later symptoms, though generally described as sequelæ, are considered by certain authorities as being due in some cases to the prolonged activity of the original dose of virus. Thus von Economo reports the case of a man who was taken ill in April, 1917, and his apparent recovery was followed by remittent attacks in which pseudo-bulbar paralysis and athetosis were conspicuous features; the patient died in January, 1919, and his brain showed both old and recent lesions of encephalitis lethargica: such a case is not unreasonably described as one of "chronic lethargic encephalitis." Among our own records is the case (No. 228) of a docker, aged 19, who was attacked with encephalitis lethargica on 19th January, 1919, and whose illness lasted until 31st December, 1921, when the patient died.

2. **Relapses.**—These are said to have occurred in some 20 cases only and in a certain proportion of these the term "relapse" is probably incorrect. In the case of two patients whose attacks were separated by intervals of one and two years respectively, the possibility of re-infection cannot be excluded. Thus a man of 59 was taken ill on 30th April, 1920, with headache, giddiness, diplopia and increasing lethargy from which he slowly recovered; two years previously he had suffered from ophthalmoplegia, stupor and aphasia and was ill for about 12 months.

In other instances the invalid does not relapse into a former condition, but reappears after an interval of some weeks or months as a patient with different manifestations of the disease. Thus a man of 45 was treated in hospital during December, 1920, when his symptoms were comparatively slight and consisted chiefly of diplopia and glycosuria. The diplopia cleared up completely and he remained fairly well, though weak, until July when he was admitted to another hospital complaining of aching and stiffness in the limbs; it was significant also that he had been for some time troubled with dribbling of saliva. While in hospital the muscular stiffness and weakness increased and was very marked in the lower jaw. The gait was stiff and definitely festinate, the joints of the limbs hypotonic; there were also pronator-supinator tremors of both hands. Diplopia did not recur, the fundi were normal and cerebation was good: the Parkinsonian effects in this case were probably late symptoms, or sequels, of the original illness. In a few cases, however, there is a definite return of symptoms during the time that the patient was thought to be well on the road to recovery. For instance, a schoolboy, aged 14, became ill at the beginning of February

1920 with pain in the head and eyes, mental confusion and slight fever; later he became lethargic and developed a left-sided paralysis. He returned home on 24th February and remained apparently well until 3rd March when he became restless and then semi-conscious; he was admitted to Guy's Hospital on 7th March and treated as a relapse case of encephalitis lethargica. By 6th May he was perfectly well and back at school.

Notes on a case in which there were two relapses will be found in Appendix I, Case H.

A remission in the symptoms is comparatively common and occurs especially in connection with stupor, from a bout of which a patient may emerge and may be thought to be "taking a turn for the better" when he subsides once more into semi-consciousness. Similarly, a general improvement accompanied by a clearing of paralysis may be interrupted by the appearance of new palsies or of involuntary movement together with a relapse into lethargy, or possibly an access of delirium.

3. Complications.—Cases of encephalitis lethargica are rarely beset with complications and this may be partly explained by the fact that the many fatal illnesses have a comparatively short course. Excluding hyperpyrexia, the cases in which complications occurred form only 2 per cent. of the total number, and much the greater part of this percentage is made up of pulmonary conditions occurring towards the end of the illness. Boveri (119, b.) emphasises the extreme rarity of bronchial or pulmonary localisations in encephalitis lethargica, either at the beginning or during the course of the disease, and considers that this immunity from lung complications is an important diagnostic sign. The complications recorded are :—

TABLE 46.

Cases.	Cases.
Pneumonia - - 9	Cardiac incompetence 3
Broncho-pneumonia 2	Jaundice - - - 1
Hypostatic congestion 5	Femoral thrombosis 1
Septic pneumonia - 1	Nephritis - - - 1
Bronchitis - - 9	Arthritis - - - 1
Hæmaturia - - 3	

Temperatures of 105° F. and over were recorded in 36 instances; in some six cases the fever was associated with pulmonary complications, while three patients with high fever exhibited strong emotional symptoms. In the remainder of instances the sudden rise of temperature coincided with a deepening of coma and may possibly have been due to involvement of the heat centres so that in such cases the hyperpyrexia might be rather regarded as an occasional and grave symptom of encephalitis lethargica.

4. Sequelæ.—As previously mentioned, it is not easy to draw a line between the sequelæ and the long-continued symptoms of this disease, but the Table in Appendix II. gives particulars of 271 patients who were seen at periods varying from 2-18

months after the onset of their respective illnesses. Below are set out the after-effects recorded in the case of some 250 of these patients of whom reports were received after an interval of not less than three months from the onset of symptoms.

TABLE 47.

Sequelæ.

After condition.	No. of cases.	After condition.	No. of cases.
General weakness, asthenia -	35	Psychic changes - - -	47
Mental impairment - - -	57	Insomnia - - -	12
Drowsiness - - -	15	Headache - - -	11
Neurasthenia - - -	7	"Chorea" (chronic movements) - - -	3
Melancholia - - -	4	Fits - - -	4
Loss of memory - - -	6	Tremors - - -	45
Speech changes - - -	14	Athetosis - - -	10
Ocular defects—		Rigidity - - -	9
Indefinite palsies - - -	17	Abnormal gait - - -	4
Squint - - -	16	Inco-ordination legs and arms - - -	1
Impaired vision - - -	16	Exaggerated tendon reflexes - - -	3
Diplopia - - -	8	Pain - - -	3
Nystagmus - - -	6	Vertigo - - -	1
Blindness - - -	3	Vomiting - - -	1
Ptosis - - -	3	Exophthalmos - - -	1
Paralysis or paresis face -	17	Enlarged thyroid - - -	1
" " upper limbs -	17	Adiposity - - -	2
" " lower limbs -	15		
" " leg and arm -	3		
Incontinence of urine -	3		
Constipation - - -	1		
Muscular wasting - - -	1		

It may be noted that in their nature the various sequelæ resemble generally the preceding symptoms and probably imply in many cases a prolongation of conditions originating during the active course of the disease; some of the eye palsies, cases of disordered speech, and instances of continued drowsiness may be quoted in this connection. On the other hand, there are some marked changes that come more naturally within the comprehension of sequelæ and which are evidently common to a large number of patients. Firstly, in all but mild cases there is a good deal of *physical exhaustion* and *muscular weakness* evident to patient and friends alike and more marked sometimes than the length of illness would seem to warrant. With this physical exhaustion is combined, in a large number of cases, some degree of *mental impairment*—ranging from a slight blunting of the intelligence to amentia—so that the patient cannot resume his previous work; he may be further handicapped by a defective memory; occasionally the patient may sink into a neurasthenic state or become melancholic. More striking, perhaps, are the "positive"

signs of a disordered mentality and the change in character which are frequently noted; "highly emotional," "nervous and excited," "very irritable" are expressions often used, with the rider in many cases that such states were quite foreign to the patient before his illness. In children especially, the *disposition* may alter very markedly for the worse; thus two boys, previously honest, became thieves; another, usually of peaceful disposition, became a bully; while in other cases there were uncontrollable fits of temper. Our records show but one instance of the tendency to unclean habits, which is said by some observer of the disease in children to be a frequent sequel. In some three or four known instances it was necessary for the patient to pass part of the convalescence in a mental asylum owing to the development of maniacal symptoms. It will be noted that "choreic movements" appears but three times in the list of sequelæ; in two of these patients known to one of us the condition has become chronic, and in one, a boy of 12, the *thyroid gland* has become enlarged at the same time. Fits are recorded three times, and in one instance where epilepsy had developed, the patient subsequently died during an epileptic attack.

As regards localising sequelæ, the chief stress falls, as in the attack itself, upon the eye; very commonly in such cases a squint remains, and in three instances diplopia was observed more than nine months after the illness began. Difficulty in reading and writing may be experienced, and there is a frequent note as to the need for glasses; two patients became blind and in another there was optic atrophy on the left side.

Next to the eyes the face most frequently remains affected both with paralytic and with irritative disabilities; the loss of power may be slight, or great enough to leave facial disfigurement, and in muscles near the mouth the paresis may remain for some time, as in the case of a patient who was still troubled with dribbling of saliva 14 months after his illness. Twitchings or *tremors* of facial muscles may re-appear when the patient is tired or mentally excited; in other cases facial tremors have remained constantly present for over a year.

Comparatively common also are the records of paresis or paralysis in the limbs; in some case the term *residual palsies* seems applicable, while in others again the weakness has appeared for the first time during a prolonged convalescence.

Such belated palsies may supply another argument in favour of the supposition that the virus continues present and potent for some time after the acute symptoms have gone, and in these cases of late limb palsies it may be extending its action to the spinal cord. The "*paretic crises*" experienced and described by a medical man might also suggest that occasionally the smouldering embers of infection may be fanned into small flames by some circumstances adversely affecting the patient.

Complete histories of the 10 cases in which *athetosis* was noted as a sequel are unfortunately not available, but the spasm affected

the hand alone in six cases, the foot alone in two, hand and foot in one, while in one case the left side of the body as a whole was involved. In one instance only is there recorded the presence of a pre-existing paralysis or paresis in the part affected, but in the great majority of patients there were significant mental changes and two patients became maniacal. Express references to *symptomatic paralysis agitans* do not occur, but mention is made of the mask-like face, stiffness, festinant gait and other features of the Parkinsonian picture.* Reference may be made to the two cases of abnormal *fatness* since this condition is also recorded as a rare sequel by other observers. In the case of a girl who was seen by one of us the adiposity was marked all over the body and the patient had also become choreic.

On the other hand, there may be very great *emaciation*, such as was observed by MacNalty in the case of two children (brother and sister) at Eastbourne.

"Not the man he was" is an oft-repeated remark that fairly sums up the mental and physical deterioration that marks the patient who has come through a severe attack of *encephalitis lethargica*, and in such a one there often linger for some time traces of those lesions which made the deepest impression during his acute illness.

5. Mortality.—In the two tables immediately below are set out the mortality figures at different age periods, in respect of a total of 1,273 cases notified from England and Wales (including London) during the years 1919 and 1920: *the case mortality works out at 48.3 per cent.*

* Those who are particularly interested in the subject of post-encephalitic paralysis agitans should refer to the *Revue neurologique* for June 1921 (see Bibl. No. 1222). In this number is reported at length the proceedings of the second *Réunion neurologique annuelle* which was held in Paris on the 3rd and 4th June, 1921. The Congress, conducted by the Société de Neurologie de Paris, was international in character and was attended by official delegates from Belgium (M. Marchal), Britain (S.A. Kinnier-Wilson), China (S.A. Chuan), Denmark (Pr. Vizzo Christiansen), Italy (le Pr. Camillo Negro), Japan (M. M. Takasogi Shiniciro), Portugal (Pr. Antonio Flores), as well as by a great many other eminent neurologists from various parts of the world. The subject for discussion was a report by M. Souques (France), entitled: "Les syndromes Parkinsoniens," and the interest centred mainly in the statement introduced in the opening sentence of the report, which was: "La paralysie agitante ou maladie de Parkinson n'est pas, à mon avis, une entité morbide." M. Netter opened the discussion with some remarks on "les relations entre l'encéphalite léthargique et la maladie de Parkinson," and many others who joined in the discussion compared or contrasted the etiology, symptomatology, pathology, prognosis, &c. of "la maladie de Parkinson" with similar aspects of "le syndrome Parkinsonien encéphalitique." In winding up the discussion M. Souques maintained his stand with those who would admit more or less completely the identity of the two affections, and he repeated, in conclusion "il n'y a pas, à mon avis, de signe tiré de la symptomatologie ni même de l'évolution, jusqu'ici, qui permette de distinguer un Parkinsonien post-encéphalitique d'un paralytique agitant classique."

TABLE 48.

Quinquennial periods 1-20 years.

0-1 year.			1-5 years.			5-10 years.			10-15 years.			15-20 years.		
Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.
12	9	75.0	74	50	67.5	95	45	47.3	151	50	33.1	176	74	42.0

TABLE 49.

Decennial periods 20-80 years.*

20-30.			30-40.			40-50.			50-60.			60-70.			70-80.		
Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.	Cases.	Deaths.	Death Percentage.
191	80	42.1	193	93	47.8	187	94	50.0	121	72	59.5	57	36	63.3	15	12	80.0

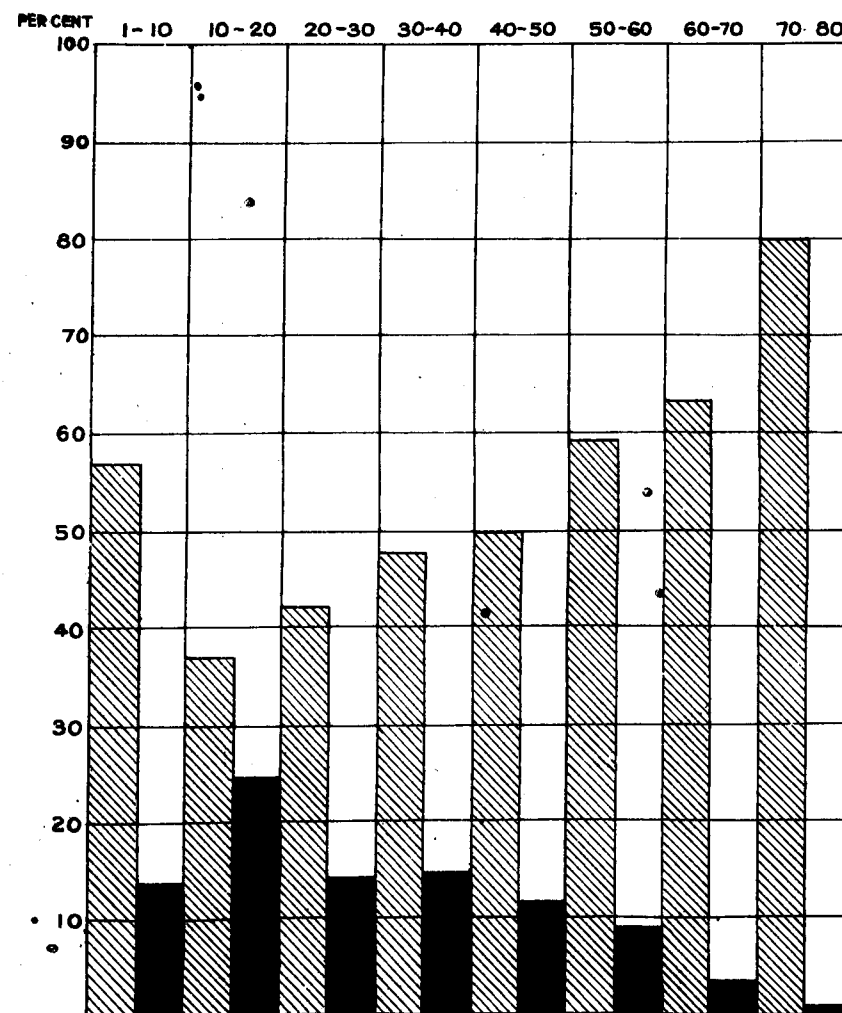
* In addition to the cases tabulated above a woman of 84 was notified as having died from encephalitis lethargica.

From the statistics given above it will be seen that in infants the death rate is high, but the percentage figures decrease in each succeeding age-period until the quinquennial period 10-15 is reached; from this point the case mortality figures steadily rise without any break, and in the last period tabulated the percentage of deaths has reached 80 per cent.

This gradation is shown in the accompanying chart, No. VI, where the black columns represent the percentage proportion of the total number of cases occurring at various age-periods, while the shaded columns show the percentage of deaths in respect of the numbers of cases grouped under the various age-periods. For example: of the 1,273 cases, reports which were finally accepted for analysis 15 (1.1 per cent.) referred to individuals between the ages of 70 and 80, and of these 15 patients, 12, or 80 per cent., died.

Chart VI.

**SHOWING THE INCIDENCE AND MORTALITY.
RATES AT VARIOUS AGE PERIODS.**



Black Columns show case incidence according to ages; Shaded Columns represent case mortality at various age periods.

As regards the sexes, the statistics do not show any appreciable difference in the mortality rate; in a total of 615 deaths, females preponderate by 25 only.

The mortality (1920 series only) according to the type of case is seen in the following table :—

TABLE 50.

Type of case. (See footnote p. 55).	Number of cases.	Case mortality.
I — — — —	31	61·2 per cent.
I + — — —	12	41·6 „
II + — — —	449	47·6 „
II — — — —	123	47·9 „
II + + — —	55	49·0 „
II + — — —	28	32·1 „

There is practically no difference between the death rate in England and Wales during the years 1919 and 1920 and the figures for each year respectively approximate very closely to those for the two years combined; thus :—

TABLE 51.

1919.	1920.	1919 and 1920.
48·7.	48·0.	48·3.

With the exception of London, no town supplies a sufficiently large number of cases from which to draw any useful comparisons between the death rate in different districts. For London during 1920 the rate was considerably lower than that representing the county as a whole and amounted to 36·4 per cent.; in Bristol where just over 50 cases were notified during the two years under review the mortality rate was 46·0.

The death rate among patients treated in hospitals during 1920 is shown in the table below :—

TABLE 52.

1920.				
District.	Type of hospital.	Number of cases admitted.	Percentage of deaths.	Percentage of deaths among hospital patients throughout the country.
London -	General Hospitals -	86	24·0	41·4 „
	Fever Hospitals -	6	0·0	
Provinces	General Hospitals -	200	50·0	
	Isolation Hospitals	34	38·0	

We realise that a general case mortality rate of 48.3 is not only high, but that it is considerably higher than the figures quoted by many other observers, though these vary considerably as will be seen in the following list :—

TABLE 53.

Country or district.	Year.	Number of cases.	Number of deaths.	Case mortality rate.	Remarks. (The numbers refer to the bibliographies.)
England and Wales.	1918 Jan.-June	168	37	22.0 per cent.	Cases investigated by MacNalty (629.a.).
Manchester district.	1920	32	17	53.0 "	McClure. (617).
Edinburgh district.	1919	57	9	15.7 "	Bramwell. (126).
Scotland	1919-20-21	22	11	50.0 "	Forty cases "intimated" to S.B. of Health; result not stated in 18 cases.
Ireland	1919-20	30	10	33.0 "	Information supplied by L.G.B., Dublin.
France	1917	40	—	28.5 "	Crutchet, Moutier & Calmettes. (246.a.).
"	1918-21	32	12	37.5 "	Crutchet (243.g.).
"	1920	464	118	25.4 "	Official figures.
Denmark	1919-20	58	13	24.5 "	
Italy	1919-20	3,900	1,013	25.9 "	Chiefly in Northern and Central Italy.
Switzerland	1920	984	—	29.4 "	Official figures.
Portugal	1920	65	20	30.0 "	" "
Vienna	1920	71	31	42.0 "	" "
United States	1918-19	122	42	34.4 "	" "
"	—	100	—	31.0 "	Dunn and Henegy from American literature. (300).
Winnipeg	1920	75	29	38.6 "	Boyd (121.b.).
From the literature.	—	864	—	20.5 "	Strauss and Wechsler (1001).
South Africa	1920	13	12	92.3 "	Native patients. Butt (155).

The case mortality rate of 48.3, as derived from the notification figures for 1919 and 1920, must certainly not be taken as a true estimate of the proportion of deaths that occur among people attacked by encephalitis lethargica. The notification returns assuredly do not represent the total number of cases that have occurred and it is just the mild non-fatal cases that are not included; probably also there are other instances in which notification did not take place owing to the diagnosis being in doubt. It is, of course, more than likely also that the cases accepted for analysis include a few fatal ones which were wrongly given the benefit of the doubt in favour of encephalitis and under the circumstances of the survey some degree of error in this respect is inevitable. At the same time, the notifications of encephalitis lethargica, since the beginning of 1919, have been subjected to very special scrutiny such as rarely obtains in the

case of other notifiable diseases where the incidence and mortality figures are officially accepted without further question: many cases originally notified as encephalitis lethargica have been excluded for purposes of this analysis, and if these are set against the number which, through human fallibility, may have been improperly included, the mortality rate of 48.3 among notified cases is probably not far out. Some measure of control is provided by the mortality percentages that obtain in the different sets of circumstances which are compared below.

TABLE 54.

Circumstantial references.	Case mortality. Per cent.
Cases notified from England and Wales, including London, during 1919 and 1920	48.3
Cases notified from England and Wales, including London, during 1919	48.7
Cases notified from England and Wales, including London, during 1920	48.0
Cases notified from England and Wales, including London, during 1921, as far as is known at present	42.0
Cases notified from Bristol during 1919 and 1920	46.0
Cases (109) in which a consultant concurred in the diagnosis	50.0
Cases (236) treated in hospital	41.4
Cases (264) in which examination of the cerebro-spinal fluid suggested no other diagnosis	47.0
Cases (71) in which the cerebro-spinal fluid was stated to be sterile	50.0

It may be urged that the figures in the case of hospital patients tell the other way; it is doubtful, however, whether as regards encephalitis lethargica the hospitals receive mainly patients who are seriously ill, and obscurity of diagnosis or inexperience of the disease, rather than gravity of symptoms, often suggest removal to hospital where such is available.

F.—DIAGNOSIS.

The typical manifestations of encephalitis lethargica are characteristic enough to make diagnosis comparatively easy in those cases which conform to the usual type, and more especially when the disease is exhibiting epidemic tendencies. On the other hand, some of the typical clinical signs are so impressive that their absence or modification in mild and abortive cases of the disease has made diagnosis in such instances a matter of some uncertainty, especially during inter-epidemic periods. The chief diagnostic difficulty is found in cases where the symptoms closely simulate those of other maladies, both allied and alien;

indeed, the mimetic powers of encephalitis lethargica are very striking as shown abundantly by the clinical reports and by post-mortem investigations. The disease, too, at different stages of its course may adopt various guises; thus, one patient was thought at first to be suffering from typhoid fever, later cerebro-spinal meningitis appeared to be the correct diagnosis, and eventually the case proved to be one of encephalitis lethargica. A comprehensive view of all the historical circumstances, together with a close and continuous study of the clinical symptoms of each case are most important, trite as such an observation may appear; often the diagnosis in obscure cases can only be reached by a process of exclusion, and in not a few instances the diagnostic riddle is only solved at the bench of the histo-pathologist.

In the absence of any confirmatory evidence from the laboratory or mortuary it has, indeed, been difficult for us to be sure, from the clinical data alone, that a diagnosis of encephalitis lethargica was justified in some of the reported cases. Such doubts have become strengthened, too, by a consideration of the evidence supplied in certain special circumstances where careful bacteriological and post-mortem examination was possible. Thus Dr. Perdrau, who examined the brains of 14 subjects in whom a provisional diagnosis of encephalitis lethargica had been made during life, found that in only two instances was death due to encephalitis lethargica. Dr. Brincker (London County Council), also, has kindly sent us notes of 45 cases of illness, occurring in the Metropolitan Boroughs during 1921, and concerning the diagnosis of which the help of the medical department (London County Council) was invoked. A provisional or alternative diagnosis of encephalitis lethargica was suggested in 41 of these cases by the practitioners in charge and, as a result of clinical consultation, examination of cerebro-spinal fluid or post-mortem investigation, the diagnosis was upheld in 17 instances and rejected in 24; on the other hand, three cases which at first were thought to be tuberculous meningitis and one which was described as toxæmia (pneumo-coccal or influenzal) were eventually considered to be more probably encephalitis lethargica, and three of the four cases were notified as such.

Such evidence, though referring mainly to cases occurring during 1921, and therefore outside the scope of this report, suggests very strongly that mistakes in diagnosis were also made in regard to cases notified as encephalitis lethargica in 1919 and 1920. Furthermore, as we have said elsewhere, a certain small proportion of such cases have undoubtedly been included in the analysis of material which forms the basis of this report, for some errors of judgment were inevitable in a scrutiny of this sort. It should be said, however, that, in these further investigations of a comparatively new syndrome, a concept wider than that which was possible or desirable in the more exclusive methods of the first inquiry has been deliberately adopted in order that the analysis should include, not only those

atypical forms that have already been described in the literature, but possibly also new clinical variants which such a large number of cases might be expected to yield. It will be seen, therefore, that the more catholic choice of cases used for the present inquiry has carried with it greater opportunities for error, but the net results of our investigations are for the most part in striking conformity with those obtained by MacNalty in the first inquiry.

The guiding signs to diagnosis as set up by MacNalty in the previous report seem so clear, and for the greater part so consonant with those suggested by our further studies, that we reproduce them here—noting afterwards where these directions seem to require addition or amendment as a result of later experience.

“In the Prodromal Period.”—An initial catarrhal affection, particularly conjunctivitis, may be suggestive. A change in the patient's mental attitude, taking the form of emotional changes, apathy, or extreme restlessness, progressive lethargy, and drowsiness indicate the possibility of the disease. If these symptoms are accompanied by headache, vertigo, asthenia, diplopia and diminished visual activity, the possibility is further strengthened.

“In the Acute Illness.”—The initial period of pyrexia, the characteristic attitude and mask-like face, the profound stupor, from which the patient can usually be aroused, and its alternation in the early stages with delirium, the asthenia, the rigidity, the emotional characteristics, the speech changes, retention of urine, tremors, early vomiting and obstinate constipation, are typical of the disease. The occasional presence of transient rashes may be helpful. Transient remission periods in the disease should be remembered. In the types presenting localising nervous signs the progressive nature of the paralysis is most important. The clearing of one paralysis and the advent of another is to be looked for, as is also the frequency of bilateral lesions, which, however, are never truly symmetrical being more marked on one side than another. The transient nature of cranial nerve paralysis, when present, is almost conclusive. The frequency of ptosis, paralysis of the ocular muscles, diplopia, facial paralysis and ocular inco-ordination are of value.

“When the rarity of bilateral facial paralysis in other forms of nervous disease and its frequent occurrence in encephalitis are remembered the presence of this sign is an additional aid to diagnosis.

“A negative sign of value is the absence of optic neuritis.”

Attention perhaps should be further drawn to the possibility of a sudden onset, which in some cases may suggest an apoplectic seizure. The later series of reports show also how frequently

localising signs of an irritative or "positive" nature may commingle with the palsies, and the occurrence of myoclonus and choreic movements are particularly significant. The absence of optic neuritis is the rule and constitutes a negative sign of value; the present series, however, supply some undoubted exceptions to the rule and some degree of neuritis in one or both eyes is probably less uncommon than is supposed.

As stated previously, the "initial catarrhal affection" was not reported very frequently in the present series of cases, nor—according to the reports again—was constipation so often noted as in the first series of cases.

Differential Diagnosis.—The three outstanding pathological conditions with which, as the present series of reports show, encephalitis lethargica is liable to be confused are *tuberculous meningitis*, *cerebral tumours*, and *cerebral hæmorrhage*, and more mistakes have been made in respect of tuberculous meningitis than of any other single condition. The character of the cerebro-spinal fluid, however, affords valuable help in the diagnosis between the two diseases, and the family history is also a point of some importance. As regards the gross cerebral lesions there is no ready means of making a distinction and reliance must be placed mainly on the history of the illness, the circumstances of the patient, the character of the localising signs and the order in which they appear; even so, however, diagnosis may be extremely difficult. Among the other maladies which have indeed suggested a mistaken diagnosis of encephalitis lethargica are the following:—

Cerebro-spinal fever.	Cerebral embolism.	Alcoholism.
Acute poliomyelitis.	„ abscess.	Nephritis.
Enteric fever	Extra-dural abscess.	Diabetes.
Influenza.	Sub-dural hæmorrhage.	Syphilis.
Pneumococcal meningitis.	Epilepsy.	Tabes dorsalis.
Septic meningitis.	Concussion.	Broncho-pneumonia.
	Paralysis agitans.	Pleurisy.

In other instances, as numbered, a diagnosis of encephalitis ultimately displaced the following mistaken diagnoses:—Cerebro-spinal fever, 4; Poliomyelitis, 7; Enteric fever, 3; Typhus, 2; Diphtheria, 1; Meningitis, 3; Cerebral tumours, 2; Cerebral hæmorrhage, 2; Hysteria, 3; Insanity, 2.

Functional disorders may cloud the diagnosis, especially at first, and it should be remembered always that such manifestations are commonly associated with the organic symptoms of encephalitis lethargica.

The importance of employing laboratory tests needs perhaps some emphasis, for the ground can be cleared considerably if the cerebro-spinal fluid, the blood, and the urine are tested appropriately; the cerebro-spinal fluid as an aid to diagnosis receives special attention in the next section (Pathology); here it may be suggested that, since cerebral syphilis can produce a clinical picture very like that of encephalitis lethargica, a

Wassermann test should be applied more frequently than has apparently been the practice.* (A reference to Widal's test will be found under "General symptoms of infection." D. 6.)

G.—PROGNOSIS.

Prognosis is not an easy matter in the case of encephalitis lethargica. The disease is undoubtedly a dangerous one as far as the individual is concerned, and probably 25 per cent. or more of all those attacked are killed, and that within a comparatively short time. If mild or abortive cases, which clear up within the space of two or three weeks, are excluded from consideration, the mortality rate among the remainder becomes much higher.

In our series, as in MacNalty's, the highest case mortality is found among patients who suffer from general nervous symptoms only, and next, in order come those cases in which "positive" localising signs are predominant. Patients, too, whose general stuporose condition is constantly being interrupted by emotional outbursts or by other signs of irritation are bad subjects, while the outlook is also ominous in the case of those who steadily subside into increasing depths of coma. Prolonged stupor, provided the patient can be nourished, is not in itself of necessarily bad omen, but caution is required before predicting that the emergence from such a state is a favourable sign. Lost sphincter control occurring late in the disease has, in the majority of cases, a serious significance, and more grave still is the inability to swallow that occasionally occurs. The reports in general show that most patients who survive the first three weeks of illness will at any rate escape death, but many are condemned to a prolonged and tiresome convalescence. The evidence available in our series shows that the number of those patients making a complete recovery is equalled by the number of those who suffer, for different periods, from various sequelæ. Recovery is slow in those whose acute illness is marked with severe asthenia, and there may be considerable delay before full power returns to muscles that have been specially affected. Patients with a strong emotional bias are likely to remain for some time a prey to psychic instability, and parents and relatives should be warned of the startling changes in the disposition that sometimes take place. It is not necessary to repeat here the various other disabilities (*see under "Sequelæ"*) that may follow an attack of encephalitis—such as insomnia, loss of memory, defective

* Tables showing the points of difference between encephalitis lethargica and many of the diseases and conditions cited above will be found in L.G.B. Public Health Report, No. 121—pp. 4, 5, 32, 33 and 37. (588 (a).) On pages 4 and 37 of the report quoted above the histological and clinical differences between encephalitis lethargica and poliomyelitis in particular are set out; further information concerning the comparative ætiology, general pathology and changes in the cerebro-spinal fluid, in the case of these two diseases, will be found in the present publication on pages 25, 137, and 159 respectively.

vision, tremors and others—but no mention has hitherto been made of sexual aberrations which, though only reported in one or two cases of our series, is not uncommonly mentioned as a sequel in foreign literature.

Considerable caution, then, is needed in making any forecast about individual cases of encephalitis lethargica, and further experience may show that the crippling capabilities of the disease, within the limits of its incidence, are not at present fully realised.

H.—TREATMENT.

The subject of treatment hardly comes within the scope of this report, since no information on the matter was asked for in the schedules of inquiry, and little or none was given. Skilled nursing and constant medical attention are needed in most instances, and 45 per cent. of the patients in 1920 received this in hospitals; treatment at home may suffice in mild cases or where other circumstances are favourable, but the ordinary household, in which the mother or wife is suddenly saddled with the duties of nurse, is no place for a helpless, semi-comatose patient who requires expert nursing, and who may at any moment need the services of catheter or feeding tube. As a general rule, then, hospital treatment is strongly advised. Among special points to be kept in mind wherever the patient is being treated are:—(1) the restlessness and delirium that frequently develop as night draws on; (2) the outbursts of excitement that may interrupt periods of stupor; (3) necessity of vigilance regarding the action of the sphincters and the prevention of bedsores; (4) possibility of a sudden dysphagia; (5) attention to the eyes.

The cases are comparatively few in which the withdrawal of cerebro-spinal fluid is strongly indicated, but *lumbar puncture* for curative reasons was occasionally performed, and with good results, notably in the diminution of stupor; in one patient the operation was undertaken on six or seven occasions, and always gave transient relief, though the case ended fatally. Brill (135) advocates the displacement of 25–35 c.c. of cerebro-spinal fluid by an equal amount of the patient's own serum under aseptic conditions (*auto-serum treatment*); while Netter in 1920 suggests, but does not recommend, "injections intra-rachidiennes de serum d'anciens malades." Subcutaneous *autohaemotherapy* is a method of treatment recommended by Bourges and Marcandier (1166); Collatino and Vegni (214) suggest that a subcutaneous injection of *convalescent's cerebro-spinal fluid* and Grunewald (399) favours intra-gluteal injection of *convalescents' serum*. Maggiore (63,) found in nine cases that an all-round improvement followed intravenous or subcutaneous injections of *sensitized vaccines prepared from young cultures of Pfeiffer's bacillus*, while Crofton and Costello were successful in some cases with a *pure influenza antigen* injected subcutaneously. In two cases Laubie (548) considers that *anti-tetanic serum* did good, and in Buenos Aires

anti-diphtheritic serum is reported by two observers to have proved efficacious.

On the strength of his own "happy results" Netter counsels the use of turpentine injections in order to form a *fixation abscess*, but this line of treatment has not been widely adopted in this country; for the same reason presumably Muller (1191) recommends intramuscular injections of iodine oil. For the control of myoclonic movements Marie and Boultier (644) find that subcutaneous injections of *cicutine* are useful; in one of the cases of our series it was necessary to use chloroform to subdue the violent contractions.

The importance of keeping the fluid intake at a high level is stressed by Dunn and Heagey (300), and Hoffman (458) thinks that in certain cases *hypertonic solutions* given intravenously are necessary. In a case reported from Dorsetshire active *purgation* was considered to have had a radical action in ameliorating the symptoms, while in another instance *leeches* applied to the head brought great relief.

As regards drugs *urotropine* seems to be most in favour and is reported to have been useful in some cases; internal evidence from the reports and our own personal investigations, however, suggest that caution is still needed in the use of the drug, and the urine should be tested daily. With a view to hastening the elimination of the virus which he considers is found in the salivary gland Netter advocated the use of *pilocarpine* or *jaborandi*, but no mention of this treatment is found in the schedule reports. Other drugs including *arsenic* and *antimony* have been used, but the mere number of treatment methods which have been briefly mentioned suffices to show that there is no specific cure and that, until more is known of the virus, all treatment must remain largely empirical and symptomatic.

I.—PROPHYLAXIS.

A limited knowledge of its aetiology makes it difficult also to say exactly how encephalitis lethargica should be prevented, but the following precautions against infection (suggested in the *Memorandum on Encephalitis Lethargica* issued by the Ministry in February 1921) represent the line of action that seems most desirable to adopt.

Precautions against Infection.—The other occupants of a house in which a case of encephalitis has occurred or is being treated, may be assured that the disease is one of low infectivity, and that very slight risk is run by association with the patient. At the same time it is desirable that such association should be limited to what is necessary for proper care and nursing, and the patient should be well isolated in a separate room.

School children in the affected household may be kept from school, as a precautionary measure; for three weeks after the isolation of the patient. There is no necessity to place restriction

on the movements of other occupants, provided they are frequently examined and remain well. Those in contact with the case, however, should be advised to use antiseptic nasal sprays or douches, and to gargle the throat with solutions such as those advised for influenza.

For example, any of the following :—

- (1) One per cent. solution of peroxide of hydrogen.
- (2) A solution of permanganate of potash, 1 in 5,000, in 0·8 per cent. solution of chloride of sodium (common salt).
- (3) Liquor sodæ chlorinatæ, 0·5 per cent.

These solutions can be used as ordinary throat gargles or snuffed up the nostrils, or applied by an efficient spray.

Any persons in the infected household who suffer from sore throat or other symptoms suggesting an abortive attack should be treated from this point of view and isolated as far as possible until they have recovered.

The sick room should be thoroughly cleansed and disinfected at the end of the illness.

As instances of case-to-case infection occurring in general hospitals are extremely rare there would seem to be no reason for suggesting that patients should not be admitted to such institutions, provided that personal precautions be taken and that the cases are nursed on a "barrier" system; where possible, a separate ward should be used to ensure as much isolation as possible. Some French authorities, too, maintain that convalescent patients should be isolated, but this contention is based on the post-mortem evidence of prolonged infection rather than on clinical proof of infectiousness in convalescent patients; in any case it is not possible to say how long a convalescent should remain in quarantine. The schedule reports provide but one doubtful instance of infection through fomites, but, in addition to the sick room, the patient's clothes might well be disinfected, while any fabrics used in the toilet of the mouth or nose should be destroyed.

ALLAN C. PARSONS.

SECTION IV.—PATHOLOGY.

AN ACCOUNT OF THE MORBID HISTOLOGY, BACTERIOLOGY AND EXPERIMENTAL PATHOLOGY OF ENCEPHALITIS LETHARGICA, WITH A CRITICAL OPINION AS TO THE RESULTS ACHIEVED.

By A. SALUSBURY MACNALT, M.D.

1.—*Historical Introduction.*—Although it is possible and even by some medical historians considered more than probable that outbreaks of encephalitis lethargica occurred prior to the winter of 1916–17, it is indisputable that the pathological study of the disease begins at this date, when a remarkable outbreak occurred in Vienna.

Von Economo described in detail the clinical symptoms of thirteen cases presenting the cardinal phenomena of stupor, accompanied by disturbances of the ocular muscles, while von Wiesner (1083) made himself responsible for the pathological and experimental work. Of the thirteen cases, five died, and were examined both macroscopically and microscopically after autopsy. The histological lesions observed were so uniform as to suggest a common etiology for the whole outbreak. A Gram-positive coccus recovered from all fatal cases was held to be the agent of the specific infection. The disease was conveyed to apes and from an infected ape a pure culture of a diplo-streptococcus was obtained by von Wiesner. Von Economo gave to the outbreak the name of Encephalitis Lethargica, as descriptive of its salient clinical and anatomical features. Encephalitis Lethargica next made its appearance in France, where the outbreak was studied by Netter (731), who recorded histological findings identical with those of von Wiesner.

In the course of the months March to April 1918 a certain number of cases of an obscure disease, characterised usually by stupor or lethargy and ophthalmoplegia, were reported in England. Early attention was directed to the subject by Harris (429) and by Hall (419). A likeness to the clinical descriptions of "Botulism" was observed in these cases and the suggestion was hazarded that they were due to the consumption of food infected with the *Bacillus botulinus* of Van Ermengem. A detailed investigation of the outbreak was made under the auspices of the Ministry of Health, then the Local Government Board, and the Medical Research Council, with the following results :—In the first place, MacIntosh (621) failed entirely to recover *botulinus* from the tissues and fluids of fatal cases and from articles of food which had been regarded as suspect; he failed also to obtain evidence that serum from recovered patients agglutinated this bacillus or protected mice from the effects of this toxin; and he found microscopical evidence of changes in the nervous system which indicated an acute infective disease, rather than an intoxication. The epidemiological enquiries were in accord with these findings and consequently Botulism was ruled out of court as responsible for the outbreak.

The histological researches on certain of the fatal cases were carried out by Marinesco (648) and MacIntosh, who recorded their results in separate contributions to the report of the Local Government Board.

They found lesions in the brain indicative of an inflammatory process resembling that observed in general paralysis, sleeping sickness and acute poliomyelitis. The lesions, however, displayed certain distinctive features and could be differentiated from those which they resembled, as, for example, the lesion of acute poliomyelitis.

In addition to the histological examination of some twenty cases, MacIntosh carried out complete bacteriological examinations

of material obtained from a number of cases during life and *post-mortem*. Cultures from the blood were invariably sterile, and cultures from the cerebrospinal fluid, nasopharynx, and faeces failed to show the constant presence of any particular class or type of micro-organism (aerobic or anaerobic). Material from eight fatal cases of the disease was inoculated into *Macacus Rhesus* monkeys by MacIntosh. On this occasion he did not succeed in transmitting the disease to these animals.

As a result of their separate researches both Marinesco and MacIntosh arrived independently at the conclusion that encephalitis lethargica, as it appeared in the outbreak, was identical with the illness described by von Economo in Austria and Netter in France, and that it is a disease anatomically and clinically distinct from analogous affections.

A similar conclusion concerning the novel and distinctive character of the disease was arrived at independently by James (485) and MacNalty, who approached the problem by pursuing different lines of enquiry, the former undertaking an epidemiological investigation, the latter a clinical study of the cases reported in England and Wales. Kinnier Wilson (1,091) and Buzzard (157) also early adduced evidence supporting the above conclusion. Cleland and Campbell (1,126) described an epidemic of a form of Encephalomyelitis which occurred in the late summers of 1917 and 1918, and was restricted almost entirely to certain dry inland parts of New South Wales. By some writers this epidemic is confused with encephalitis lethargica, but microscopical examination of the nervous system in the cases showed a generalized acute Encephalomyelitis and the condition appears to be quite distinct from the disease under review.

In the autumn of 1918 cases of Encephalitis Lethargica were reported from the United States, and Bassoe has published accounts showing that the usual pathological picture of the disease was revealed by a study of the cerebral lesions.

The more recent researches upon the pathology of encephalitis lethargica have been carried out by MacIntosh and Turnbull (622), Greenfield (392) and Da Fano (251) in this country, in France by Levaditi and Harvier (571), and in the United States by Loewe, Hirshfeld and Strauss (592, 593), Thalheimer (1,016) and Amoss (27).

2. *Certain Anatomical and Physiological considerations bearing upon the Pathology of the Disease.*—The phenomenon of stupor is produced by a central inhibition of afferent stimuli to the sensorium such as is already recognised in certain forms of cerebral disease. It is known that all the afferent stimuli from the environment stream into the Optic Thalamus, thence pass to the neo-pallium or cerebral cortex and to the essential organ of the thalamus (*vide* Head and Holmes. Brain, 1912, XXXIV, p. 102).

In taking this route, in the main, they pass upwards through the crus cerebri in near proximity to the nucleus of the IIIrd nerve,

Furthermore, although the chief course of the IIIrd nerve fibres in the mesencephalon is given as passing out by way of the Red Nucleus, yet it is established that certain of the fibres have a more upward curve; in so doing they come into close relation with the fibres conveying afferent stimuli to the thalamic region. Hence it follows that any illness affecting this anatomical localisation of afferent paths is associated with stupor, for it involves a blockage of the afferent stimuli.

Pathological evidence had already proved that the lesions found in fatal cases of encephalitis lethargica preponderated in sections of the mesencephalon made through the region of the nucleus of the IIIrd nerve, while one of the principal features of the disease, as its name implies, is the progressive lethargy, going on perhaps to stupor or complete coma. There is thus ample evidence indicated by local signs that, in a large number of cases of the disease at all events, there is interference in or near the region of anatomical localisation of afferent paths. The usual absence of optic neuritis, indicating that the lethargy or stupor is not part of a general cerebral condition, such as would be caused by hypertension, as in a cerebral tumour or abscess, is supplementary evidence on this point.

A lesion in or near the nucleus of the IIIrd nerve causes some ophthalmoplegia; unless extremely selective and restricted in action, it will also, by affecting the afferent paths, result in stupor, for the nucleus of the IIIrd nerve and certain of its emergent fibres bear intimate relation to the afferent fibres streaming towards the thalamic region, neo-pallium, &c. The association of stupor and ophthalmoplegia is thus explained. If, however, the chief stress of the disease falls outside the IIIrd nucleus and its emerging fibres, or if the disease terminates too quickly to allow of involvement of the IIIrd nerve being apparent, in either case there will be interference with the afferent paths conveying environmental stimuli, and the phenomena of stupor without localising signs will obtain. This pathological distribution will account for two groups of the disease recognised clinically, namely, cases with general symptoms and without localising signs and cases with localising signs in the central nervous system. In addition, the explanation is adequate for the group of cases with mild or transient manifestations. The anatomical localisation is common for all three groups, but in the last-mentioned one the intensity of the interference is diminished.

The anatomical relationship of the paths of the rubro-spinal and the pyramidal tracts to the nuclei of the IIIrd nerve explains the tremors (affection of the rubro-spinal fibres) and the presence of Babinski's sign seen in certain cases of encephalitis lethargica.

In the group of cases of the disease with nervous localising signs clinical evidence shows that certain types occur, displaying, as a rule, stupor, but with no ophthalmoplegia. Paralytic phenomena are exhibited, one type being associated with local lesions of cranial nerves other than the IIIrd, the VIIth nerve being notably affected, and another type with spinal manifestations.

Pathological evidence furnishes similar proofs to the effect that although lesions in or about the nuclei of the IIIrd nerve preponderate, yet, frequently, they are not limited to this region, but may involve or be primary in the pons, bulb, the cerebellum (Marinesco), the cerebral cortex as described by Buzzard and Greenfield (158) and by Watson (1,069), and, rarely, the spinal cord.

In the case of a woman of 49, who presented in life general symptoms, together with facial paralysis, Spilsbury (588a., App. p. 72, Case 6) found the characteristic lesions of encephalitis lethargica in the pons and bulb. Such cases may in reality have a slight affection of the mesencephalon, which is clinically non-apparent, or they may have no mesencephalic lesion.

A series of polyneuritic cases was clinically described in the outbreak of 1918. In the absence of pathological information it is uncertain whether such cases are anatomically best explained as of mesencephalic origin with a secondary toxic effect upon the peripheral nerves or whether a primary or concomitant spinal lesion were present.

From what has been said, it appears that the anatomical situation of the lesion of encephalitis lethargica is usually mesencephalic and in or near the nucleus of the IIIrd nerve. But the situation of the preponderating lesion may be in any part of the brain-stem and bulb, and lesions may also be found in the cerebellum, the cerebral cortex, and even in the spinal cord, so that to complete the histological study of a case of encephalitis lethargica it is desirable to make a complete investigation of the brain and spinal cord.

3. Post-Mortem Findings.—The *post-mortem* examinations carried out at the Pathological Institute of the Vienna University revealed no noticeable changes in the internal organs, except for purely degenerative lesions of the heart-muscle, as well as of the parenchyma of the liver and kidneys. MacIntosh, who analysed a number of post-mortem examination reports in the English outbreak of 1918, found no macroscopic changes present sufficient to account for death. He observed that many of the cases showed some congestion of the brain, increase of cerebro-spinal fluid, with a dilatation of the vessels of the pons and the thalamic region; no actual hæmorrhages into the brain-substance were seen, but two superficial hæmorrhages were recorded; one, a small one on the cortical surface and the other of some considerable size, in the cerebellar region. In one or two cases the grey matter was more prominent than usual, while in others again it was yellowish in colour. In a few cases there was slight broncho-pneumonia which was probably a terminal affection.

Buzzard would recognize a group of cases in which the encephalitis has its chief incidence on the cerebral hemispheres, and in which hæmorrhage is a conspicuous feature. He states that in studying the morbid anatomical features of the disease in several fatal cases he has been struck by the fact that the brunt

of the inflammation falls upon the vessels, with the result that arterial and capillary hæmorrhages and venous thromboses are of very frequent occurrence.

4. Morbid Histology.—The morbid histology of encephalitis lethargica has been carefully studied by von Wiesner, Netter, MacIntosh and Marinesco. Their findings harmonise with one another, and as a result the main histological picture of the disease can be regarded as clearly delineated.

The lesion is definitely inflammatory and the changes consist of a cellular infiltration of the perivascular lymphatic sheaths and of certain areas of grey matter (MacIntosh).

There is no marked change in the meninges; insignificant increases of the cellular elements of the pia-arachnoid, particularly in the neighbourhood of the blood-vessels, have been observed. In three cases of encephalitis lethargica examined by Bassoe and Hassin (60) there was a mild leptomeningitis, the vessels of the pia being congested with perivascular infiltration with plasma cells and leucocytes.

In the brain substance, as already mentioned, the changes are most conspicuous in the upper part of the pons and in the basal nuclei. In the spinal cord the lesions are very slight or entirely absent. The lesion in the grey matter is focal in character; it consists of what von Wiesner terms a "colossal" small-celled infiltration of the perivascular lymphatic sheaths and of the parenchyma. Marinesco points out that the endothelium and fibro-blasts may also have taken part in the inflammatory process. The parenchyma may be cedematous; the extent of infiltration here varies considerably, but it is usually proportional to the perivascular lesion. According to MacIntosh, the cellular exudate is composed chiefly of large and small mononuclear lymphocytes, along with a certain number of the polyblasts of Maximow and a few plasma cells. In most of the cases the small or medium sized veins were most affected. MacIntosh states that polynuclear leucocytes of any variety are noticeably absent; von Wiesner, in his cases, detected "nests" or small groupings of polynuclear leucocytes in isolated spots of the parenchyma. The extra-adventitial infiltration is usually diffuse, but bearing a relation to the perivascular infiltration is largely due to emigrated cells in MacIntosh's opinion. At the same time proliferation of the neuroglial cells undoubtedly occurs. In certain areas, particularly in the basal nuclei and upper parts of the pons, MacIntosh found the infiltration might be so dense as to form actual foci; such foci resemble those seen in the grey matter of the cord in poliomyelitis, but one striking difference between the two forms of disease is that in the cases examined in the 1918 epidemic the ganglion cells were still present in considerable numbers. Marinesco describes foci of interstitial inflammation consisting of neuroglia cells of various kinds, including large cells with voluminous eccentric nuclei and many fibrillar prolongations, lymphocytes and polynuclears. Such foci have

been noticed by von Wiesner and Marinesco as sometimes occurring in the roots of the cranial nerves, *e.g.*, hypoglossal, pneumogastric. Marinesco considers that the interstitial changes may be independent sometimes of vascular infiltrations and Bassoe and Hassin have come to a similar conclusion. Both von Wiesner and MacIntosh note that there is no special involvement of the ganglion cells which, as a rule, are well-defined with the Nissl granules present. In the areas of nervous tissue most affected some of the ganglion cells may be globular in shape with the chromatine diffuse and some mononuclear cells in close proximity, but neuronophagia is extremely rare and when seen is never marked.

Marinesco described lesions of the nerve-cells which do not correspond with those usually seen in anterior poliomyelitis. He found dissolution of the Nissl granules, relative achromatosis, reduction in volume of the cellular body and of the number of prolongations and multiplication of the satellite cells. He agrees with von Wiesner and MacIntosh as to the rarity of neuronophagia. In one case he had the opportunity of examining the cerebellum. The small vessels and capillaries of the cerebellar cortex showed a somewhat intense hyperæmia, but there was almost complete absence of inflammatory infiltration of the vascular sheath. In contrast to this the cells of Purkinje were profoundly affected. No cells were normal; the Nissl bodies had disappeared from all; the cytoplasm was pale, slightly granular, or most usually homogenous. The lesion was not limited to the chromatophilic substance but involved also the neuro-fibrillæ of Purkinje's cells which had sometimes completely disappeared or were fragmented or reduced to granules. The pericellular plexus was well preserved. Golgi's cells were little affected. Marinesco concludes: "the lesion of the cells of Purkinje was a primary one; it did not depend on lesions of its axis cylinders traversing the pons. At all events, the type of alteration shown by these cells is that which I have designated by the name of '*lésions primitives*.'"

In a preliminary communication Da Fano and Ingleby (252) confirm the histological observations of previous workers but lay stress on alterations in the nerve cells. Their cases of encephalitis lethargica showed changes more severe and widespread than those described hitherto. They ranged from slight chromolysis to complete disappearance of Nissl granules, atrophy of the protoplasm and shrinkage, atrophy and final disappearance of the nucleus. They also direct attention to the presence of a granular black, pigment-like, material occurring within the nerve-cells where black or brown pigment is not generally found. In a further communication Da Fano (251) reports that within and without the nerve cells minute forms have been observed, to all appearance consisting of a central generally basophil particle and of a delicate little stainable body, irregularly round or oval in shape. These he terms "minute bodies." They are

generally discrete and provided with one granule, but dumb-bell-shaped forms occur, as well as others with two central particles arranged in pairs. A relation, as yet not defined, appears to exist between these forms and the granular pigment like material. Similar "minute" bodies have been traced within and without the cells infiltrating a salivary gland from an acute case of encephalitis lethargica.

After discussing the supposition that such findings might be associated with degenerative changes caused by the malady, Da Fano tentatively puts forward the suggestion that they may be due to the presence in the tissues of a living agent, the cause of the disease, on the analogue of the Negri bodies in rabies.

In this connection one may note that Levaditi, Harvier and Nicolau (527a) have found in rabbits which have died of encephalitis lethargica, produced by cerebral inoculation, corpuscles in the cerebral tissues which they describe as similar to Negri bodies.

According to von Wiesner and to MacIntosh, hæmorrhages are usually absent in the sections examined. Netter in his series of cases, however, found that the grey and white matter might be the seat of numerous hæmorrhages ("*piqueté hæmorrhagique*"). In two brains examined by Marinesco hæmorrhagic foci were numerous; nevertheless the vessel wall did not appear to be necrosed, but sometimes a solution of continuity of the vessel wall could be seen.

Netter states that the salivary glands in cases of encephalitis lethargica may be affected. (1,196b.) Netter and Durand (734) have examined systematically the parotid and submaxillary glands from fatal cases of the disease, and find that round-celled infiltrations are very numerous in histological sections of the glands.

Such is the outline of the characteristic morbid histology of encephalitis lethargica. The descriptions given by the different investigators have been based upon the examination of well-marked cases of the disease and the stages in the development of the lesions require yet to be worked out.

Perhaps the tentative suggestion may be made that the inflammatory lesion is somewhat slow of manifestation. In Lereboullet and Hutinel's case (564), which proved fatal after a week's illness, the histological findings were almost completely negative, although the clinical symptoms were typical; in a case of encephalitis lethargica without localising signs which proved fatal within six days, reported by MacNalty (629c. Case I), Graves noted only a few areas of round-celled infiltration in the basal ganglia with marked distension of the small cerebral vessels suggesting an early stage of the disease; and in four of MacNalty's acute cases from the Derby outbreak proved experimentally to be one of encephalitis lethargica, MacIntosh and Turnbull (622) and Da Fano and Ingleby (252) reported negative findings as regards microscopical examination of the cerebral

tissue. All four cases proved fatal within a period of 3 to 10 days.

5. *Bacteriological Findings*.—Examination of material from cases of encephalitis lethargica during life has been undertaken by von Wiesner (1,083) and by MacIntosh (621).

The examination of the cerebro-spinal fluid showed, as a rule, no specific changes. The Nonne-Appelt test was negative; total protein was below the upper normal limit given as 2.2 by Nissl; the goldsol test showed no typical alteration of the curve. The cell-content remained at the upper normal value in the majority of cases (von Wiesner). The cerebro-spinal fluid was in some of the cases under a slightly increased pressure and in certain of the cases there was a slight increase in the number of lymphocytes (MacIntosh). No organisms could be found in any of the specimens and the Wassermann reaction, both in the fluid and the serum of the patients, were negative in every case. The blood taken by veni-puncture was in each case sterile. Cultures of the naso-pharynx failed to demonstrate the constant presence of any particular micro-organism. In one case with a slight nasal discharge, a coccus, apparently identical with that of Rosenow's, was isolated by MacIntosh. Samples of faeces gave nothing pointing to a definite conclusion. MacIntosh also reported that all findings were negative as regards the possibility of the bacillus of botulinus having anything to do with "the new disease."

From all fatal cases examined by him von Wiesner recovered a gram-positive organism which he termed a *Diplo-Streptococcus*. He described these organisms as pleomorphic, isolated cocci assuming both in culture and the tissues the shape of elongated, oval or short thick bacilli. They tend to degenerate in the tissues, and globoid bodies, unevenly stained but retaining some of the "Gram," are seen in abundance. In fluid cultures they readily grow in chains. The cocci are found in sections in the leptomeninges, particularly in the celled, infiltrated arachnoid meshes but they are scanty, difficult to discover and could not be found in the inflammatory foci in the brain. The isolation is difficult and the organism grows best anaerobically. It can be cultivated on glucose agar. Up to the present, the work of subsequent investigators has not confirmed von Wiesner's findings, the prevailing impression being that he was dealing with a contamination.

Working at the Mount Sinai Hospital, New York, Strauss, Hirshfeld and Loewe (1,000) and later Loewe, Hirshfeld and Strauss (593), have obtained a filtrable virus from the naso-pharyngeal membrane of fatal cases of encephalitis lethargica which they regard as the specific agent of the disease. The virus has been passed in series through monkeys and rabbits. It can be preserved for many months in 50 per cent. glycerol. In a later communication Loewe and Strauss (592) claim that by means of the ascitic-tissue culture methods perfected by Noguchi, they

have been able to cultivate a minute, filtrable organism from cases of encephalitis lethargica; this organism is present in the brain, naso-pharyngeal mucous membrane, naso-pharyngeal washings, spinal fluid and blood. A successful growth is usually manifested on the fifth to seventh day by clouding of the medium. Transfer to solid media can be obtained only with the later generations of the organism. Studies of the fluid cultures under dark field illumination reveal the organisms as minute globular refractile forms, occurring singly, doubly or in chains and clumps, the latter form predominating, especially in the older cultures. These bodies show active Brownian movement, but no true motility. The organism has an average diameter of 0.25 mikrons. The curious statement is made that the organism may be Gram-positive or Gram-negative; it stains by Giemsa and by methylene blue. Thalheimer (1,016) has confirmed the work of Loewe and Strauss, but otherwise it is at variance with the results of English and French bacteriologists. Kling and Liljenquist (513) in Sweden claim to have found the virus of encephalitis lethargica in the nasopharyngeal secretions and in the intestinal contents.

MacIntosh (621) in 1919 showed by his experimental results that the disease was caused by a living virus, in all probability a filtrable virus.

Levaditi and Harvier (571 b. and e.), from the cerebral and spinal nervous tissue of rabbits inoculated with encephalitis lethargica from human cases of the disease occurring in the Paris outbreak of 1919-20, have isolated a filtrable virus which can pass wholly or partially through Chamberland filters Nos. 1 and 3. This causal virus can be preserved for some length of time in glycerine, is destroyed at 56° C., is killed by prolonged contact with phenol and preserves its virulence for at least forty-eight hours after death. In spite of numerous attempts they have not been able to cultivate the organism on Noguchi's medium, but they have succeeded in preserving its virulence at 37° C. in association with cells of the testicle of the rabbit cultivated *in vitro*.

Attenuated varieties of the organism exist.

In a communication of recent date, Levaditi, Harvier and Nicolau (572) give some further particulars relative to their experiments with the virus:

(1) They find that the serum of human subjects convalescent from encephalitis lethargica, although it only rarely and partially neutralises the virus *in vitro*, yet may be shown to possess specific qualities. Two volumes of serum (in one instance convalescent serum and in the other normal human serum) are mixed with one volume of virus and kept in contact for five hours at 37° C. The rabbit inoculated on the cornea with the mixture of virus and convalescent serum does not exhibit keratitis and survives, while the rabbit inoculated with the mixture of virus and normal serum develops keratitis and dies

of encephalitis. This may lead to the application of a "kerato diagnostic" method. The serum of a sheep which has been given subcutaneous injections of virulent emulsions of the brains of rabbits dead from encephalitis lethargica is able to neutralise the virus in vitro. It gives a positive Bordet-Gengou reaction with the infected brain which is much more intense than with the normal brain. Levaditi, Harvier and Nicolau anticipate that this serum will enable them to differentiate the virus of encephalitis lethargica from other similar viruses such as rabies and poliomyelitis.

(2) They find that anaesthetics, particularly chloroform, ether and chloral, have a manifest and appreciable effect in increasing the pathogenicity of the virus inoculated intracerebrally.

(3) They have established the following new properties of the virus: (a) Prolonged and intense centrifugalisation of a cerebral emulsion of the virus does not deprive the supernatant liquid of its pathogenic power.

(b) The virus emulsion is still active for intra-cerebral inoculation after dilution up to 1/1000 in physiological saline.

(c) The virus dried *in vacuo* is still pathogenic after preservation for forty days at room temperature. Mixed with milk, it still remains virulent after forty days' preservation under the same conditions of temperature.

(d) A fragment of virulent brain placed in glycerine does not contaminate the glycerine medium or pieces of normal brain or normal kidney with which it is placed in contact. This experiment joined to others made with solid gelatine covered with virus emulsion proves that the virus is not diffusible.

6. *Experimental Investigation*.—In 1917, von Wiesner injected a *Macacus Rhesus* subdurally with 0.2 c.c. of an emulsion of the brain and the cord of one of the fatal cases of encephalitis lethargica. "A few hours after the injection this animal hitherto lively and running about the cage, became decidedly quiet and appeared tired. The next morning it lay crouched in a corner of the cage, was somnolent, with eyes almost closed but reacted to being spoken to. It ate, but had difficulty in chewing. Left to itself, it passed again into slumber. On being repeatedly interfered with, it kept on waking up, making an impression of a person "struggling with sleep." No stiffness of the neck, slight paresis of the left posterior limb, painful gait, tendon reflexes present, pupils reacted sluggishly. In the afternoon the gait became ataxic, the pharynx paralysed, the somnolence more marked. The following morning the animal was lying completely apathetic on the floor of the cage, would not react, and finally died, forty-six hours after injection."

Extensive acute hæmorrhagic encephalitis was found *post-mortem*, though such a feature was not displayed even microscopically in the brain used for the injection. A Berkefeld filtrate of an emulsion of this monkey's brain when injected into another *Macacus* proved innocuous. Von Wiesner recovered his

diplo-streptococcus from the brain of the lethargic monkey. The pure culture of this organism injected into a *Cereopithecus sabæus* caused mild somnolence and muscular weakness in the animal. The ape was killed on the twelfth day and the only lesion found consisted of some hæmorrhages in the medulla. Von Wiesner's experiments were not carried out on an extensive scale, owing probably to the difficulty experienced at that time in procuring monkeys, and considerable doubt remains as to the conclusions to be drawn from this early experiment.

When studying the outbreak of encephalitis lethargica in 1918, McIntosh inoculated material from eight of the fatal cases into *Macacus Rhesus* monkeys. The inoculum consisted of emulsions of pieces of the basal nuclei, pons, medulla and cervical cord, and in one case pieces of the cord were used alone. The material used was preserved for three or four days before the inoculation in 50 per cent. glycerine; this strength of glycerine is known not to affect the virus of poliomyelitis. The inoculations were all done intracerebrally, while an intraperitoneal injection was given simultaneously. McIntosh reports that in no case did any definite paralysis develop, even after an interval which could be regarded as the longest average incubation period. One or two of the monkeys, however, some ten days after the inoculation, showed some slight excitement and tremblings. This was only noticed for a day or two and then passed off, the animals regaining their usual vigour. The relative insusceptibility of *Macacus Rhesus* monkeys to the virus of encephalitis lethargica as compared with that of poliomyelitis was thus clearly shown.

Then in 1919 Strauss, Hirschfeld and Loewe produced an acute encephalitis in monkeys by intracerebral inoculations of material derived from cases of encephalitis lethargica. The type of lesion observed, and the short incubation period, suggest that in all probability these workers were dealing with another infection. Later they successfully transmitted the disease to rabbits and to monkeys.

In 1919 McIntosh succeeded in transmitting encephalitis lethargica to a *Patas* monkey. In August of that year an institutional epidemic of encephalitis lethargica occurred in a home for girls at Derby (629e). Out of 21 inmates of the home there were 12 cases with five deaths. A *Patas* monkey, inoculated by McIntosh with material from one of the fatal cases, presented lethargic symptoms and tremors, and died. On examination, Turnbull (622) found that the monkey's brain showed lesions similar to those found in human cases of encephalitis lethargica. The monkey was inoculated intracerebrally and intraperitoneally. Pieces of the cervical cord, pons and basar nuclei, each about the size of a large pea, were placed in a conical glass vessel, and, after the excess of glycerine had been washed off with sterile saline, were pounded up by means of a glass rod. The pounded material was then emulsified in 20 c.c. of sterile saline. About 10 c.c. of this emulsion was diluted with saline until it could be drawn through a Berkefeld filter with the aid of an exhaust pump.

The *Patas* monkey was injected with the filtered emulsion. Subsequently McIntosh and Turnbull (622) have reported the successful transmission in series of experimental encephalitis lethargica to monkeys and rabbits, which has completed the evidence necessary to show that the disease is caused by a living virus. Additional proof was provided by the spontaneous infection with encephalitis lethargica of a *Macacus cynomolgus* monkey kept as a control along with an inoculated monkey.

As McIntosh and Turnbull pointed out, this was the first indisputable occasion upon which experimental transmission of encephalitis lethargica to an animal was successful, and the experiments indicate therefore a conspicuous advance in our knowledge of the disease.

Commencing experimental researches in December 1919, Levaditi and Harvier (571a) for some time essayed without success to transmit encephalitis lethargica to monkeys and rabbits. In February 1920 they succeeded by using an emulsion of cerebral and spinal tissue, obtained from a fatal case of the disease, in transmitting the malady to a rabbit. As already mentioned, by this experiment they obtained a specific filtrable virus which they regard as the causal agent of the disease, thus confirming the experimental results of McIntosh.

Levaditi and Harvier state that this virus is pathogenic for the rabbit and guinea-pig, but it is either slightly virulent or non-virulent for the monkey. The lesion which this virus produces in the affected animals, and also the symptoms of the experimental disease are practically identical with those in the human subject. They find that encephalitis lethargica may be transmitted to the rabbit intracranially, through the orbit, and by inoculation of the virus into a peripheral nerve, such as the sciatic. The virus is harmless when injected subcutaneously, intravenously, into the peritoneum, into the trachea, into the alimentary canal or into the parenchyma of the salivary gland; if injected into the testicle, however, it produces encephalitis lethargica in the experimental animal, and retains its virulence in the testicle for at least 17 days. Levaditi and Harvier further find that the intact nasal mucous membrane offers a barrier to the penetration of the organism, and that this protection is only overcome when the nasal mucous membrane is injured and inflamed. The organism follows then, probably, the ascending path of the olfactory nerve in order to reach the brain. The virus exists in the cerebrum and in the spinal cord of the subjects of encephalitis lethargica, but is absent from the blood, cerebrospinal fluid and the organs of the body, including the salivary gland.

Netter (1196b), from his histological observations, considered that the virus was probably present in the salivary glands, and that the saliva was the chief source of infection. Levaditi and Harvier consider that the chief route of infection is by way of the naso-pharynx. In addition to the above important findings they have also conducted certain immunological experi-

ments. They find that there is no spontaneous cure in the experimentally-produced malady, and consequently no succeeding refractory stage; nevertheless, immunity can be artificially established by living or dead vaccines; it is not an absolute immunity, but is manifest to a sufficient extent to permit of further researches into the subject. The serum of vaccinated animals as a rule is neither endowed with microbicidal properties *in vitro*, nor can it act prophylactically. The same usually holds true of the sera of persons convalescent from encephalitis lethargica, according to Levaditi and Harvier's studies. This lack of protective value in the sera of these patients has been confirmed by Amoss (27). In his tests he used the blood-serum of four cases, one serum being taken from a patient convalescent in the fifth week of the disease, the second in the fourth month, the third in the fifth month, and the fourth from a patient 15 months after the attack. He compared his results with controls, and with the effect of sera from poliomyelitis patients which protected against inoculation with the virus of poliomyelitis.

The following table summarises the important pathological points of differentiation between encephalitis lethargica and acute poliomyelitis (Heine-Medin Disease).

Differentiation between Encephalitis Lethargica and Acute Poliomyelitis.

	Acute poliomyelitis.	Encephalitis Lethargica.
<i>Histology.</i> Distribution of the lesions.	They are most extensive in the spinal cord (affecting the anterior cornua), and become progressively less so upwards to the brain. The pia arachnoid is frequently involved.	They are most extensive in the basal nuclei of the brain; nearly equally so in the upper part of the pons; much less extensive in the medulla; absent or very slight in the spinal cord. The pia arachnoid is only slightly involved.
Character of the infiltration.	Plasma-cells less frequent; polynuclear leucocytes more frequent. Hæmorrhages common and obvious, making the grey matter of the cord pink.	Plasma-cells numerous; polynuclear leucocytes rare. Hæmorrhages not common and microscopic.
Changes in the nerve-cells.	Degenerative nerve-cell lesions, pronounced (neuronolysis). Motor cells surrounded and infiltrated with partially phagocytic round-cells and finally destroyed completely, leaving in their place a heap of round-cells (neuronophagia).	Perivascular infiltration is the dominant lesion. Degenerative lesions are less pronounced. As a rule, the ganglion-cells are not much affected; they remain present in considerable numbers, are usually well-defined and with the Nissl granules present; Neuronophagia is very rare and when present is not marked.

	Acute poliomyelitis.	Encephalitis Lethargica.
Bacteriology.		
Virus	A filtrable virus readily passing through Chamberland, Berkefeld, Reichel or Pukall filters (Flexner, Lewis, Leiner and Wiesner, Levaditi and Landsteiner). Resistant to freezing and to glycerine. Dried in vacuo (away from light) at room temperature it is potent for four weeks (Romer and Joseph). Heating for 30 mins. at 45°-55° C. destroys it. Appears to be destroyed by potassium permanganate, by hydrogen peroxide, by formaldehyde, by 1 per cent. thymol and by three days' contact with 0.5 per cent. phenol.	A filtrable virus passing wholly or partially through Chamberland filters (McIntosh, Levaditi and Harvier), can be preserved for some length of time in glycerine. Dried in vacuo at room temperature it is potent for forty days (Levaditi, Harvier and Nicolau). Destroyed at 56° C.; killed by prolonged contact with phenol. Preserves its virulence for at least forty-eight hours after death. Virulence preserved at 37° C. in association with cells of the testicle of the rabbit cultivated <i>in vitro</i> (Levaditi and Harvier). It is not diffusible.
Localisation of virus	In brain, spinal cord, cerebro-spinal fluid, mesenteric glands, tonsils, in mucous secretions of naso-pharynx, trachea and intestines. Also in dust of rooms.	Exists in brain and spinal cord. Absent from blood, cerebro-spinal fluid and organs of the body (McIntosh, Levaditi and Harvier).
Transmission to animals.	Virus readily transmissible to monkeys. Not easily transmissible to rabbits.	Virus transmissible with difficulty and only from acute cases of the disease. Pathogenic for rabbit and guinea-pig; slightly virulent or non-virulent for the monkey (Levaditi and Harvier), but virulent for certain species of monkeys (McIntosh).
Immunity		
Active	Early appearance of immunity after attack of poliomyelitis. Animals immunised by gradually increasing doses of fully virulent cord (Flexner and Lewis). Monkeys actively immunised by a mixture of virus and immune serum (Landsteiner and Levaditi and Romer and Joseph).	No spontaneous cure in the experimentally produced malady (Levaditi and Harvier).
Passive immunity.	Serum of animal immunised against poliomyelitis possesses capacity of inactivating the virus <i>in vitro</i> (Levaditi and Landsteiner, Romer and Joseph, Flexner and Lewis). Immune serum protects animals	Serum of animals vaccinated with the virus is, as a rule, neither microbicidal <i>in vitro</i> , nor can it act prophylactically. This is true also usually of sera of convalescent patients (Levaditi and Harvier).

	Acute poliomyelitis.	Encephalitis Lethargica.
Bacteriology.		
Passive immunity—cont.	against inoculation with virus, serum of convalescents protects against poliomyelitis.	
Cross-immunity experiments (Amoss).	Serum of subjects of poliomyelitis does not protect against encephalitis lethargica.	Serum of subjects of encephalitis lethargica does not protect against poliomyelitis.

7. *Conclusion.*—The claims of encephalitis lethargica to be regarded as an independent disease, with a lesion hitherto undescribed by pathologists, appear to be sufficiently well established. To this statement there is perhaps one exception. It is possible that paralysis agitans, or at all events certain cases of the disease, are subsequent or chronic manifestations of an unrecognised attack of encephalitis lethargica. The site of election of the lesion is in some degree similar in the two diseases; the clinical symptoms of encephalitis lethargica, for example, the mask-like face, resemble Parkinson's disease; more convincing still, symptomatic paralysis agitans has been described by S. A. Kinnier Wilson (1,091b), Crouzon (242), and others as following on encephalitis lethargica, although Pierre Marie and Mlle. G. Lévy (645) consider chronic paralysis agitans should not be identified with such symptomatic cases.

The histological researches of Marinesco and McIntosh excluded identification of the "new disease" with "Botulism," and then the assertion was made that the disease was an example of acute poliomyelitis or Heine-Medin disease in which the stress of the lesion fell upon the cerebral nervous tissue. Much ink was poured out in this controversy into which it is now unnecessary to enter upon in detail, involving as it does the consideration of epidemiological and clinical as well as pathological findings. The whole question will be found fully discussed in the Special Report of the Local Government Board, issued in 1918. Pathological differences between the two diseases have already been summarised in the Table on pages 137-139, and it is now clear that in spite of resemblance with poliomyelitis, the two affections are quite distinct. It is also probable that many morbid conditions unconnected with the true disease have been classed as "poliomyelitis," and that certain cerebral or bulbar types previously reported as "poliomyelitis" were in reality cases of encephalitis lethargica. Certain writers would urge that encephalitis lethargica is an example of the encephalitis of influenza. In the latter malady, however, the anatomical features are invariably those of an acute hæmorrhagic encephalitis. Pfeiffer's bacillus, not infrequently isolated from cases of influenzal encephalitis, has not been found in cases of encephalitis lethargica.

From the epidemiological and clinical standpoints, this question has been carefully investigated and no reliable evidence is forthcoming in favour of the identity of influenza and encephalitis lethargica.

The histological findings in encephalitis lethargica are indicative of a characteristic inflammatory lesion; it is distinct, for example, from the polioencephalitis of Wernicke, which is of toxic origin. The bacteriological and experimental results indicate that the causal organism is a filtrable virus present in the tissues of the central nervous system which can be experimentally transmitted in series to monkeys and rabbits. The lesions, as Mott (719) and Marinesco (648a) point out, present a striking similarity to those of the encephalitis of African sleeping-sickness due to trypanosome infection. Changes of an inflammatory nature in the grey matter of the brain are produced, and thus encephalitis lethargica is comparable to the infections of syphilis, sleeping sickness, rabies and poliomyelitis. Although comparable to these maladies in its pathological *modus operandi*, the weight of evidence has demonstrated the independent character of the disease.

Since the above Report was written Levaditi, Harvier and Nicolau (572c) have published papers indicating that their experimental researches into encephalitis lethargica have been conducted several stages further. It is only possible here to note briefly the conclusions of these Authors. For the details of the experimental studies and the technique adopted the original articles should be consulted.

1. Sources of the virus.—The virus of encephalitis can be detected in the naso-pharyngeal secretions of patients suffering from the disease by inoculating these secretions by scarification of the cornea of a rabbit. Encephalitis lethargica is contracted by the rabbit and the virus can be transmitted in series by corneal inoculations.

It is of interest to compare this experimental method with the frequency of initial conjunctivitis in cases of encephalitis lethargica clinically observed (MacNalty).

2. Receptive Animals.—In addition to the rabbit, guinea-pig and monkey, the mouse has been found susceptible to the virus of encephalitis, inoculated not only intra-cerebrally, but also by the peritoneal and subcutaneous routes.

3. Study of the Incubation Period.—The period of incubation in the experimental animal is characterised by an elevation of temperature which, beginning on the second or the third day, progressively increases and rapidly attains 41° C. This fever recalls the premonitory fever described in rabies by Babès and Högyes (1109 E), Himmel (1159 A), and Löte (1177 B). It is maintained until the symptoms of the disease are declared and then gives place to pronounced hypothermia, e.g., 34° C. During incubation the blood shows a polymorpho-nuclear leucocytosis

and there is “une diminution de la résistance globale.” The virus of encephalitis progresses rapidly along the nerves. Certain anaesthetics, such as chloral, ether and chloroform, shorten the incubation period and accelerate the appearance of the declared disease.

4. Pathological Histology.—The authors have found in the experimental animals the cell-inclusions described by Da Fano and Ingleby (252) in human subjects. The presence of such bodies in the nerve-cells was detected in about 13 per cent. of cases; they are thus rarer than Negri bodies, but the resemblance between them and Negri bodies constitutes a marked analogy between encephalitis lethargica and rabies.

5. New properties of the virus.—The virus of encephalitis can be preserved for a long time in water and milk and also in the dry state; it is destroyed by bile and by certain dyes and antiseptics (such as methylene blue and potassium permanganate); it is pathogenic, by intracerebral injection, in a dilution of 1 in 1,000 in isotonic saline, and does not appear to be readily diffusible in glycerin and gelatin.

6. Routes of Penetration of the Virus.—The virus of encephalitis is pathogenic for the rabbit by the intramuscular, intraperitoneal and conjunctival routes (in the latter after a lesion of mucous membrane). Inoculated on the cornea, it gives rise to a corneal pustule, accompanied by kerato-conjunctivitis and followed by encephalitis. As already stated, the malady can be transmitted in series by passage from cornea to cornea. The causal organism displays a marked affinity for cutaneous epithelium (ectoderm).

7. Virulence of Body-Fluids and Organs.—The virus has not been detected in the blood (arterial or venous) or in the lymphatic system; it does not appear to exist in an appreciable quantity in the spinal ganglia in contradistinction to rabies and poliomyelitis, where these ganglia are constantly virulent. It has not been recovered from the salivary glands of experimentally infected animals or by catheterisation of the duct of the gland. The authors have been at some pains to conduct experiments, employing the corneal method of inoculation, with a view to explaining the presence of the virus in the saliva while it is found absent from the salivary glands.

In accordance with the clinical observations of Kling (1165 A(b)), Dopter, Achard (1135(b)) and others, they have found that the saliva of certain healthy individuals who have never had encephalitis, contains a filtrable virus identical in all respects with that of encephalitis lethargica. This virus displays a variable pathogenicity; the degree of the activity of the virus can be estimated by the method of corneal inoculation. The authors' experiments on crossed immunity clearly reveal the identity between these varieties (attenuated or virulent) of the salivary virus and the organism of encephalitis. The salivary

virus is a saprophyte of the mouth, where it is present in contact with the epithelial contents of the saliva, the latter being derived from desquamation of the buccal mucous membrane. The authors consider that the organism is probably an obligatory parasite of these epithelial cells.

This important work links up clinical and epidemiological findings in regard to healthy "carriers" of the disease. In encephalitis lethargica, as in poliomyelitis, there exist healthy "carriers"; the active rôle of these "carriers" in the transmission of infection, must now be considered as clearly demonstrated by experiment.

The salivas containing the virus are divided by the authors into "Keratogenous" and "Encephalitogenous," according as their inoculation on the rabbits cornea produces a simple corneal pustule or transmits encephalitis to the animal. In their investigations they find that 80 per cent. of normal salivas are "Keratogenous," whilst 15 per cent. are endowed with properties both "Keratogenous" and "Encephalitogenous." Fortunately, therefore, the healthy "carriers" capable of transmitting encephalitis appear to be comparatively rare, although as yet a large number of healthy persons have not been studied, and the work naturally requires to be further extended.

The authors find that the virus of herpes is of the same nature as that of encephalitis lethargica. The former virus is only less virulent than the latter. The organism can exist in a herpetic vesicle without being simultaneously present in the saliva, just as it may appear in the saliva without, of necessity, giving rise to labial herpes.

As a result of these important pathological findings the authors propound, in their second article, an etiological conception of encephalitis lethargica. This conception is in close accord with that put forward on page 7 of the "Report on an obscure disease—Encephalitis Lethargica," made to the Ministry (then the Local Government Board), in 1918 and elsewhere (629f).

The former conceptions were put forward as the result of analogy with allied diseases and from clinical and epidemiological evidence. They now receive pathological support.

The remaining portion of the French authors' valuable contributions are devoted to the study of immunity and sero-diagnosis, experimental spontaneous contagion and hereditary transmission in encephalitis lethargica; the differences between the virus of encephalitis and that of rabies and of poliomyelitis, respectively, are defined; finally, an inquiry has been initiated into the neurotropic affinities of the virus of the vaccine following on the results obtained by A. Marie (1182 B).

THE CEREBRO-SPINAL FLUID IN ENCEPHALITIS LETHARGICA.—By J. R. PERDRAU, M.B.

The present report deals with the results of an investigation into the chemical and cytological changes in the cerebro-spinal fluid of patients suffering from encephalitis lethargica. This inquiry was started early in 1921 at the same time as another into the nature of the causative agent of the disease.

The specimens of cerebro-spinal fluid were obtained from various sources, as the result of an appeal by the Ministry of Health to the medical profession generally through Medical Officers of Health. For purposes of control a non-selected number of specimens were added, in which some other condition than encephalitis lethargica had been provisionally diagnosed. The present series, therefore, deals with a variety of conditions and diseases of the central nervous system, and the same methods of examination were adopted for the whole series. These methods were such as could be carried out easily in any laboratory without involving the use of complicated and elaborate apparatus and measurements, so that the figures obtained must not be looked upon as absolute ones.

Methods.—These methods in the order in which they were carried out in an average case are as follows:—

(i) *Total Cell Count.*—A Fuchs-Rosenthal counting chamber was employed either with or without the addition of a trace of a stain (e.g., methyl green) to the fluid.

(ii) *Estimation of Total Protein Content.*—Mestrezat's well-known method was adopted:—0.3 c.c. of a 33 per cent. solution of Trichloroacetic acid was added to 2 c.c. of the clear supernatant fluid obtained after centrifugalisation, and after boiling the opacity of the fluid was compared with that of a set of 10 tubes containing from 0.01 to 0.1 per cent. of albumen.

(iii) *Estimation of Sugar Content.*—For reasons to be given presently this was done by a rough naked eye estimation of the amount of reduction obtained on boiling a variable amount of the fluid with half its volume of Fehling's solution. Absence of reduction was only recorded after the tube had been allowed to stand for a quarter of an hour after boiling.

(iv) *Chloride Content.*—A known quantity of the clear fluid obtained after centrifuging (the larger the amount, the smaller the technical error), was diluted with about twice its volume of distilled water, a few drops of a 5 per cent. aqueous solution of potassium chromate were added, and the chloride was titrated with a decinormal solution of silver nitrate. The amount of the latter, equivalent to 1 c.c. of the cerebro-spinal fluid multiplied by 0.585, gives the percentage of chloride present. The error due to the presence of albumen is quite small and can be neglected for all practical purposes.

(v) *Differential Count of Cells.*—The deposit obtained on centrifugalisation was transferred to two slides by means of a capillary pipette or a platinum loop, and a drop of Pappenheim's methyl green-pyronin stain added to one, and a drop of a weak aqueous solution of methylene blue to the other. The slides were mounted as wet preparations, the coverslips ringed with vaseline and the preparations were examined not less than an hour afterwards. The protoplasm of plasma cells take on an intense pink stain and that of lymphocytes a pale pink in the Pappenheim preparation and are easily counted, but polymorphonuclears are best seen in the methylene blue preparation. In the case of freshly withdrawn fluids it is best to add a slight excess of methyl green to the Pappenheim preparation, as the green colour is quickly discharged otherwise. This method is of particular advantage when the total number of cells present is small. Alzheimer's classical celloidin method is obviously unsuited to the clinical pathologist, who has to send a report within a few hours of receipt of the specimen.

The methods set out above are quite sufficient for fluids which are quite clear and free from clot. But if a clot appears on standing, or the fluid is obviously turbid on removal, some additional examinations will have to be made, *e.g.*, dried films of the deposit should be stained by Gram's and Ziehl-Neelsen's methods, and various culture media inoculated, &c.

One point should be noted, though, in the case of fluids from cases of tuberculous meningitis; these fluids are mostly clear immediately after withdrawal and should be collected in at least two tubes. The methods set out above should be applied to the contents of one tube as soon after withdrawal as possible, but the second tube should be put away and allowed to stand undisturbed over night. This second tube will be found next day to contain a thin, threadlike, central coagulum, which should be gently loosened at the top and allowed to settle at the bottom of the tube, and under no circumstances should it be lifted out of the undisturbed fluid. After pouring off the fluid or after centrifuging, the clot should be transferred to a slide, spread out, allowed to dry, fixed and stained for tubercle bacilli in the usual way. A careful search for these organisms will generally prove successful if the case is one of tuberculous meningitis. During the five years that I have used this method I have obtained 85 per cent. of positive results with it, whilst immediate and prolonged centrifuging has only rarely yielded a positive result.

The reason for emphasising this point in regard to tuberculous meningitis is that roughly 1 in 5 of the specimens of cerebrospinal fluid, which were received with a provisional diagnosis of encephalitis lethargica, turned out to be tuberculous. But, as will be found further on, a confident diagnosis of tuberculous meningitis can be made from the general characters of the fluid even when one fails to find tubercle bacilli in the clot or deposit.

The reason for the formation of a central coagulum in tuberculous fluids seems to be the small amount of fibrin present in these fluids, whilst a large clot loosely adherent to the sides of the tube will be found in fluids from cases of septic meningitis, where the amount of fibrin is comparatively much greater.

The presence of these clots, which enmesh a large proportion of the cellular exudate, greatly interferes with the total cell count, which must necessarily be only an approximate one. As some of these fluids reached the laboratory 24 hours or longer after removal, it was soon found that many had to be rejected on account of autolysis of the cells, or bacterial contamination, with the result that the sugar content of the fluid in particular became an uncertain quantity. It was therefore decided to preserve the cells by supplying sterile bottles containing one drop of a 1 per cent. aqueous solution of methyl green, which acts as a fixative as well as a nuclear stain and whose colour is almost entirely discharged in a very short time. An accurate estimation of the sugar was therefore found to be impossible, so that only a rough estimation of it will be given in this paper. As a trace of the green colour returns on boiling an allowance had to be made for it in the estimation of the total protein content by Mestrezat's method, but no difficulty was encountered as the result of it.

In the case of fluids containing an admixture of blood a deduction of one cell for each 500 red blood corpuscles present per c.m.m. was made from the total cell count, but if the number of red corpuscles exceeded 2,000 per c.m.m. the specimen was not included in this series.

General Results.—Out of the number of specimens received in which a provisional diagnosis of encephalitis lethargica had been made, 30 were found to be sufficiently well preserved and to be accompanied by clinical notes of the case to justify their inclusion in this report. The general results in these 30 cases were as follows:—

TABLE I.
Clinical Diagnosis: probably Encephalitis Lethargica.

Result of examination of fluid or of post-mortem examination.	Total number of cases.	Percentage.
Tuberculous meningitis - - - -	6	20 per cent.
Brain tumour - - - - -	5	17 " "
Cerebral thrombosis - - - -	2	7 " "
So-called meningismus of infants - -	1	3 " "
Encephalitis lethargica - - - -	16	53 " "

In connection with these figures it is interesting to note here that as part of an attempt at isolating the virus of the disease a post-mortem examination was secured in a certain number of

cases in which a provisional diagnosis of encephalitis lethargica had been made, but in which the cerebro-spinal fluid had not been examined during life, with the following results:—

TABLE II.

Clinical Diagnosis; probably Encephalitis Lethargica (Cerebro-spinal Fluid not examined during life).

Result of post-mortem examination.	Total number of cases.	Percentage.
Tuberculous meningitis - - - -	4	22.2 per cent.
Brain tumour - - - -	5	27.8 " "
Brain abscess - - - -	1	5.6 " "
Pachymeningitis hæmorrhagica - -	1	5.6 " "
Cause not encephalitis lethargica - -	3	16.7 " "
Encephalitis lethargica - - - -	4	22.2 " "

As regards the three cases where the cause of death is stated not to be encephalitis lethargica, no naked-eye lesion of the central nervous system was found, and a microscopical examination of the brain as well as intra-cerebral injection of an emulsion of the brain into rabbits proved completely negative. The cause of death in those three cases respectively was apparently as follows:—Uræmia, Epilepsy and Pneumonia complicating a mental disorder.

It is evident from Tables I. and II. that tuberculous meningitis and brain tumour are the conditions most commonly confused clinically with encephalitis lethargica, being together responsible for 37 per cent. and 50.0 per cent. respectively of erroneous diagnoses in the two classes of cases considered here; the average for the two classes combined is therefore 43.5 per cent.

This figure is important enough to necessitate a careful study of the characters of the cerebro-spinal fluid in these three diseases, in order to try and discriminate between them and thereby render such examination of real help to the clinician in arriving at a correct diagnosis. The results of such an examination are set out in detail in Tables III., IV., V., and VI.

Normal Cerebro-spinal Fluid.—Before discussing any departures from the normal as revealed by Tables III. to VI., one must first be quite clear as to what the figures are for the various constituents of a normal cerebro-spinal fluid.

The figures most often quoted in this respect are those of Mestrezat (1,186) and of Nawratzki (1,193). "Normal" figures have often been given since then. They are often not the figures obtained in a series of normal subjects, but are stated to be the "normal" figures found in cases of insanity, epilepsy, hydrocephalus, &c., &c. Mestrezat's figures are based on the examina-

tion of the cerebro-spinal fluid in a series of subjects suffering from some simple, non-nervous surgical condition, such as hernia, &c. As Nawratzki's observations were largely made in calves, the figures which will be quoted here are those of Mestrezat and of Pereira (1202), both of whose figures were reached from the systematic investigation of a large number of cases. The technique employed by those two investigators has been essentially the same all through, namely, those described by Mestrezat.

Total Protein.—Mestrezat, 0.013–0.030 per cent.; average, 0.018 per cent. Pereira, 0.018–0.023 per cent.

Sugar.—Mestrezat, 0.048–0.058 per cent.; average, 0.053 per cent. Pereira, 0.053–0.058 per cent.

Chloride.—Mestrezat, 0.725–0.740 per cent, average, 0.732 per cent. Pereira, 0.731–0.732 per cent.

Cells.—Mestrezat adopts Mme. Nageotte's figures, viz., 0.5–1.5 per c.m.m. Pereira gives 1 per c.m.m.

Nageotte's counting chamber was used in both cases, and the cells are stated to be lymphocytes only by both investigators.

The figures of Mestrezat and Pereira therefore agree very closely.

In the case of the chloride content the figures given above have been almost universally confirmed, but the same cannot be said of the figures for the sugar and the cell contents and to a small extent of the protein content.

Total Protein Content.—The figures of Mestrezat and Pereira for the total protein content have been abundantly confirmed by other investigators, except that Mestrezat seems to have fixed the upper limit of normal variations at too high a figure (0.030 per cent.). Fontecilla and Sepulveda (1,143), who give 0.01 per cent. as the normal average, have criticised this figure (0.030 per cent.) as much too high, because they have never found even 0.015 per cent. of total proteins in fluids which did not show some other abnormalities or which were not obtained from subjects suffering from some pathological condition of the central nervous system. This criticism, in so far as it applies to the figure of 0.030 per cent., seems to be abundantly justified, when one examines the detailed figures which have been published in recent years for large series of fluids obtained from very varied sources. In the course of this paper 0.02 per cent. will be accepted as the higher limit of normal variations.

Sugar Content.—In the case of the sugar content some doubt has been thrown on the correctness of the figures quoted above as the result of some recent work on the so-called hyperglycorrhachia of encephalitis lethargica, and it seems best to delay

the discussion of the normal figures until those obtained in encephalitis lethargica come to be considered.

Cell Content.—The systematic cell count of the cerebro-spinal fluid in affections of the central nervous system by means of counting chambers, such as those of Nageotte or of Fuchs-Rosenthal, has only come into general use within recent years. They give very constant results and they have revealed that the normal cell content of the cerebro-spinal fluid is much smaller than used to be thought. A cell count in excess of 3 per c.m.m. in an otherwise apparently normal fluid is generally admitted now to be a very rare occurrence, but higher figures are still given occasionally. Boyd (1,115), for instance, says that from 1 to 5 cells per c.m.m. may be taken as normal and anything over 10 as abnormal, with an indefinite zone between 5 and 10. These figures are much higher than the majority of observers would agree to at the present time.

The figures of Nageotte and Pereira have received abundant confirmation from many quarters. In order to allow a margin of safety for possible individual variations counts of 5 or more per c.m.m. will be treated as abnormal in this paper.

The differential count offers no difficulty, as lymphocytes are generally admitted to be the only cells present in a normal fluid. Fontecilla and Sepulveda (1,143), however, as the result of an examination of 3,000 "normal" fluids give the following formula for a normal differential count—

Lymphocytes	-	-	93.0 per cent.
Large mononuclears	-	-	2.0 "
Polymorpho-nuclears	-	-	5.0 "

The work of these authors has only been recently published (1921) and stands in need of confirmation, as it is diametrically opposed to the generally accepted opinion that no other cell than the lymphocyte is present normally in the cerebro-spinal fluid; the presence of the polymorpho-nuclear in a normal fluid is a particularly doubtful point. A specimen of cerebro-spinal fluid obtained by lumbar puncture, which fails to show a few red blood corpuscles either in the counting chamber or on centrifuging, is not very common. In only 15 out of the last 71 specimens examined in this laboratory was blood not detected microscopically. It is therefore quite possible that an occasional polymorpho-nuclear cell can gain entrance into the cerebro-spinal fluid through injury, however slight, of a small venule in the process of lumbar puncture, and when one remembers that the percentages of a normal fluid are calculated on a very small total of cells, a single polymorpho-nuclear acquires undue prominence when expressed as a percentage of the total number of cells present.

In the course of this report no difference will be made between the small and the transitional lymphocytes, the two being grouped together under the term "lymphocyte."

TABLE III.*

Encephalitis Lethargica (Acute Stage).

No. of Case.	Total Protein per-centage.	Sugar.	Total Chloride per-centage.	No. of cells per c.m.m.	Differential count of cells.			
					Lymphs per-centage.	Plasma cells per-centage.	Poly-morpho-nuclear per-centage.	Large Endo-thelial per-centage.
1	0.02	N +	—	91	c. 100	occasnl.	—	—
22	0.05	N	0.702	5	c. 100	occasnl.	—	—
27	0.02	—	0.761	6	100	—	—	—
31	—	—	0.702	14	100	—	—	—
35	0.01	N	0.731	11	100	—	—	—
88	0.04	N —	0.702	56	c. 100	occasnl.	—	—
average.	0.03	N	0.720	30	c. 100	occasnl.	—	—

TABLE IV.

Encephalitis Lethargica (Subacute or Chronic Stage).

No. of Case.	Total Protein per-centage.	Sugar.	Total Chloride per-centage.	No. of cells per c.m.m.	Differential count of cells				Remarks.
					Lymphs per-centage.	Plasma Cells per-centage.	Poly-morpho-nuclear per-centage.	Large Endo-thelial per-centage.	
1b	0.02	N	0.819	9	100	—	—	—	Congenital mental deficiency.
1c	0.02	N	0.737	3	100	—	—	—	
3	0.02	N+	0.743	5	100	—	—	—	
6	0.02	N	0.731	1	100	—	—	—	
7	0.03	N—	0.907	1	100	—	—	—	
8	0.01	N+	0.731	1	100	—	—	—	
9	0.04	N+	0.702	3	100	—	—	—	
10	0.03	N	0.848	3	100	—	—	—	
12	0.02	N	0.731	1	100	—	—	—	
13	0.02	N	0.761	1	100	—	—	—	
14	0.02	N+	0.702	3	100	—	—	—	
15	0.02	N—	0.614	2	100	—	—	—	
20	0.02	N+	0.746	3	100	—	—	—	
Average.	0.022	N	0.752	3	100	—	—	—	

* In this table and the following ones the following signs are used: " ? " denotes that no estimation could be made through the quantity of fluid available being too small or through the presence of a clot (e.g., protein and cell contents). The letter " N " in the sugar content column denotes " normal amount," and the " + " or " — " signs denote a somewhat greater or smaller amount of reduction than is normally obtained. In the cell content column the number " 1 " represents one or less than one cell per c.m.m.

The " + " sign in the protein and in the cell content columns denotes that the actual quantities present were larger than the figures given, but could not be estimated on account of the presence of a clot.

The Cerebro-spinal Fluid in Encephalitis Lethargica.—When the results obtained in undoubted cases of encephalitis lethargica came to be analysed, a difference was noticed in several particulars between cases, which were of recent onset and those in which the disease had lasted for some time. These results have therefore been classified under two headings: (i) acute, (ii) subacute or chronic.

Many cases, especially of a mild type, are not seen by the doctor until a week or more after the onset, so that the first lumbar puncture is not performed until the second week of the disease; a fortnight therefore has been arbitrarily assigned to the "acute" stage, whilst those cases, which had lasted more than a fortnight, are here referred to as "subacute or chronic" cases. It should be stated here that in all the cases of encephalitis lethargica the fluid was clear and colourless and no coagulum ever formed on standing.

A consideration of Table III. shows that in the acute stage the following are the main points observed: (1) an inconstant, small increase in the total protein content; (2) a practically normal chloride content; (3) a constant increase in the cell content; (4) a cellular exudate consisting of lymphocytes, occasionally associated with a few plasma cells.

Case I., which shows the highest cell content, is also clinically the most severe case of the series, a considerable amount of chorea of the Huntington type being still present, 12 months after the onset.

Table IV. on the other hand shows that in the subacute or chronic stage: (1) the protein content is almost always within normal limits; (2) the chloride content shows a general upward tendency; (3) the cell content is only rarely above normal; (4) the cells present are invariably lymphocytes.

Here also the most severe case (Case I.) shows the highest cell content, much lethargy being present at the time that the second specimen (Ib) was obtained.

Tables III. and IV. therefore show that there is an obvious difference in the composition of the fluid according to the stage of the disease when it is obtained. A careful search through the literature has shown very few records of a quantitative estimation of the various constituents of the cerebro-spinal fluid at different stages of the disease, although an initial lymphocytosis, which rapidly disappears, has been often recorded.

The records, which I have traced, mostly deal with some only of the constituents of the cerebro-spinal fluid, and very often only global results are given in the form of percentages of the number of cases examined.

Cell Content.—Netter (1196) by means of repeated lumbar punctures in three cases has shown that the initial lymphocytosis may be even exceeded subsequently at periods of exacerbation of the disease. Figures have been given by many observers (Boveri (119e), Bonnard (107)), but as the stage of the illness

is not stated no comparison can be made with those given here. Foster (244) in eight cases found counts varying from 6 to 150 cells per c.m.m. between the 2nd and the 15th day, and in four cases he found from 8 to 35 cells per c.m.m. between the 17th and 34th days. Foster's figures therefore confirm those obtained in this series for the acute stage only. In the present series a pleocytosis of a mild type was noticed in only 2 out of 13 in the later stages of the disease, i.e., 15 per cent. of the total. Rodriguez (1,213) finds from 2 to 25 cells per c.m.m. in nine cases of postencephalitic chorea ("syndrome parkinsonien").

In Appendix II., Table C., a pleocytosis is recorded in 44 out of 269 cases at all stages of the disease, i.e., in 16.3 per cent.

Most writers have insisted on the lymphocytic nature of the cellular exudate, and this fact is well borne out in this series, but polymorpho-nuclears have been recorded by a few writers (Bonnard, Rodriguez). Neal (729c) has recorded as many as 60 per cent. polymorpho-nuclears in one case!

Total Protein Content.—An increase in the total protein content is present in 2 out of 5 (i.e., 20 per cent.) in the acute stage, and in 1 out of 13 (i.e., 7 per cent.) in the later stages. If these percentages are compared with those obtained for the pleocytosis in those two stages, namely, 100 per cent. and 15 per cent. respectively, it is evident that the increase in the cell content and in the total protein content do not advance *pari passu*, at any rate in the acute stage. This point has been stressed by many observers, especially of the French school, as a valuable one in diagnosis ("dissociation cyto-albuminique").

Boveri finds a doubtful increase up to 0.025 per cent. in 2 out of 16 cases, and Bonnard records an increase in 4.3 per cent. of 42 cases, but the latter does not state the stage of the disease and neither of them mentions the method used in the estimation of the protein content.

Foster, using the Denis-Ayer method, found from 0.0219–0.080 per cent. (average 0.044 per cent.) in 11 specimens obtained during the first fortnight, and from 0.025 per cent. to 0.0727 per cent. in four observations made at later stages of the disease. He therefore found an increase of varying intensity in practically all his cases. This observation does not seem to have been confirmed by other observers, whose methods of estimation are unfortunately not stated in the majority of cases.

In Appendix II., Table C., it will be found that a total protein increase has been recorded in 26 out of 269 cases for all stages of the disease.

Chloride Content.—The figures obtained for chlorides in the acute stage agree fairly closely with the normal ones of Mestrezat and Pereira, the average being 0.720 per cent. as compared with the normal 0.731 per cent. The decrease is much too small to be of any aid in diagnosis. In the more chronic stages, however,

a higher average is obtained, viz., 0.752 per cent., three out of the 13 cases actually showing figures higher than 0.8 per cent. This inconstant increase has been noted by several investigators, e.g., Howell (476a), who records figures of over 0.8 per cent. and Rouzaud and Laporte (544a), who also record similar figures in 3 out of 10 cases.

Sugar Content.—For reasons already stated a quantitative estimation of the sugar content was not carried out in this series, none of the methods in use at the present time being exactly suited for the routine examination of fluids which reach the clinical pathologist and on which reports are wanted immediately.

A systematic investigation of the sugar content has been carried out by a few workers, amongst whom two stand out prominently, Foster (344) and Coope (223). Several French writers had described an increased amount of sugar in encephalitis lethargica, but as a rule the methods they employed are not mentioned. Rouzaud and Laporte (544a) for instance have recorded as much as 0.101 per cent. in one case.

Foster, using the Folin-Wu method, found figures varying between 0.053 per cent. and 0.113 per cent., with an average of 0.076 per cent. in 11 cases. Coope, also using the Folin-Wu method, obtained figures varying between 0.054 per cent. and 0.094 per cent., with an average of 0.074 per cent. The result obtained by those two investigators, who used the same method, therefore agree very closely; but whereas Foster accepts Mestrezat's normal figure of 0.053 per cent., and describes a hyperglycorrhachia in encephalitis lethargica, Coope does not, and questions the correctness of Mestrezat's figures for a normal fluid. Coope also investigated the sugar content in a wide variety of diseases, amongst which the figures obtained in cases of insanity are the most interesting. He found from 0.06 per cent. and over in 60 out of 69 cases and very rightly comes to the conclusion that either Mestrezat's normal figure is much too low or the sugar content is increased in a large variety of nervous conditions. Rieger and Solomon (1,211), using the Lewis-Benedict method found from 0.05 per cent. to 0.09 per cent., with an average of 0.07 per cent., in 175 cases of insanity. These figures agree closely with those of Coope.

Coope reviews very completely the so-called hyperglycorrhachia of encephalitis lethargica and leaves the impression on the reader that the great difference in the figures obtained by various investigators is most probably due to the lack of uniformity in the methods employed, and that the normal figures, which have held the field so far, should be subjected to the test of parallel estimations by these several methods on the same specimens of cerebro-spinal fluid. It should be noted that both Mestrezat and Pereira used the lead acetate method, described by the former (*loc. cit.*, p. 27).

It is therefore probable that the sugar content in encephalitis lethargica is not increased at all, or, if it is, such increase is so

common in other diseases of the central nervous system as to be of no help in diagnosis. But as to one point there is general agreement and that is that the sugar content is not diminished in encephalitis lethargica.

Conclusions.—It therefore appears from the above that the following are the chief points to be noted at the onset of the disease and at periods of exacerbation: (1) a pleocytosis which varies in direct ratio with the severity of the disease; (2) a small increase in the total protein content, the latter not keeping pace with the former ("dissociation cyto-albuminique"); (3) the cells present being lymphocytes with the occasional presence of a few plasma cells.

In the subacute or chronic stages of the disease the cerebro-spinal fluid is practically normal except for an inconstant small increase: (1) in the number of cells per c.m.m., the cells being lymphocytes only; (2) in the protein content; (3) in the chloride content; the last factor (3) is apparently the most important, especially when figures of 0.8 per cent. and over are obtained.

TABLE V.
Tuberculous Meningitis.

No. of case	Total Protein per centage.	Sugar.	Total Chloride per centage.	Differential count of cells.					Tb. Bac.	Remarks.
				No. of cells per c.m.m.	Lymphs per centage.	Plasma Cells per centage.	Poly-morpho-nuclear per centage.	Large Endothelial per centage.		
16	0.02+	Traces	0.614	54+	78	5	17	A few	Yes	Fine central coagulum on standing.
24	0.16+	Nil	0.614	150+	84	8	8	A few	Yes	Coagulum intact.
25	0.10+	Traces	0.585	80	93	1	6	A few	No	Coagulum broken up.
29	0.48+	Nil	0.527	184+	93	4	3	A few	Yes	Coagulum broken up
33	0.06+	Traces	0.512	91	80	8	12	A few	Yes	Coagulum intact.
36	0.22	Traces	0.585	371	90	3	7	A few	Yes	Fine central coagulum on standing.
51	0.12+	Traces	0.526	108+	94	2	2	A few	No	Coagulum broken up.
av.	0.16+	Traces	0.566	148+	87.5	4.5	8	A few	71 per cent.	

The Cerebro-spinal Fluid in other Conditions which may simulate Encephalitis Lethargica.

(a) *Tuberculous Meningitis.*—Table V. shows clearly the very characteristic results obtained in cases of tuberculous meningitis, some abnormality being noted under each heading; total protein

content much increased; sugar content considerably diminished; chloride content much diminished; cell content greatly increased, the differential count showing a large preponderance of lymphocytes constantly associated with a small percentage of plasma cells and of polymorpho-nuclears as well as with a few large endothelial cells.

By following the technique recommended in this paper the tubercle bacillus was found in 5 out of 7 specimens, the only exceptions being two of the specimens which came by post and in which the clot had been reduced to a fine powder. The naked-eye appearance of the clot already noted in this disease is another point to be remembered.

The various abnormalities noted above are well known to occur in tuberculous meningitis. They have all been stressed by various workers, either singly, such as the chloride content, or in association with another, such as chloride and protein, or chloride and sugar, &c. None of these characters by themselves can be described as pathognomonic of tuberculous meningitis, as has often been claimed (*cf.* Tables VI. and VII.) but the complete picture is certainly one not encountered in any other condition.

The following two cases will illustrate the point that a diagnosis should not be based on certain isolated characters only. The chloride content, which is usually much lower in tuberculous meningitis than in septic meningitis, is noted as 0.614 per cent. in two cases of tuberculous meningitis (Table V., cases 16 and 24), and also in one case of pneumococcal meningitis (Table VII., case 39a). In one patient (Table VIII., case 26) a chloride content of 0.585 per cent. was found. This was the case of a boy, aged 14, who complained of vague abdominal pains, headache and vomiting. As he was seen at a time when encephalitis lethargica was prevalent, a lumbar puncture was performed on the third day after the onset of symptoms, because it was thought that he might be suffering from that disease. The examination of the cerebro-spinal fluid threw no light on his illness; he was quite well again in another 10 days, had no recurrence of symptoms, and there was no reason for thinking that he suffered from tuberculous meningitis in spite of his unusually low chloride content.

The association of a few plasma cells, polymorpho-nuclears and large endothelial cells with a lymphocytosis has been recorded in general paralysis of the insane, but this disease is not likely to be confused clinically with encephalitis lethargica, and the difference in the Wassermann reaction and in the constituents of the cerebro-spinal are in themselves sufficient to differentiate between the two diseases.

Case 5, Table VIII., gave results which closely resemble those obtained in tuberculous meningitis, but the latter could be excluded by reason of the high chloride content (1.053 per cent.), incidentally the highest of this series. The case was one of multiple cerebral embolisms complicating a chronic rheumatic endocarditis.

Conclusions.—The examination of the cerebro-spinal fluid in tuberculous meningitis yields a very characteristic picture, and no confusion with encephalitis lethargica should be possible, even if the tubercle bacillus is not found.

TABLE VI.
Brain Tumour.

No. of case.	Total Protein per centage.	Sugar.	Total Chloride per centage.	No. of cells per c.m.m.	Differential count of cells.				Remarks.
					Lymphs per centage.	Plasma cells per centage.	Poly-morph. per centage.	Large Endothelial per centage.	
4	0.02	N	0.737	1	100	—	—	—	Intramural glioma of pons at P.M.
4a	0.01	N—	0.702	2	100	—	—	—	"
4b	0.01	N—	0.714	1	100	—	—	—	"
28	0.015	N	0.761	2	100	—	—	—	Oat-shaped cell sarcoma of cerebrum near surface at P.M.
41	0.01	N	0.731	1	100	—	—	—	Decompressed and still alive.
50	0.04	N	0.761	8	99	—	1	—	Multiple superficial oat-shaped cell sarcomata of brain at P.M.
53	0.02	N+	0.702	2	100	—	—	—	Intramural glioma of pons and basal ganglia at P.M.
58	0.015	N	0.696	3	100	—	—	—	Intramural glioma of cerebrum at P.M.
Av.	0.017	N	0.725	2.5	100	—	—	—	

(b) *Brain Tumour.*—Table VI. shows that in the case of brain tumours a practically normal cerebro-spinal fluid is encountered and the only points of difference between them and the chronic stages of encephalitis lethargica are the absence in the former of the occasional increase in the cell content and in the chloride content, which have been noted in the latter. It is interesting that the only case (case 50) which is an exception to the above in showing a small increase in the cell content and in the protein content, is one in which a wide surface of the brain was involved by the tumour with a consequent meningeal reaction and a nearer approximation in the constitution of the cerebro-spinal fluid to what obtains in the acute stages of encephalitis lethargica. All the other cases showed *post-mortem* intramural growths, with little, if any, involvement of the brain surface. It therefore seems that it is not the nature of the neoplasm, but

its position, which determines any alterations in the cerebro-spinal fluid.

It appears therefore impossible to distinguish in a given case by the chemical and cytological examination of the cerebro-spinal fluid between the subacute stage of encephalitis lethargica and those cases of brain tumour in which the growth is purely intramural, this type being practically the only one confused clinically with encephalitis lethargica according to the present series.

When the tumour is found in other situations a different picture is obtained. For instance, Christiansen, quoted by Pontecilla and Sepulveda, has insisted on the great increase in the protein content unassociated with a pleocytosis in cases of tumours of the cerebello-pontine angle.

TABLE VII.
Septic Meningitis.

No. of cases	Total Protein per centage.	Sugar.	Total Chloride per centage.	No. of cells per c.m.m.	Differential count of cells.				Organism found.	Remarks.
					Lymphs per centage.	Plasma Cells per centage.	Poly-morpho-nuclears per centage.	Large Endo-thelial per centage.		
18	?	Nil	0.702	?	75	—	18	7	Meningo-coccus.	Obvious pus.
19	0.05	N—	0.702	?	42	—	52	6	?Pneumo-coccal.	Recovery.
32	0.20	Traces	0.643	1,500+	A few	—	c. 100	—	Meningo-coccus.	Obvious pus and clot.
39	?	Nil	0.702	?	A few	—	c. 100	—	Pneumo-coccus.	" "
39a	?	Nil	0.614	?	A few	—	c. 100	—	Pneumo-coccus.	" "
40	0.21+	Nil	0.702	?	70	—	30	—	Meningo-coccus.	" "
43	0.04	Traces	0.673	960	21	—	77	2	Strepto-cocci.	" "
44	0.04	Nil	0.643	2,570	8	—	90	2	Strepto-cocci and B. Proteus.	" "
46	0.06	Nil	0.760	4,520	2	occasl.	98	—	B. In-fluenza.	" "
59	0.05	Traces	0.633	433	25	—	75	occasl.	Sterile.	Associated with acute mastoiditis.
60	0.04	A little.	0.702	115	97	3	—	Occasl.	Sterile.	Recovery. Cerebellar abscess.

(c) *Septic Meningitis.*—Although septic meningitis does not come within the scope of this investigation, confusion with encephalitis lethargica not having occurred once, a table is appended in which the results obtained in a certain number of

cases are set out for purposes of comparison with Table V. (tuberculous meningitis).

The following points are worthy of notice: the large increase in the cell content amounting in almost all cases to obvious pus; the large proportion of polymorpho-nuclears; the rarity of plasma cells; the almost total disappearance of sugar and the diminution in the chloride content, which only just reaches the upper limit of the amounts found in tuberculous meningitis.

TABLE VIII.
Miscellaneous.

No. of Case.	Total Protein per centage.	Sugar.	Total Chloride per centage.	No. cells per c.m.m.	Differential count of cells.				Remarks.
					Lymphs per centage.	Plasma Cells per centage.	Poly-morpho-nuclears per centage.	Large Endo-thelial per centage.	
5	0.25	?	1.053	153	78	8	14	—	Cerebral embolism at P.M.
21	0.01	N	0.687	3	100	—	—	—	So-called meningismus in pneumonia in a child.
30	0.01	N	0.702	4	100	—	—	—	Disseminated sclerosis.
23	0.03	N	0.760	2	100	—	—	—	Miner's nystagmus.
42	0.01	N+	0.702	1	100	—	—	—	Fracture of anterior fossa of skull.
38a	0.01	N+	0.731	5	70	—	30	—	Recovery.
34	0.03	N	0.731	12	c. 100	occasl.	—	—	Cerebral hæmorrhage (or thrombosis).
52	0.03	?	0.702	1	100	—	—	—	Nageotte's syndrome.
56	0.03	N+	0.760	3	100	—	—	—	Uræmia—granular kidneys.
61	0.01	N—	0.643	4	100	—	—	—	Caseous adrenals and septicæmia.
62	0.05	N	0.643	56	93	—	7	—	Syphilitic pachy-meningitis.
26	0.01	N—	0.585	1	100	—	—	—	Vide notes in body of report.

Other Affections of the Central Nervous System.—Table VIII. shows the results obtained in a variety of other conditions, which are too few to serve any useful purpose as far as comparison with the other conditions considered so far goes.

Is there a Meningeal Form of Encephalitis Lethargica?—It would be instructive now to test the conclusions arrived at so far in cases where a clinical diagnosis could not be made with certainty, but in which the cerebro-spinal fluid was examined. Only one case is available for this purpose and it is an interesting one because it raises afresh the question of the possible existence of a meningeal form of encephalitis lethargica, which has not been generally accepted so far.

Case 49.—An infant 13 months' old was admitted on 12.7.21 into Lambeth Infirmary under Dr. Stebbing (to whom I am indebted for the clinical notes), with a history of having been quite well until the day before admission, when it became drowsy and looked generally unwell. On the 12th it had some (?) convulsions and was brought up for treatment. The child was drowsy, had marked retraction of the head, an obvious left internal squint, a positive Kernig's sign, some rigidity of the left arm, a temperature of 103°, with signs of early bronchopneumonia at the left apex. The case was thought to be one of meningitis and a lumbar puncture was performed. The fluid contained so much blood that the puncture was repeated on the following day, when a perfectly clear fluid was obtained. Examination of this fluid gave the following results: total protein 0.03 per cent.; sugar somewhat under normal (not estimated quantitatively); chloride 0.702 per cent.; cells 218 c.m.m. as well as 615 red corpuscles per c.m.m. The differential count gave the following formula: lymphocytes, 87 per cent.; plasma cells, 13 per cent.; and a very rare polymorpho-nuclear, the latter probably being brought in with the blood.

The temperature came down by crisis in three days, but the nervous signs and symptoms persisted for another 10 days, when they passed off gradually, the drowsiness persisting longer than any other symptom.

The child was discharged quite well on 10.10.21.

It was at first thought that the so-called meningismus of children might account for the symptoms, but contrary to what usually happens in such cases, there was no immediate improvement after the lumbar puncture, and, in addition, the definite pathological alterations noted in this fluid are not seen in cases of meningismus. For instance, cases 21 and 30 (Table VIII.), in which meningismus was present in association with bronchopneumonia, show no pleocytosis and no increase in the protein content, and therefore conform with what other observers have recorded in this condition.

The general picture obtained in this case agrees closely with what has been found in the acute stages of encephalitis lethargica (Table III.).

There is a small increase in the protein content, there is a decided pleocytosis, the cellular exudate consists mostly of lymphocytes with a smaller proportion of plasma cells.

Here the high proportion of plasma cells and the pleocytosis point to meningeal irritation, of which there was clinical evidence.

Although it cannot be stated positively that this case was one of the so-called meningeal form of encephalitis lethargica, it is at any rate highly suggestive of such a condition.

The Cerebro-spinal Fluid in Acute Poliomyelitis.—Although not a single case of acute poliomyelitis was seen in the course of this inquiry a note on the characters of the cerebro-spinal fluid in this disease may not be out of place here. The following particulars are taken from the monograph of Durand (1,136), who has reviewed the literature of the subject very thoroughly.

In the acute stage of the disease before any paralysis or signs of meningeal inflammation appear, the following are the main alterations noted:—

Pressure generally increased;

Proteins generally, but only moderately, increased;

Cells very often, but not always, increased; the number of cells per c.m.m. is very variable, as many as 2,000 having been recorded, but numbers well under 100 are more common;

The differential count shows the constant presence of polymorpho-nuclears, which average 60 per cent. of the total number of cells present, the rest being mostly lymphocytes associated with a few endothelial cells.

When the disease is at its height and paralysis appear, the following are the characters of the fluid:—

Pressure very generally increased;

The fluid is occasionally turbid, but as a rule it is clear immediately on removal. A coagulum of variable size forms on standing according to the amount of fibrin present;

Proteins are usually increased to a moderate extent, but occasionally the increase is so great that massive coagulation may take place. The increase in the protein content may precede the pleocytosis.

Cells almost always increased (average 150 per c.m.m.), but counts of 2,400 are on record.

The differential count is usually the reverse of what is seen in the acute stage, i.e., the lymphocytes predominate, but polymorphonuclears are almost always present, though in smaller numbers.

Differential Diagnosis from Encephalitis Lethargica.—The presence of the polymorphonuclears is sufficient by itself to distinguish between the two diseases, but the following factors are equally important, though not constant: spontaneous clotting through presence of fibrin; a greater increase in proteins than is found in encephalitis lethargica; an increase in proteins without a concomitant pleocytosis (i.e., the reverse of what is found in encephalitis lethargica); and a pleocytosis which far exceeds that recorded in encephalitis lethargica.

When the symptoms are disappearing in poliomyelitis, a simple lymphocytosis unassociated with the presence of polymorphonuclears may be found. At that stage in encephalitis lethargica the count is almost always normal.

Finally, as in encephalitis lethargica, the cerebro-spinal fluid in poliomyelitis is invariably sterile as far as ordinary cultural and staining methods for the demonstration of bacteria are concerned.

Specimens of cerebro-spinal fluid are gratefully acknowledged from the following:—

Dr. Harold Pritchard and his house physician, Dr. Marshall,
of the West London Hospital,
Dr. Caiger, South Western Fever Hospital,
Dr. Goodall, North Western Fever Hospital,
Dr. Thackray, St. Pancras Hospital,
Dr. Carr and his house physician, Dr. Longbottom, Royal
Free Hospital,
Dr. Braxton Hicks, Westminster Hospital,
Dr. MacCormac, St. James' Infirmary,
and many others.

EXPERIMENTAL INVESTIGATION INTO THE VIRUS OF ENCEPHALITIS LETHARGICA—By J. R. PERDRAU, M.B.

When encephalitis lethargica re-appeared in this country in epidemic form in the winter of 1920–21, a series of experiments was contemplated with the object of continuing the work of McIntosh, who had been the first to reproduce the disease in laboratory animals. But these experiments of ours were only started in the month of March, 1921, when the epidemic rapidly declined, as reference to the chart prepared by Dr. Parsons, and published in another section of this report, shows very clearly. The disease took on a much milder form from about that time, and great difficulty was experienced in securing post-mortem examinations in fatal cases of the disease. From one well verified case, however, certain work was done which seems worthy of brief record.

History of the Case of Encephalitis Lethargica from which the Material was obtained.—The case was that of a woman, æt. 56, admitted into the West London Hospital on 21.1.21, under Dr. Harold Pritchard (House Physician, Dr. W. H. Marshall), to whom I am indebted for the clinical notes, &c.

She gave a history of headache, drowsiness, and diplopia dating back to 8.1.21. On admission she was very lethargic and still suffered from headache and diplopia, but no strabismus could be made out. The pupillary and deep reflexes were normal and there was no loss of sphincter control. No tremors or

pareses were noted. The temperature was 101° F. An examination of the cerebro-spinal fluid yielded the following result:—

Total protein	-	-	0.03 per cent.
Sugar	-	-	Somewhat under normal.
Chloride	-	-	0.892 per cent.
Cells	-	-	6 per c.m.m.
Differential count	-	-	All lymphocytes.

This count agrees quite closely with what has been found in well-established cases of encephalitis lethargica.

A blood count showed 11,900 white cells per c.m.m., with 81 per cent. polymorphonuclears, 14 per cent. lymphocytes, and 5 per cent. eosinophiles. She gradually improved after admission and was practically well again by 9.2.21.

On 17.2.21 she suddenly relapsed into her former condition. The left pupil was now found to be slightly larger than the right, a little nystagmus was present, and the diplopia, lethargy, and headache returned. No motor palsies were detected. Her condition gradually became worse. The lethargy increased, and by the 15.3.21 she was comatose. She developed signs of broncho-pneumonia, with a temperature of 106°–107° F., and died on 18.3.21.

A post-mortem examination was held 11 hours after death. Apart from a confluent broncho-pneumonia affecting both lungs, but more particularly the left base, and some fatty changes in the liver, nothing of interest was found in the body. The brain was somewhat congested, but showed no other naked-eye lesion.

Pieces from various parts of the central nervous system were preserved as follows: A few pieces were placed in a sterile bottle without any preservative and were used for inoculation purposes some two hours later; some more pieces were placed in 50 per cent. glycerine in saline, in 10 per cent. formalin and in absolute alcohol respectively. Some of the mucous membrane of the nasopharynx was also kept without any preservative, and was emulsified in normal saline immediately afterwards.

Experimental Inoculation of Material.

1. *Fresh Brain.*—A few small pieces of the pons and basal ganglia were emulsified in about five parts by volume of normal saline immediately after the autopsy and filtered on paper. Rabbit R. 2 received intracerebrally 0.2 c.c. of the filtrate. The animal made a good recovery, showed no signs of illness, and was killed four weeks later. No lesion was found, naked-eye or microscopically, in the brain, &c.

2. *Fresh Nasopharyngeal Mucous Membrane.*—Pieces of the membrane were emulsified in a little normal saline, filtered through a small Masson candle, and 0.2 c.c. of the filtrate was injected intracerebrally, on 19.3.21, into rabbit R. 3. Good recovery. Killed four weeks later. No lesion found.

3. *Brain kept in 50 per cent. Glycerine in the Ice Chest for three days.*—An emulsion of pieces of the pons and basal ganglia,

preserved in 50 per cent. glycerine in the ice chest for three days, was prepared as in 1 and 2, after previous thorough washing with normal saline. The emulsion was filtered on paper, and 0.2 c.c. of the filtrate was injected intracerebrally into rabbits R. 4 and R. 5 on 21.3.21. At the same time part of the emulsion was filtered through a small Massén candle and 0.2 c.c. of the filtrate was injected into rabbit R. 6. They all made a good recovery. R. 4 was killed four weeks later, and R. 5 and R. 6 six weeks later. No lesion of any kind was found in any of them.

4. *Brain kept in 50 per cent. Glycerine in Ice Chest for 13 days.*—An emulsion of the brain which had been kept for 13 days in 50 per cent. glycerine in the ice chest was prepared as in 3, and filtered through a small Massén candle. On 31.3.21 rabbits R. 7 and R. 8 received intracerebrally 0.2 c.c. of the filtrate each. They both made a good recovery, and were killed five weeks later. No lesion found.

The failure to transmit the disease in this instance may be explained by the fact that the case was a long-drawn-out one (it lasted 10 weeks in all), and the virus had probably become much attenuated in that time. Levaditi and his collaborators (571f), who have succeeded in obtaining a fixed virus in this disease, record eight failures before they succeeded in passing the disease to animals (rabbits and monkeys).

Although attempts were constantly made to secure material from fatal cases of the disease, and especially from acute ones, no further post-mortem material was forthcoming in the course of 1921 from genuine cases of encephalitis lethargica. But many experiments were carried out with material obtained at the bedside.

Inoculation of the Cerebro-spinal Fluid from cases of Encephalitis Lethargica.

The cerebro-spinal fluid from cases of encephalitis lethargica when injected intracerebrally into rabbits fails to transmit the disease.

1. On 6.4.21, rabbit R. 9 received intracerebrally 0.2 c.c. of the cerebro-spinal fluid of case 27, obtained by lumbar puncture. Good recovery. Killed six weeks later. No lesion found.

2. On 17.5.21, rabbits R. 13 and R. 14 received intracerebrally 0.2 c.c. of the cerebro-spinal fluid of case 31, obtained by lumbar puncture on the 9th day of the disease. Good recovery. R. 13 was killed six weeks later and R. 14 12 weeks later. No lesion found.

Inoculation of Nasopharyngeal Washings from cases of Encephalitis Lethargica and from Contacts.

Intracerebral injections of the filtered nasopharyngeal washings from cases of encephalitis lethargica, and from persons who have been in daily contact with sufferers from the disease over long periods, fail to transmit the disease.

1. On 19.5.21, rabbit R. 15 received intracerebrally 0.2 c.c. of the nasopharyngeal washings of case 31, filtered through a Massén candle. Good recovery. Killed 12 weeks later. No lesion found.

2. On 9.7.21, rabbit R. 17 received intracerebrally 0.25 c.c. of the nasopharyngeal washings of a fairly severe case of encephalitis lethargica, filtered through a Massén candle. Good recovery. Killed five weeks later. No lesion found.

3. On 15.7.21, rabbits R. 20 and R. 21 received intracerebrally 0.25 c.c. of the nasopharyngeal washings of a chronic case of encephalitis lethargica (case 1), filtered through a Massén candle. R. 20 made a complete recovery, but R. 21 showed some paresis of all four limbs eight days later. Found dead next morning. Unfiltered emulsions of the brain of R. 21 in normal saline were immediately injected intracerebrally into six rabbits, R. 22–27. One of them, R. 25, developed a septic meningitis on the next day, but the others made a good recovery and were killed several weeks later. No histological evidence of an encephalitis was obtained in rabbit R. 21.

Several stock rabbits developed similar symptoms to those of rabbit R. 21 at about the same time, and as only a few patches of broncho-pneumonia, and no brain lesion, could be found post-mortem, it is highly probable that R. 21 suffered from the same disease as the stock animals.

But in order to make sure of this point, fresh nasopharyngeal washings were obtained from the same patient and treated in the same way, and on 12.8.21 rabbits R. 28 and R. 29 received 0.4 c.c. of the filtrate intracerebrally. They both made a good recovery, and were killed several weeks later without showing any signs of encephalitis.

4. On 29.8.21, rabbits R. 36 and R. 37 received intracerebrally 0.4 c.c. of the filtered nasopharyngeal washings of a recurrent case of encephalitis lethargica, case 31. They both made a good recovery, and were killed five weeks and four weeks later respectively. No lesion found.

5. On 12.7.21, rabbit R. 18 received intracerebrally 0.25 c.c. of the nasopharyngeal washings from the sister of the ward in which a case of encephalitis lethargica (case 1) had been for six months, the patient still showing active signs of the disease at the time. The animal made a good recovery, and was killed five weeks later. No lesion found.

6. On 12.7.21, a similar experiment to the last one was made with the nasopharyngeal washings of the patient who had occupied for six months continually the bed adjacent to that of the same case (case 1). The rabbit, R. 19, made a good recovery, and was killed five weeks later. No lesion found.

Control Experiments.

At the same time as these experiments were in progress, a certain number of control ones were started with material obtained from cases suffering from affections (mostly nervous) other than encephalitis lethargica.

1. *Cerebro-spinal Fluid from case of Meningismus.*—On 12.3.21, rabbit R. 1 received intracerebrally 0.2 c.c. of the cerebro-spinal fluid from case 21, obtained by lumbar puncture. Good recovery. Killed four weeks later. No lesion found.

2. *Cerebro-spinal Fluid from a case of Brain Tumour.*—On 12.4.21, rabbit R. 11 received intracerebrally 0.2 c.c. of the cerebro-spinal fluid of case 28, obtained by lumbar puncture. Good recovery. Killed five weeks later. No lesion found.

3. *Brain from a case of Uræmia.*—On 12.4.21, rabbit R. 10 received intracerebrally 0.2 c.c. of an emulsion of the pons and basal ganglia, filtered on paper, from a case of uræmia complicating an ascending pyelonephritis. Good recovery. Killed five weeks later. No lesion found.

4. *Brain from a case of Disseminated Sclerosis.*—On 13.8.21, rabbit R. 30 received intracerebrally 0.4 c.c. of a thick emulsion in normal saline of the sclerosed areas and of the adjacent white matter from the brain of a typical case of disseminated sclerosis. The animal was quite well for the 14 weeks that it was under observation, and eventually recovered.

5. *Contents of Vesicles from a case of Herpes Zoster.*—The contents of the vesicles from a case of herpes zoster failed to produce an encephalitis in rabbits.

On 24.8.21, rabbits R. 34 and R. 35 received intracerebrally 0.2 c.c. of a dilution of 1 in 10 of normal saline of the contents of a fresh crop of vesicles in a case of thoracic herpes zoster in a child. R. 34 received a similar injection two days later, but made a good recovery, and was killed six weeks later. No lesion found. R. 35 made a good recovery for a whole week, but was found dead quite unexpectedly on the eighth day. No cause of death could be found at the autopsy or by the subsequent microscopical examination of the brain, but in order to make sure that the animal had not succumbed to an encephalitis, an emulsion of its brain was injected intracerebrally, on 1.9.21, into rabbits R. 35A and R. 35B in quantities of 0.4 c.c. They both made a good recovery, and were killed four to five weeks later. No lesion found.

6. *Contents of the Vesicles from a case of Herpes Labialis.*—Levaditi (572c) in his latest contributions to the study of the virus of encephalitis lethargica and that of herpes labialis says that he is unable to differentiate between the two, which appear to be identical in almost every respect. It was therefore natural

that the contents of the vesicles in ordinary herpes should be tested in the same way as the other substances used so far in the course of this investigation. Here a positive result was easily obtained, and the virus has been carried through several generations so far.

On 17.10.21, rabbits R. 39 and R. 40 received intracerebrally 0.2 c.c. each of a 1 in 5 dilution in normal saline of the contents of the vesicles in a case of herpes labialis associated with an ordinary cold in the head in a boy. R. 40 became ill the next day, and died in two days from a septic meningitis due to a streptococcus. R. 39 was rather quiet for the next three days, but took its food well. Temperature 103.6° F. On 23.10.21, it became excited, running round its cage and throwing its head backwards. It was found dead on 24.10.21. At the autopsy the brain was found congested; the point of inoculation was only just recognisable and two small petechiæ of the size of a pin's head were found at the base of the brain. Cultures made from the brain and cerebro-spinal fluid remained sterile. Microscopically a typical severe encephalitis was found. The first passage therefore killed in 7½ days. Pieces of the brain were preserved in 50 per cent. glycerine, and have been used for further experiments, which are still in progress. The following are some of the points made out so far:—(1) the virus can be transmitted in series in rabbits; (2) the second passage killed in six days, and subsequent passages in four days; (3) the virus from the first passage retained its virulence in 50 per cent. glycerine in the ice chest for 34 days (it has not been tested beyond this length of time); (4) the virus from the third passage has kept its virulence unimpaired under the same conditions for 42 days (it has not been tested further than this); (5) no bacteria have, so far, been isolated during any of the passages.

7. *Nasopharyngeal Washings from the same case of Herpes Labialis.*—Intracerebral inoculation of the filtered naso-pharyngeal washings from the same case of herpes labialis gave a completely negative result in the rabbit.

On 17.10.21, rabbit R. 38 received intracerebrally 0.3 c.c. of the nasopharyngeal washings from the case of herpes labialis already referred to under 6, after filtration through a Massén candle. It made a good recovery and remained well up to the 18th day, when it received an intracerebral injection of the herpetic virus referred to under 6. It developed a typical attack of encephalitis, and a microscopical examination of the brain showed the usual lesions associated with this condition. The failure of R. 38 to develop the disease in the first instance was, therefore, not due to a natural refractoriness to the virus.

A few of the experiments recorded in this paper were carried out by Dr. Da Fano whose valuable help is gratefully acknowledged.

NOTES OF FURTHER EXPERIMENTAL WORK.

Since the above was written a further series of experiments have been carried out with the virus of herpes and some of the conclusions arrived at as the result of these experiments are here set out.

REACTION OF THE PRESERVING FLUID ON THE VIABILITY OF THE VIRUS.

At Prof. McIntosh's suggestion the reaction of the preserving fluid for virulent brain from cases of E.L. (50 per cent. glycerine in normal saline) was made roughly neutral to phenol-red by the addition of NaOH. In the case of the virus of herpes it was found that this medium when kept in contact with pieces of virulent rabbit's brain in the ice chest over long periods gradually became very strongly acid to litmus and yet under such conditions the virus was found to be still alive 95 days later, as the following experiment shows:—

Rabbit R. 50 received an intracerebral injection of an emulsion of virulent brain which had been kept for 95 days in 50 per cent. glycerine in normal saline, the latter being strongly acid to litmus at the end of that time (exact degree of acidity not determined).

The rabbit died six days later. A microscopical examination showed a typical encephalitis, and a further passage to another rabbit proved positive. It is interesting to note that Levaditi, Harvier and Nicolau (572c) found the virus of encephalitis lethargica still alive after being kept 96 days in milk in the ice chest.

In another experiment under exactly the same conditions, except that the virus had been so kept for 114 days in the ice chest, the rabbit (R. 71) made a good recovery and remained well for several weeks, but succumbed to a second intracerebral injection of fresh virulent brain.

On the other hand, if the brain of a rabbit which has died of an encephalitis is placed straight away into an acid medium the virus is found to be dead within 21 days. These experiments are not completed yet, but a medium of P_{11} , about 7.4, has been found to be the best preservative of the virus, provided the fluid is changed from time to time. It seems likely that under such conditions it will be possible to preserve the virus of herpes for much longer periods than has hitherto been the case.

ATTEMPTS AT IMMUNIZING RABBITS BY SUBCUTANEOUS INJECTIONS WITH THE KILLED VIRUS.

Four rabbits, R. 55, R. 56, R. 57 and R. 58, received every other day for one month 2 c.c. of an emulsion of fresh virulent brain in normal saline containing 0.5 per cent. phenol, the proportion being 6 grammes of brain to 100 c.c. of saline. At the

end of that time a small quantity of blood was obtained from the marginal vein of the ear from each of the animals and was allowed to clot. The clear serum was pipetted off and, without heating, was mixed with an equal quantity of a finely ground emulsion of virulent brain; the mixture was allowed to stand overnight in the ice chest and was then injected intracerebrally into new rabbits. All the latter developed the disease and died in five to six days, as the following experiment shows:—

On 1.3.22 rabbit R. 76 received an intracerebral injection of 0.3 c.c. of a mixture of equal parts of the fresh serum of rabbit R. 57 and a fine emulsion of virulent brain, the mixture having been kept overnight in the ice chest. At the same time a control animal R. 74 received intracerebrally the brain emulsion only. Both rabbits developed the disease. R. 74 died four days later and R. 76 six days later.

A microscopical examination of their brains showed an encephalitis and further passages were positive. No immune body to the virus of herpes was therefore present in the blood serum of rabbit R. 57. No conclusion can be drawn from the fact that the illness lasted two days longer in the case of R. 76 than in the control animal, as it is not uncommon to find rabbits living for six days during the ordinary passages of the unmixed virus.

The four rabbits which had been vaccinated were next tested with virulent brain. Two of them, R. 55 and R. 56, had an emulsion of virulent brain rubbed into the mucous membrane of the left nostril with an ordinary throat swab until a faint pinkish tinge of blood was seen on the swab. (This method had been found very suitable for intranasal injections of the virus.) They both developed signs of an acute vestibular lesion on the left side (this will be referred to again later on), but whereas R. 55 made a good recovery without developing an encephalitis, R. 56 developed an encephalitis subsequently to the occurrence of a lesion of the vestibular mechanism and died 12 days after the injection, a further passage from brain to brain in a fresh rabbit proving positive.

The other two rabbits, R. 57 and R. 58, received an intracerebral injection of virulent brain and they died six days and three days later respectively. A fresh brain-to-brain passage to new rabbits proved positive. So that, of the four rabbits which were vaccinated, three showed no evidence of being immunized, whilst in the fourth a vestibular lesion, but not an encephalitis, developed, the animal recovering ultimately.

IMMUNIZATION BY VACCINATION WITH THE LIVING VIRUS.

The abdomen of a rabbit, R. 91, was shaved and the skin gently scarified over a wide area with the blunted point of a scalpel. An emulsion of virulent brain in normal saline was

firmly rubbed into the scarified area and the animal put back into its cage: some small vesicles developed over the scarified area during the next three days and these quickly dried up and disappeared. Eleven days later this rabbit and a control one, R. 98, received an intracerebral injection of an emulsion of virulent brain: the control animal, R. 98, died four days later with the typical signs of an encephalitis, confirmed microscopically, but R. 91 developed no signs of any illness and is still alive and well several weeks after the experiment.

So far this is the only case in which an intracerebral injection of the virus has not been followed by the usual fatal result.

It is therefore possible to protect the rabbit by simple inoculation of the skin with the living virus.

Levaditi and his collaborators (572e) have recorded similar results in the case of rabbits which had recovered from an experimental herpetic keratitis and which survived a subsequent intracerebral injection of the same virus.

PRODUCTION OF A LESION OF THE VESTIBULAR MECHANISM IN THE RABBIT BY INTRANASAL INJECTIONS OF THE VIRUS OF HERPES,

In the course of an experimental encephalitis in the rabbit, as the result of an intracerebral injection of the virus, it happens sometimes that the animal shows a definite tendency to turn its head towards the side on which the inoculation has been made and then to rotate on itself always in the direction of the side of the inoculation. These symptoms occur also as the result of an intranasal inoculation.

In the latter case the animal shows a definite disturbance of equilibrium without lethargy or paralysis, it flexes its head towards the side of inoculation and, in advanced cases, its body is curved to one side or other so that the head may meet the hind limbs of the same side; also, the limbs of the opposite side are extended from the body, more especially the fore limb.

In one case, that of rabbit R. 55, referred to in the last paragraph but one, a complete 7th nerve paralysis of the same side and nystagmus developed at the same time as the disturbance of equilibrium and the animal lay completely on its left side for two days. It went on feeding as usual whilst lying on its side and gradually recovered its sense of equilibrium; the 7th nerve paralysis disappeared in another 10 days and the animal is quite well now. In the case of R. 56, which received an intranasal injection at the same time as R. 55, similar symptoms developed, but without the 7th nerve paralysis. In this case the animal developed an encephalitis subsequently to the lesion of the vestibular system and died 12 days after the inoculation. A further brain-to-brain passage into another rabbit proved positive.

Recovery from an encephalitis which is the result of an intracerebral injection has never been noted in the course of

these experiments, whilst confirmatory evidence of the symptoms being due to a lesion of the vestibular system is shown in the case of R. 55 which developed a 7th nerve paralysis at the same time.

In the case of a very young rabbit these symptoms were noticed 24 hours after an intranasal inoculation. Such an early onset suggests the possibility of the path of infection being the eustachian tube and the middle ear.

The complete histological and experimental proof that the symptoms described are due to a lesion of the vestibular mechanism are still lacking both in the intranasal and intracerebral cases, as the experiments are not completed yet.

Mr. G. J. Jenkins saw both the rabbits, R. 55 and R. 56, at the time that the symptoms recorded above were at their height and was of the opinion that the signs were those of a damaged vestibular apparatus on the side of inoculation. He thought it impossible to express an opinion as to which part of the vestibular system had been affected, but in the case of R. 55 with the 7th nerve paralysis there was a close analogy to the condition in the human subject associated with geniculate ganglion herpes. In this condition there are symptoms of a disturbance of the auditory-vestibular mechanism associated with 7th nerve paralysis and herpes of 5th nerve distribution.

FILTRABILITY OF THE VIRUS.

A large number of experiments have been carried out with a view to testing the filter-passing capacity of the virus of herpes.

Levaditi and Harvier (571f) have shown that the virus of encephalitis lethargica is capable of passing through Chamberland filter candles Nos. 1 and 3, and Blanc and Caminopetros (97) have succeeded in the case of the virus of herpes with a No. 1 candle. The question arises as to what micro-organisms can these candles arrest? Pasteur Chamberland candle L1 lets through all bacteria, whilst L3 is stated by the makers to "arrest the diphtheria bacillus and tetanus spores, whilst the other candles (numbers higher than 3) are true filters." It is clear, therefore, that L3 is not included amongst the true filters. The candles used in this series of experiments were tested with B. Prodigiosus. The filtrate of L1 contained as many organisms as the original suspension, L2 let a few bacteria through, but L3 let none through.

Up to now it has been found impossible to demonstrate the filtrability of the virus of herpes.

Very thin finely ground emulsions of brain were used, which were capable of producing an encephalitis by intracerebral injection in the control animals. It seems, therefore, that the virus clings very firmly to particles of organic matter with which it comes into contact and cannot be separated from it with any ease at all.

INTRANASAL INOCULATION.

Simple intranasal insufflation of an emulsion of virulent brain produces no symptoms of any kind in the rabbit, but if the virus is rubbed into the mucous membrane of the nose, symptoms of an encephalitis or of a vestibular lesion, or of both, appear generally within six days. The intranasal route yields a positive result in the majority of cases but not invariably.

COMPARISON BETWEEN THE INTRACEREBRAL AND THE INTRANASAL ROUTES.

An intracerebral injection of the virus of herpes into a new rabbit is always followed by symptoms of an encephalitis, which proves invariably fatal. On the other hand, after an intranasal injection by scarification, not all the animals develop symptoms of infection with the virus; of those that become infected recovery has only been noted once, when signs of a lesion of the vestibular mechanism and not of an encephalitis developed.

INTRAPERITONEAL, SUBCUTANEOUS AND INTRACESOPHAGEAL INJECTIONS.

Negative results have been invariably obtained by these routes. The animals which received the injections (1 c.c. of a thick emulsion of virulent brain) developed no immunity to the virus, as they all succumbed to a subsequent intranasal or intracerebral injection of the virus.

RESISTANCE OF THE VIRUS TO HEAT.

Exposure for half-an-hour to a temperature of 50° Cent. kills the virus of herpes, as the following experiment shows :—

Two young rabbits, R. 70 and R. 83, both received an intracerebral injection of 0·3 c.c. of a thick emulsion of virulent brain at the same time, but in the case of R. 83 the emulsion had been subjected to a temperature of 50° Cent. for half-an-hour in a water bath. R. 70 died of an encephalitis three days later, but R. 83 survived and showed no symptoms whatever.

APPENDIX I.

CLINICAL NOTES OF CASES.

Case A. No. 449/1919.—Full report of a typical case, apart from the hyperpyrexia, of encephalitis lethargica, by Dr. F. D. Deas, Merton, Surrey; including notes on the post-mortem findings by Dr. J. G. Greenfield, Pathologist, National Hospital, Queen Square.

E.T., aged 19 years, domestic servant. Well developed. Never had any serious illness. Family history good.

Patient was in good health up to 28th January 1919. On that day she was doing her domestic duties, and was perfectly normal. In the early afternoon she became suddenly paralysed on the left side of the face, and had ptosis of left eyelid. She was said to have squinted and had diplopia. She saw a doctor, who told her it was due to cold and would clear up. She continued her domestic duties during the next week, but her mistress noticed that she was getting very slow and dull—a marked contrast to her previous condition.

Patient was brought to me late on the evening of the 4th February 1919—the 8th day of the disease. I at once sent her into the Nelson Hospital, Merton. She was able to walk, but very slowly. Her mental condition was very dull. She had paralysis of left side of face—ptosis of left eyelid. There was no squint or diplopia. She could frown. There was no loss of sensation. When told to put out her tongue, she made the attempt very slowly, and did not get more than half an inch of the organ beyond the lips. Made no attempt to retract the tongue unless spoken to sharply. Speech very slow and voice a mere whisper. During the following week she gradually became more and more dull and lethargic. She could not protrude the tongue beyond the teeth. The facial paralysis remained as before, except that the ptosis improved. No paralysis anywhere else. Had control over bladder and rectum, but was very constipated. Could feed herself with fluids, but not solids. Could not masticate. Knee, wrist, and ankle jerks brisk. Plantar reflex normal—flexor. No loss of sensation. Voluntary movements very slow in starting and in execution. With arm extended in a line with shoulder, when told to touch the tip of nose with forefinger, had to be told several times, and sharply, before she made any attempt. Having started the movement, she took ten seconds to complete it.

The condition of the pupils was peculiar. They were unequal in size, but sometimes the one and sometimes the other was the larger. Both were very slow in reacting to light and accommodation, and under both stimuli they reacted unequally.

Up to this time—the 20th day of illness—the temperature had remained between 98° F. and 99° F. The pulse was regular, and ranged between 68 and 96 per minute. Respirations 18–22 per minute. Urine, Sp. G. 1014–1020. No albumen. No sugar. No deposit. Quantity 40–50 oz. per day.

On the 16th February (20th day of illness) patient had retention of urine, and this continued for four days—up to 24th day of illness—bladder being relieved by catheter. Probably this was not a true retention, but due to her increasing lethargy. Bowels only acted with enemata. From February 20th (24th day of illness) to the day of her death (23rd February) she had no control over bladder or rectum. She had gradually become more and more lethargic, and by the 20th February (24th day) she could not be roused to say any words which were audible, although it was evident that she understood what was said to her. She lay in bed, without the slightest movement, mouth slightly open, tip of tongue just showing. She could still swallow fluids, but with difficulty, but on 20th February (24th day) she could not swallow fluid, and from then onwards had to be fed nasally.

On 20th February (24th day) she developed a fine tremor of hands and eyelids, also a horizontal and vertical nystagmus. The plantar reflex on this day, and until the end, was peculiar in that it was flexor to begin with, but slowly changed to extensor. All other reflexes present, but response to necessary stimulus slow.

On the 20th February (24th day) the temperature went up slowly to 101° F. Pulse to 136 and respirations to 22 per minute. The urine became scanty; no albumen, no sugar—Sp. G. 1040, high colour, no blood.

On 21st February and 22nd (25th and 26th days of illness) the temperature gradually crept up to 106° F., pulse to 160, and respirations to 36 per minute.

On 22nd February (26th day of illness) patient was quite comatose, and passing everything involuntarily—could not make the least voluntary effort. In the evening of this day the temperature fell to 105° F.

On the morning of the 23rd February (27th day of illness) the temperature suddenly shot up to over 108·6° F., and patient died.

F. DEAS.

Post-mortem Report.

Pieces were taken from the medulla, pons, mid-brain, and motor cortex, and were stained with hæmatoxylin and Van Gieson's stain, Nissl's method and Gram's method counterstained with neutral red.

Medulla-Lower Part, Below the Olivary Nucleus.—Beyond slight thickening of the pia and the sheaths of the vessels, there is little abnormality. A few of the larger veins have a collection of small round cells in their adventitial sheaths, but this is only of slight extent and occurs in a small minority of the vessels.

Most of the ganglion cells appear normal; a few show eccentric nuclear and peri-nuclear chromatolysis.

Upper Part, Level of Olivary Nucleus.—Here, the small-celled infiltration of the vessel walls is more general though no greater in degree. The cells of the Xth and XIth nuclei are normal, but some of those of the XIIth show considerable changes, chiefly in the accumulation of pigment and powdering of the Nissl granules. It is remarkable that a perfectly normal cell is often seen close beside a cell which shows considerable alteration. There is a general increase in the neurological nuclei in this region, and numbers of the less healthy cells have round them several parasitic ganglial cells (neurophagy).

The cells of the olivary nucleus appear fairly normal, although perhaps they contain more pigment than normal.

Pons, Lower and Middle Portions.—Here the changes are slighter and less definite than in the upper part of the medulla. Only a few vessels show any infiltration of their sheaths and only a minority of the nerve cells of the central nuclei (VIth) show any neurophagy and increase of pigment. These changes, where seen, are not of very great degree.

Junction of the Pons and Mid-brain.—One or two of the smaller venules in the anterior part of the pons are thrombosed, apparently recently. The cells of the oculomotor nuclei and some of the cells posterior to these in the median raphe show considerable changes of the type described above. There is more neurophagy of the cells of these nuclei than elsewhere in the pons and medulla. Very little infiltration of the vessel sheaths is seen at this level, but there is a definite increase in the number of nuclei along the walls of the smallest capillaries. This change which is almost constantly found in "Encephalitis lethargica," is very patchy in character, some capillaries showing a row of closely placed nuclei, while others are normal.

Mid-brain, Upper Part, at the Level of the Substantia Nigra.—Here, the changes in the vessels are rather more apparent than those seen in the pons, especially in the region of the substantia nigra. Here one or two

small venules are seen filled with leucocytes, among which the polymorphonuclear type predominate. The increase of cells along the walls of the capillaries is more general here than in the pons, little strings of rounded nuclei being seen everywhere in the field.

The cells of the oculomotor nuclei and those of the substantia nigra show no definite alterations in their Nissl granules.

Cortex, Pre-Rolandic Area.—Here, the vascular changes are similar to those seen in the brain stem but of slighter degree. Several of the smaller veins show a certain degree of infiltration of their sheaths with rounded cells—lymphocytes and plasma cells. The Betz cells and the smaller cells of the cortex show no appreciable change in their Nissl granules.

No Gram-positive organisms were seen in any of the sections.

Case B. No. 212/1919.—History of a non-fatal attack of encephalitis lethargica in the case of a medical practitioner, compiled mainly from notes supplied by the patient himself.

"X," a medical practitioner of 49, was attacked on 25th December 1918 with vomiting and diarrhoea, which incapacitated him for five days; this gastro-intestinal illness could not be associated with any dietetic indiscretions, against which, at this time, the patient was particularly on his guard.

From now onwards he was never quite well; he continued at work, however, despite languor, pains in the back and limbs, as well as headache and earache. There was also a feeling of contraction down the anterior aspect of the body associated with a flexure of the head downwards and forwards.

Meanwhile his patients and members of his household had been noticing that his expression was altering, and that he had evidently some difficulty of vision. Early in January 1919 there was definite diplopia.

Dr. "X" carried on, however, until 11th February, when the disease unmistakably declared itself, and the patient retired to bed with a temperature of 101° F. The fever, which averaged about 101° F., remained for eight weeks, and the highest temperature recorded was 104° F. There was a much quickened pulse, the rate of which varied between 120 and 150; profuse and continuous sweating, and troublesome constipation. The urine was normal in every respect, and there was nothing abnormal found in the lungs or abdominal viscera. No rash.

These general symptoms were accompanied by various manifestations of mental disturbance and nervo-muscular disability.

(a) There was marked lethargy; patient lay like a log, in the supine position, with arms folded across the chest, legs fully extended, and face devoid of all expression. For weeks the voice was not raised above a whisper, but all questions and answers were quite normal as regards articulation. Dull and apathetic notwithstanding, the patient's condition never approached unconsciousness, and he spontaneously discussed the symptoms and treatment of his case, but during this time there was considerable confusion of ideas as remembered by the patient during his convalescence. Neither during the few outbursts of nocturnal delirium nor in his irrational thoughts and remarks did the patient "give himself away" or "live over again" in scenes of his professional occupation. Among the delusions recalled by the patient were the following: "Thought he was in an institution and was being changed from room to room"; "spending the night in a dock unloading vessels"; "with a dog in a country lane, where he was expected to curl up and sleep under a tree."

There were curious delusions concerning his own anatomical configuration. Thus, under the impression that he possessed more than one entrance to the pharynx, he would poke various parts of his face with the tooth brush in an attempt to find other mouths and attend to other sets of teeth; he imagined that he had several rows of eyes and a double set of finger nails. He associated his legs with those of a grand piano, so that

he pointed out to the nurse who was attending to the toilet of his limbs the apparent absurdity of the proceeding.

During the latter part of the patient's three months' confinement to bed insomnia was a troublesome feature.

(b) *Ocular Affections*.—Diplopia was a very early symptom, but no sign of strabismus was recorded. There was a double ptosis early in the course of the disease and this was preceded by conjunctivitis. The right pupil was dilated and twice the size of the left. Nystagmus was a later sign.

(c) *Other Paralysis*.—Weakness of muscles of right face, paralysis of right ala nasi. Paralysis of intercostals on both sides and partial paralysis of diaphragm. Retention of urine for five days. Dysphagia. Slight general tremors.

Progress.—Dr. "X" came downstairs on 21st May 1919, and on 13th June he went to Margate, where he was wheeled about in a bath chair for three weeks. He resumed general practice in September 1919.

Present Condition, December 1920.—The face has resumed its normal symmetry. Breathing is easy and regular. The sphincters function properly and there are no obvious tremors. Weight has been recovered and capacity for physical and mental work largely regained. Memory appears good and questions are answered intelligently and promptly. There is, perhaps, a certain deliberateness of manner and action, and a far-away expression at times; patient gives the impression of having passed through a severe illness. Dr. "X" himself finds that he is much more inclined to nervousness, and easily gets upset, while his emotions are much less under control. He "nearly always falls asleep at a concert or while in the theatre or church." Sexual desire is practically lost. In addition, Dr. "X" is subject to what he calls "paretic crises," in which there may be very troublesome dysphagia or stiffness and weakness of the right arm; at such times, too, and at others, there is excessive salivation and tremors. The power of writing has been greatly impaired, so that the hands need re-educating in this respect; walking, too, is not quite the automatic action it should be, and there is a tendency to drag the feet.

June 1922.—There is still some inequality of pupils, and diplopia often occurs.

Case C.—Autobiographical notes by a solicitor who suffered severely from *encephalitis lethargica* and continued at work during the early stages of his illness (ambulatory case). Privately communicated.

April 9th–29th.—Bad attack of influenza.

December 4th.—Feeling fit, but had a busy day in London and a rush to catch 6 o'clock train. Had dinner on arrival home and went out after to a lecture. In bed about eleven. Woke up about 1 o'clock with temperature, headache. Did not sleep again that night.

December 5th.—My sight began to go wrong. Everything I looked at seemed duplicated. I put it down to a liver attack. Went to bed early, but only slept a short time, and then woke up with headache and had no further sleep.

December 6th.—Did not go to work. Sight getting worse. Rested most of day, but only had a short sleep, and lay awake rest of the night with the headache the same as before.

December 7th (Sunday).—The double vision seemed less accentuated and had more sleep at night, but lay awake from about 2 to 6 a.m.

December 8th.—Went to London. Sight very bad. Could not read either with or without glasses unless I closed one eye. Came home and went to bed early. Slept very heavily and could hardly rouse myself in the morning to get up.

December 9th.—Slept at every available opportunity in the train and in office. Eyes practically useless; had to get clerk to read letters to me. Went to bed early and slept heavily, and had the same difficulty in waking up in morning.

December 10th.—Practically a repetition of the previous day. My clerk rang up my oculist and explained the trouble with my eyes, and he appointed to see me next day.

December 11th.—Could scarcely keep awake all day. Went to see G. He tested my sight and said he could do nothing, and advised me to see nerve specialist at once. He said he thought I had a new infection, which was very rare. I was too tired to go to see doctor that night, so asked him if he would write to my own doctor and I would see him the next day. My sight was now very bad, and it was only with difficulty I could keep awake.

December 12th.—I went to bed in the course of the day and the Dr. came to see me. I was now in a kind of stupor and practically unconscious. A London specialist and my brother came to see me and a nurse was put in charge. Relations also came to see me; but I knew nothing and lay practically in a stupor until, I think, December 26th.

December 26th.—From this date I gradually became less drowsy, and my vision by degrees ceased to duplicate the object. While in the stupor I felt nothing; no pain or any sensation. It was absolutely dreamless, heavy sleep. I believe my temperature was generally below normal and pulse erratic, but this I cannot speak of with certainty. The chief medicine I had was a drug Hexamine(?) When I stopped taking it the first and second times, my vision was again slightly affected. I had no paralysis.

January 27th.—I dressed for first time and went down for a few hours. I was very weak.

February 6th.—Went down to Budleigh Salterton and Wiltshire for a month. Much better on return, but have since developed a slight difficulty in speaking. Capacity for work just as good, but tire more quickly.

Case D. 37/1919.—Notes by Dr. J. H. Crocker, Richmond, concerning a case of *encephalitis lethargica* complicated by pregnancy.

Mrs. A., who was expecting to be confined in about a fortnight, did not sleep on Sunday night, the 12th January, nor on Monday night, the 13th January. On Monday, 13th January, she complained of a frontal headache and double vision; the double vision became gradually worse on Tuesday and Wednesday, but she was able to go down into the town and do a little shopping on Tuesday and Wednesday. Upon returning home she said she felt as if she was in a dream the whole time. On Wednesday night, the 15th inst., the nurse was sent for; pains began at 8 p.m.; the doctor and nurse arrived at midnight, and owing to the pains having gone off a little chloroform was given, and the case completed by 3 a.m. of the 16th inst. The child (second child), a girl, was alive and well. The patient became steadily worse, with cerebral symptoms, on the Thursday, and Dr. Buzzard was called in in consultation on the 17th and again on the 19th. A lumbar puncture was performed on the 19th; 4 drachms of fluid were removed and forwarded to St. Thomas's laboratory, the report eventually received being negative as regards any specific organisms.

At the request of Dr. Wadd, the medical attendant, I saw the patient on the 20th January at 5 p.m. The patient was lying on her back, shoulders and head propped up on pillows, head and face inclined towards left. There was not complete coma, and the patient would reply when spoken to more or less coherently. There was complete ptosis on both sides; slight external strabismus, right eye being turned slightly out and up, conjunctivitis, and both pupils contracted to about a large pin-head; photophobia. The facial muscles were paralysed, the mouth being open and lips retracted; loss of the naso-labial groove on the right side. There was a rash, which I was informed had only just appeared that morning, on the arms and thighs, very like erythema multiforme. No quinine had been given. The temperature was 103° F., and the patient looked very collapsed. She rallied during the night, and on the 22nd January her condition had improved to such an extent that she asked after the child.

and the brain seemed considerably clearer. At evening, however, the symptoms came on more severely, and from then onwards she became worse, getting into a comatose condition on the night of the 24th, and died at 9 a.m. on Saturday, the 25th. She has been seen by Dr. Buzzard five or six times, and his diagnosis in the first instance was that of encephalitis lethargica.

J. H. CROCKER.

Case E. 673/1919.—Report of a case by Dr. John Lambie, in which encephalitis lethargica was associated with scarlet fever.

The patient, H.K., was admitted as a case of scarlet fever to the Forest Hospital on 2nd January 1920. The case was notified by Dr. Smith, of Pleasley. The history was that the illness began on 19th December 1919, with headache, vomiting, and diarrhoea. On 25th December the patient had a faint rash on the chest, and Dr. Smith was called in. On 26th December he was allowed out, went to a football match and got very wet. The following day his face was puffy and swollen. On 30th December the doctor was called in again, and scarlet fever diagnosed.

On examination after admission I found a general anasarca typical of scarlatinal nephritis. I obtained a sample of urine, and found that blood was present, and a very thick deposit of albumen on boiling.

There was no peeling on the hands such as one would expect to find if the nephritis occurred at the usual time in the course of the disease. The boy was drowsy but no unusual mental symptoms were noted.

The usual treatment—blankets, hot water bottles, aperients, and abundant fluid—was adopted.

A few rhonchi were heard on auscultation of the chest.

On 5th January I noted more oedema but the skin was acting.

On 6th January a note was made that there was vomiting and incontinence of faeces and urine during the night, that the oedema was rather less, and that there were delirium and excitement. I also noted that speech was staccato in character.

On the 8th I found that the right arm was completely, as far as I could judge, paralysed. The right side of the face was also paralysed, the paralysis being more marked in the lower part of the face. No strabismus was observed.

The patient used the monosyllables yes or no in reply to all questions, even when such answers were quite unsuitable. He appeared to understand when I asked him to protrude his tongue, but he protruded it, apparently with difficulty, a little way and I was not certain of deviation, although it may have deviated slightly to the right. He performed the action of showing his teeth and closing his eyes, following my example in doing so. Perspiration was exceptionally profuse.

It was reported that he had difficulty in swallowing, and consequently had not taken nourishment well.

On the 9th he appeared to have improved. He seemed to be more intelligent, the oedema was less, the paresis of the face was less, and he was able to move the right arm.

The amount of albumen in the urine was also much less.

On the 10th I noted that the improvement was maintained and that eye reflexes were normal but the plantar reflex was extensor in type.

On the 12th the oedema had gone, and the mental condition was much clearer. The paralysis was rapidly improving. The patient's speech was remarkable, and, I think, characteristic. He still answered a hesitating "yes" or "no" to all questions, but when the question was insistently repeated he attempted another answer in a hesitating and drawing manner, but failed to give a sensible reply. For instance :—

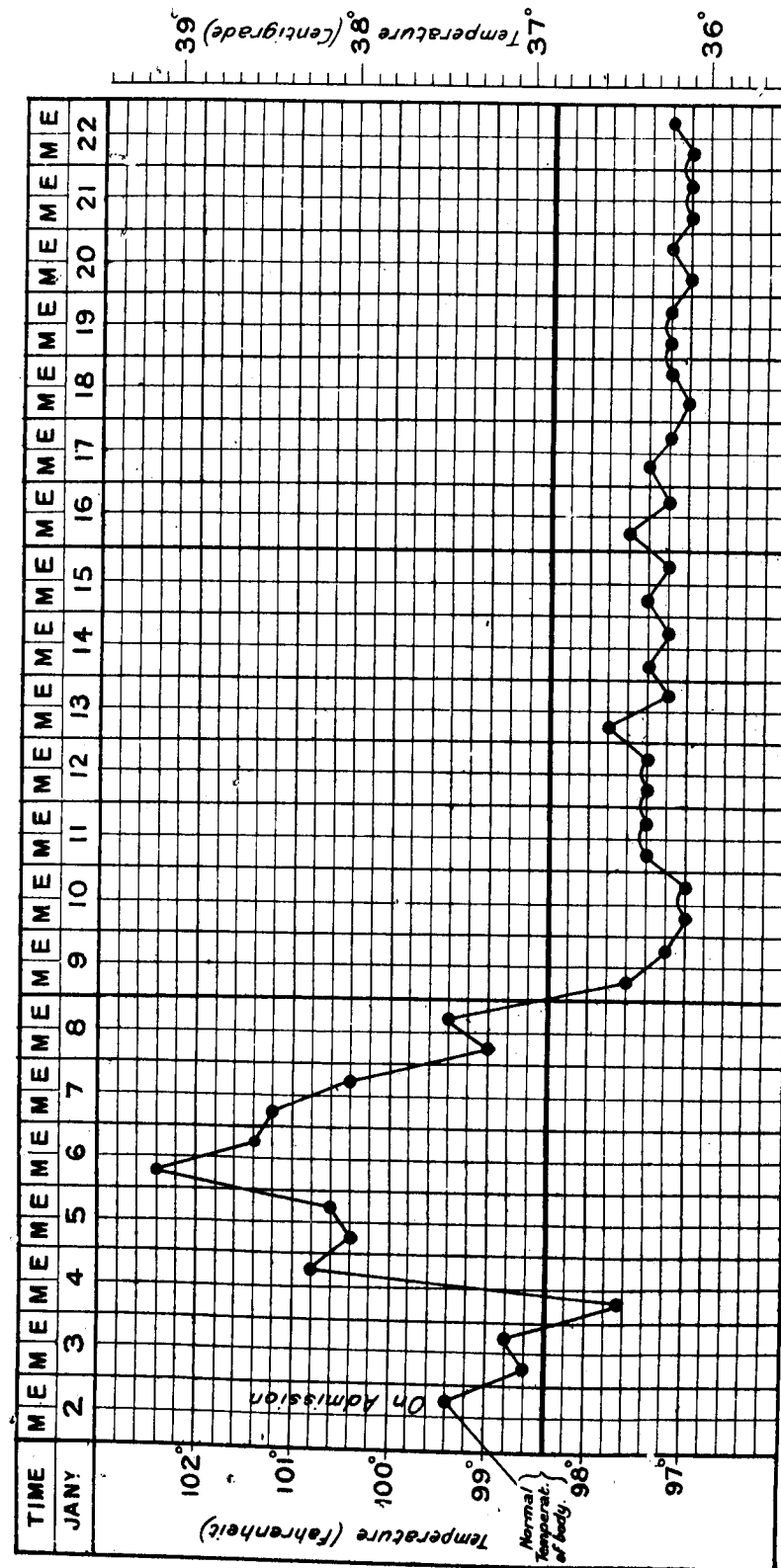
Q. "Where do you live?"—A. "Yes."

Q. "Where do you live?"—A. "I—I can't—get."

Q. "What do you want to eat?"—A. "Yes."

Q. "What do you want to eat?"—A. "Some—ah—ah—some—."

CASE E 673/1919.



This condition lasted several days.
On the 15th I noted a slight occasional convergent strabismus brought out when I asked him to follow with his eyes the movements of a pen held about a metre in front of his eyes. This was the only time strabismus was noticed. On this date I found his speech still hesitating. He was unable to say where he lived but gave correct affirmative or negative monosyllabic answers when questioned. He was able to name simple articles such as pen, key, and knife, but only after several attempts and with much stumbling.

When handed a paper he could name letters and short words such as "new."

On the 15th I found that his fingers were definitely peeling.

On the 17th the plantar reflex was found to be flexor. The paralysis appeared to be gone and the right grip as good as the left. From this date onwards there was a gradual improvement, but hesitation in speech was last noted on the 24th.

Hands and feet desquamated, so that there can be little doubt concerning the primary illness.

He was discharged apparently perfectly well on February 14th.

The case appeared to me to be one of encephalitis lethargica following scarlet fever. The unusually early appearance of nephritis obscured the diagnosis and masked the onset of encephalitis.

As the illness occurred in an infectious hospital, I am not sure that notification was necessary, but I felt that it was desirable to place the case on record. See copy of the temperature chart for the first three weeks.

JOHN LAMBIE.

Case F. 618/1920.—*Encephalitis lethargica in an infant.* Notes by Dr. A. G. Ledger, Shoreham-by-Sea.

D.H.B., born 22nd October 1919. Normal birth, fifth child, others healthy but said to have been puny babies. Father demobilised January 1919, in indifferent health, "gassed" November 1918. Mother had severe attack influenza November 1918. Mother got up 10th day after confinement and resumed full housework. Breast-fed baby.

24th November 1919.—Baby and mother came first under observation. The baby in fair health, said to be always crying. Mother in poor health, anæmic and debilitated. Treatment, tonic for mother and extra milk. Results good.

30th December 1919.—Baby, "cold in nose," and cough. Still tendency to much crying, but condition greatly improved. Inunction of mercury in small doses. Cold soon cleared up.

10th January 1920.—"Baby can't cry." Is lying still, face pale and expressionless. Can be roused and made to take some notice, and to grasp with fingers. Takes breast, relapses at once to sleep if left alone. No rigidity, no paralysis, no vomiting. T. 98° F. Fontanelle normal but pulsation feeble.

11th January 1920.—Said to have been squinting. Not apparent at visit. No change. Respiration very quiet and shallow.

12th January 1920.—Nystagmus. Coarse horizontal movements of eyes.

13th to 17th January.—Extreme somnolence with occasional intermissions. Will lie as long as left alone, eyes completely or half shut. No crying. Can be roused to feed. Neck very flaccid, breathing quiet and shallow. No pyrexia vomiting, nor constipation; no rigidity, tremors, nor paralysis, except on 14th January, transient partial facial paralysis (L.). No sweats, no eruptions.

18th January.—Marked twitching lips and alæ nasi, especially on left side. Doubtful Kernig's sign, no paralysis. More difficult to rouse. Breathing very shallow and a good deal of glairy mucus about mouth and throat. Apparently desires to vomit and to be prevented by asthenia. Evidence bronchial catarrh at bases posteriorly. Remains well nourished,

but muscular weakness more pronounced. Temperature 98°F. to 99°F. Reflexes: knee and biceps jerks obtained readily. Pupils do not react to light.

19th January.—Much less lethargy. No twitching. Moves limbs more freely, and face has slight pink tinge. T. 97°F.

p.m.—Improvement maintained and colour continues to improve. Pupils react to light.

20th January.—Further improvement in colour and sensibility. An erythematous rash on trunk. Evident but slight paresis of right facial muscles, neither pupil reacts to light.

NOTES ON CASES OF ENCEPHALITIS LETHARGICA IN WHICH Relapses OCCURRED.

Case G. 376/1919.—A.L.C., F., 18, engaged in household duties, was treated in St. Thomas's Hospital from 26.4.19 to 10th June 1919, when she was discharged cured.

Her case on this occasion was diagnosed as "encephalitis lethargica and epilepsy."

In April, 1919, the patient had influenza.

On, or about, July 2nd 1919, the same girl was noticed to be walking unsteadily; there was stiffness in the legs and difficulty in the swing of her limbs. By the 11th there were general pains all over her body, and noticeably in the limbs and ankles; stiffness was still present, giving rise to a staggering gait and helplessness. At times she was semi-unconscious, and later completely so. Understands questions but never speaks intelligently.

There was apparently no fever though delirium was noticed; speech became weak and indistinct. The "sight was affected" and ptosis was noticed; in addition, the right arm and side appeared to be paralysed, but there was no involvement of the face muscles. There was a progressive wasting and deafness.

She was admitted to the South Eastern (M.A.B.) Hospital on July 15th, when she is reported to have been "very drowsy, talks with difficulty. No eye symptoms. Reflexes all normal. Some incontinence. Slow improvement. August 2nd quite rational. August 12th epileptic fit. Later: Very slow gradual progress. Bed sores formed on both heels."

Case H. 946/1920.—C.Y., a plumber, aged 57, suddenly developed diplopia in November 1918 and continued to "see double" until February 1919.

About 14th March 1920, he manifested influenzal symptoms, and on the third day of this illness diplopia recurred together with facial paralysis and deafness in the left ear. He apparently got well again and resumed his work until April 10th, when he complained of a bad headache, again diplopia and feverishness. He was admitted to St. Thomas's Hospital on 22nd April, and there treated as a case of encephalitis lethargica. There was no lethargy and the mental condition was normal. The diplopia was associated with a paresis of the left external rectus and there was slight nystagmus in all directions; facial paralysis was complete and there was still deafness in the left ear. Fourteen days after onset there was loss of power in left arm and slightly in left leg, preceded by uncontrollable movements. On the left side plantar response was extensor, all the other reflexes were normal. Both the blood and cerebro-spinal fluid gave negative Wassermann reaction. Discs normal.

Case I. 2391/1920.—R.O.Y., a schoolboy, aged 14, was attacked early in February with pain in the head, eyes, and left ear. He was removed to the school sanatorium on 7th February, when his mind was not clear, though the temperature was no higher than 99°F. He was lethargic but could be roused, his memory was bad, and he could only recognise his mother. The pupils were dilated and there was paralysis of the left side

of face. He went home to Chingford on 24th February and remained apparently quite well until 3rd March, when he became restless and then semi-conscious. Admitted to Guy's Hospital on 7th March, he was treated as a relapse case of encephalitis lethargica. Lumbar puncture was done twice and afforded relief, and in addition, he was treated with cystopurin. He returned home once more on March 23rd, and was described on May 6th as "perfectly well and back at school."

Case J. 411/1919.—T.J., a commercial traveller, aged 41, who had been working at high pitch and was affected by domestic grief, was suddenly taken ill at Bridgwater on July 3rd, 1919. He then suffered from faintness, giddiness, headache, and diplopia. Of these symptoms he "partially recovered and was practically well" when he went to stay with relatives at Birmingham early in August 1919. Here he was again suddenly "struck down" while gardening, with vertigo and faintness, so that his relatives considered he was suffering from a "stroke." He was seen by a medical man on August 9th, who found no evidence of any paralysis; there was still vertigo and the blood pressure was 148. Drowsiness supervened, and by August 18th the patient had become comatose, with incontinence of urine and inability to swallow. Rectal feeding became necessary, but consciousness was never regained, and the patient died on September 3rd, 1919.

NOTES, BY THE LATE DR. W. F. BOWEN, ON FIVE OF THE *mild cases of ENCEPHALITIS LETHARGICA OBSERVED among children in LAMBETH DURING THE EARLY PART OF 1921.*

Case.	Age.	Sex.	Date of Onset.	Length of Illness.	Symptoms.
(K.) G. S.	—	M.	9/2/21	7 days	10th.—Ill one day. Very hot and sleepy. No difference day or night. No headache. Very weak in limbs. No squint. No vomiting. T = N. 11th.—T = 102° F. (Ax.) Slept only two hours yesterday. Very restless during night. Pain and tenderness in neck. Weak and tottering when on legs. No sickness. No squint. No nasal catarrh. Bs. = 1. 12th.—T = 102° F. in groin. Frontal headache only when other children making a noise. Sleeps more during the day, restless at night. Pain in thick part of thigh. Jumps when asleep, and is delirious. No vomiting, no squint. Slight catarrh. 16th.—Child well.
(L.) C.	5	F.	10/2/21	6 days	10th.—Ill during night. Headache. Pains in pit of stomach. Drowsy day and night. Pains both legs. Vomited this morning. Twitching of hands. Very constipated for two days. Frequency and incontinence of urine. Nasal catarrh. T = 102·6° F. 12th.—T = 104° F. Frontal headache. Very restless at night, shouting about play at school. Asleep all day. Pains in shoulder and stomach. Vomiting everything taken. Constipated three days. Twitching of hands. Eyes appear fixed. 13th.—Child brighter. T = N. 16th.—Child well.

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(M.) E. K. H.	5	M.	11/2/21	6 days	
(N.) E. M. H.	5	F.	12/2/21	10 days.	
(O.) A. R. B.	3	M.	14/2/21	—	

11th.—Drowsy at school, vomited then, but not since. Continual frontal headache. T = 101° F. (groin). Continually pushing back hair and holding head. Very sleepy since illness began, chiefly by day; restless at night. Pain in stomach and chest. Very weak and shaky in legs.
13th.—Said he "saw mummy twice," but no subsequent diplopia. No constipation. No twitchings, except in legs, with cramp. Troublesome cough, no pains in chest. Flushed, slight pharyngitis with enlarged glands. Tenderness in epigastrium.
17th.—Child quite fit. T = N.
18th.—Very feverish since onset. Headache. "Drops about anywhere with hand to forehead, and goes off to sleep." No occipital headache. Much more sleepy in daytime, but restless at night. Asks continually for rags. Weakness of legs. No squint. Vomited once. Anorexia. All family have had colds for about six weeks.
19th.—Much better. T = N.
20th.—Better, but still lethargic.
21st.—Better. T = N.
18th.—Frontal headache. T = 102·2° F. (groin). Drowsy day and night. Weakening of limbs, unable to stand. Usually very active. No squint, but twitchings of eyelids. Vomiting after all food. All household have had colds lately. Cat turned out of house on account of sneezing.
20th.—Better on the 19th, but worse to-day. T = 103° F. (groin). Drowsy. Frontal headache. Herpes on legs. Unable to stand. Twitchings of arms and legs when asleep. No vomiting since 18th.
21st.—T = N. No further twitchings. No vomiting. Very weak in limbs.
22nd.—Still drowsy, but T = N.

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NOTES ON CASES WITH CEREBRAL SYMPTOMS IN WHICH THE DIFFERENTIAL DIAGNOSIS PRESENTED SPECIAL DIFFICULTIES.

Case P.—*Case of Tuberculous meningitis suggesting, in turn, typhoid fever and encephalitis lethargica. Reported by Dr. Cyril Banks, Stafford.*

C.F., age 31, Leather bag dealer. Formerly in regular Army.

Previous History.—Trench fever 1918, chronic otorrhœa (Rt.) since infancy, operation (via meatus) 1918. Since then he has had no ear trouble whatever. During the past year he has had occasional sore throat with transient glandular enlargements in neck.

Present Illness.—He visited Liverpool on 4th October in search of work; but so far as is known he never entered any building, the offices being closed; he did not go into any café. He returned same day. Up to this time his health was good, and he was robust and well nourished. A few days later he seemed rather unwell,—fatigued and listless. On 11th October he consulted Dr. Mitchell for sore throat. On the evening of 14th October, Dr. Jones (for Dr. Mitchell) saw him; he had a temperature of 103° F. and he was delirious. Next morning, conscious, and temperature 101° F. He improved until October 18th when he had temperature 101° F. and was delirious almost to violence. Typhoid suggested by Dr. Marson, but blood returned "negative," 20th October. Seen by M.O.H., 19th October, lying quietly in bed, placid, eyelids drooping. Responded to commands. No paralysis. K.J. present, not exaggerated. Fumbling with fingers on edge of sheet. 20th October.—Urine, no sugar, no albumen. Since then he has lain quietly in bed and seemed to be getting no worse.

Seen by M.O.H., 25th October. Lying in bed stuporous. Left eyelid drooping. Left pupil much larger than right. Left arm helpless. Legs both retain power of movement. On being handled he moved his right hand to feel the person examining him. Passing urine and faeces below him: (he was constipated, but the faeces thus voided were after-results of erema treatment). Able to swallow fluids.

Special symptoms.

Vomiting.—Vomited before seeing Dr. Mitchell on 11th October, not since.

Headache.—In early stages, frontal chiefly, occasionally occipital.

Ear trouble.—No complaint during this illness.

Stiffness of neck.—None.

There has been no muscle-group rigidity or spasm. No fits.

Pulse.—Slow and regular—56–60.

Cheyne-Stokes breathing noticed occasionally during past week.

Irritability.—This does not appear to have been noticed. On the contrary he has been languorous, stuporous, and finally comatose.

He has never given the "meningeal cry."

Rash.—None reported.

25th October.—Admitted to Stafford Borough Isolation Hospital as notified case of encephalitis lethargica.

Diagnosis.—?Temporo-sphenoidal abscess resulting from old middle-ear disease. ? Encephalitis lethargica.

26th October at 4.30 a.m. the condition changed very remarkably. The pulse from 56 rose to over 100 and by 10 a.m. was uncountable. Temperature from 99·8° F. at 6 a.m. rose to 101° F. at 10 a.m. and 103·4° F. by 6 p.m. He became deeply comatose. Breathing stertorous (Cheyne-Stokes at times). Double divergent squint. Right pupil very small, left dilated. Knee jerks, right slight, left increased. Kernig's sign present both sides. No ankle clonus. Tendon reflexes in arms:—triceps present both sides, supinator absent both sides.

Death ensued, 12.55 a.m., 27th October.

Post-mortem (head only).—Dura mater firmly adherent to brain over an area size of a shilling at vertex immediately to the right of the longitudinal sinus. Superficial veins on both hemispheres much engorged with blood. The whole surface of brain somewhat pink and inflammatory. No pus on membranes. The temporal bones appeared normal, no signs of any inflammation of bone. On section the brain tissue was very soft, especially around the lateral ventricles. Both lateral ventricles greatly dilated and full of clear fluid. No pus found anywhere in hemispheres or in cerebellum. About the optic *chiasma* the membranes were matted and infiltrated with serum somewhat fibrinous. This condition extended into Sylvian fissures, and in the left Sylvian fissure four or five small but definite tubercles were found.

Cause of death.—Tuberculous meningitis.

CYRIL BANKS,

Medical Officer of Health,
Stafford Borough.

Case Q.—*Case of tuberculous meningitis in an infant who showed certain clinical signs of encephalitis lethargica: the value of cerebro-spinal fluid examination is exemplified in this instance.*

L. N., a child aged one year and seven months, had been ailing for about a month, and was taken by its mother to a hospital, where apparently its condition was not then considered serious.

On 15th September 1920 the child was convulsed, and thereafter became drowsy, so that on 26th September it was admitted to another hospital as an in-patient, with a provisional diagnosis of encephalitis lethargica. The drowsiness deepened into coma which was almost continuous, and there was pyrexia. No ocular symptoms had been noticed while the child was at home, but soon after admission to hospital slight ptosis was noticed, and there was an intermittent external strabismus.

A left hemiplegia also developed, and was accompanied by general tremors. A lumbar puncture was made while the child was in hospital, and the cerebro-spinal fluid was drawn off under pressure; Fehling's solution was not reduced, albumen was present and there was a leucocytosis, the lymphocytes being twice as numerous as the polymorpho-nuclears.

At the post-mortem examination miliary tubercles were found along the Sylvian fissure and in the choroid plexuses. The tubercle bacillus was also isolated from the spleen.

Case R.—*Another case of tuberculous meningitis in a young subject, which was at first supposed to be one of encephalitis lethargica.*

G. H., a male child nearly three years old, had been dull and otherwise "off colour" for a week or so when, at the end of February 1921, he was attacked with copious vomiting lasting for three days. On 2nd March

he became drowsy; and complained of headache in the frontal region; he was admitted to hospital. The temperature varied between 100° F. and 102° F. till the day before death, when it rose to 104° F. The pulse was rapid throughout. Vomiting continued for two days after admission, and there was constipation. Patient lay perfectly quiet, always sleeping with muscles relaxed, and with free movement of the head; there was no Kernig's sign. Ptosis of the right side, and slight squint left eye; incontinence of urine; twitching of face muscles. There were no physical signs in the chest. Died in a state of coma 11 days after admission. Microscopically the brain tissue showed no evidence of encephalitis lethargica, but very numerous tubercle bacilli were found.

Case S.—Case of tuberculous meningitis in a young adult in which a provisional diagnosis of encephalitis lethargica was first negatived after examination of the cerebro-spinal fluid, and finally disproved by the post-mortem discovery of tubercle bacilli in the brain substance.

A. D. W., a boiler-maker, aged 20, had been feeling ill early in November 1920, and on the 5th of the month complained of headache, lassitude and abdominal pain. There was no vomiting throughout the illness and little pyrexia (N. to 99·5° F.). There was progressive lethargy, patient was unable to answer questions, and during the last five days of his illness there was incontinence of urine and faeces. The left eye squinted, and there was slight nystagmus. Occasionally the patient became restless, and the limbs were subject to tremors; there was some rigidity of limbs and neck. On 18th November the patient became less torpid and attempted to speak, the attempt failed, however, owing to paralysis of the muscles of phonation.

The cerebro-spinal fluid was found to be slightly turbid, with a decrease of sugar but an increase of globulin; marked lymphocytosis with some endothelial cells. Patient died on 22nd November 1920, and Dr. Da Fano demonstrated the presence of tubercle bacilli in the brain substance. Patient's mother died two years ago from influenza, but there was no history of family tuberculosis.

Case T. 1049/1920.—Cerebral tumour in a woman of 43 giving rise to symptoms which were thought to be due to encephalitis lethargica.

Mrs. E. T., æt. 43, began in January 1920 to get cerebral symptoms, which consisted of violent headaches, shivering attacks, and ocular palsies; the predominating feature of the case was a stupor which was present from the start and at times was severe.

These symptoms fluctuated in severity, and the last "relapse" occurred in June 1920, when the patient was admitted to hospital for treatment. At this time a ptosis of the right eyelid was noticed, but this paralysis cleared up later; no squint and no nystagmus.

Similarly, a right-sided facial paralysis developed on 24th June 1920 but disappeared on the 9th July 1920. During the exacerbations control of the sphincters was lost.

The patient's mental condition was impaired, and speech was "slow"; at times she "talked foolishly."

There was marked swelling of the discs; no skin eruption.

The cerebro-spinal fluid was not under marked pressure and contained but a few mono-nuclear cells; sugar and albumen were present. Blood examination revealed nothing abnormal. The patient had suffered from influenza in the autumn of 1919, but otherwise there had been no other possible predisposing illness.

The case was notified in June 1920 as one of encephalitis lethargica, but before the fatal termination took place, and as a result of prolonged observation, cerebral tumour was diagnosed. This revised diagnosis was confirmed at the autopsy when a new growth in the frontal region was discovered.

Case U.—Another case of cerebral tumour simulating Encephalitis lethargica.

H. W., a man aged 53, became ill (whether suddenly or not is unstated) on 26th May 1919. The physical signs consisted of a subnormal temperature, lethargy, paralysis of left external rectus muscle, paralysis of left face, and paralysis of the left arm and leg. There was delirium on one occasion, and patient could be roused from his lethargic condition; speech was described as "slow."

The mental condition, together with the face and eye palsies, improved, but the arm and leg remained powerless. The man was admitted on the 14th June 1919 to a general hospital, where he died in the following August. At the post-mortem a cerebral tumour was found.

Case V.—Case of cerebral hæmorrhage, at first considered to be one of encephalitis lethargica.

T. B., a baker, aged 58, had been feeling "seedy and out of sorts" for a fortnight, when on 6th July he was suddenly taken ill with vertigo and vomiting. He became collapsed, and it was found that he was paralysed in the left arm, both legs being normal.

The left arm, from a condition of complete paralysis, changed into one of fairly free movement in two days.

The left half of the face was said to be paralysed; no ptosis or squint, but an occasional upward deviation of both eyes.

At night there was delirium and mental excitement, but from 4 a.m. till 4 p.m. the patient was lethargic, lying quite still, and taking little notice of his surroundings. Tremors were not noticed. For the first week after the arm became paralysed speech was perfect; later it required an effort for the patient to speak plainly, but no slurring was noticed. There were no inequalities of pupils; reactions to light and accommodation were constantly varying.

Three weeks after onset the man was removed into hospital where paresis of left arm and unequal pupils were noted.

The mouth was twisted to the left, and the tongue also deviated to the same side. The mental condition was clear, but articulation was difficult.

The blood pressure was very high and the arteries thickened. The man was discharged five days after admission as a case of hemiplegia due to cerebral hæmorrhage.

Case W.—Case in which a diagnosis of encephalitis lethargica was subsequently changed to one of pneumococcal meningitis.

P. H. H., aged 17, an engine cleaner, complained of pain and a slight swelling on nose on the 13th November 1919. His head began to ache on the 17th and the pain became worse though intermittent in character. On the 22nd he gave up work as he felt drowsy, and by the 28th there was loss of power and increased drowsiness. He was unable to feed himself on the 29th.

There was no pyrexia found on the occasion that the temperature was taken.

There were no eye symptoms except a nystagmus that began on 1st December 1919. There was weakness of the left orbicularis muscle, however, as well as of the left face generally.

The patient was dull and lethargic but was easily roused. Speech was slow but distinct, and there was no deafness. The knee jerks were markedly increased. No history of vomiting or of neck rigidity.

While in hospital the cerebro-spinal fluid was examined and was found to contain a diplococcus similar in appearance and staining to the pneumococcus; it was not the diplococcus intracellularis. Death occurred soon after admission to hospital in December 1919.

Case X.—Encephalitis lethargica and catalepsy.

M. P., a woman of 49, and a chronic alcoholic, after having been strange in manner for two weeks (during which time she was also irritable and listless) was suddenly taken ill while shopping on 2nd January 1919. Walking was accomplished with difficulty, and after being led home she was found to be incoherent and delirious. The next three weeks were spent in bed at home, and it was reported that during this period the previous physical lethargy had given place to pronounced mental lethargy.

The patient very occasionally talked coherently for a short time, but "otherwise the powers of the higher brain centres seem completely in abeyance." She lay motionless with eyes fixed in one direction taking no notice of her surroundings, and from this state it was impossible to rouse her. At times, however, there was a "barking cry" at regular 15 seconds intervals resembling the "hysterical cries," and occasionally there were fits of raving. Speech, when present, was slurred and difficult to follow. There was a "possible suggestion" of facial paralysis, but no ocular palsies were noted, while in the case of the sphincters there was paresis but no paralysis.

The muscles of the trunk, limbs, jaw and the extrinsic muscles of the eyes were in a state of spasm; it was impossible to close the mouth and the feet also were hyper-extended.

The trunk, limbs, &c., remained in any position in which they were placed. At one stage there was considerable rhythmical movement of right arm; the knee jerks were exaggerated. At the end of January the patient, who had become very emaciated, was removed to an isolation hospital, and subsequently to the county asylum, as a case of catalepsy. She was subsequently reported as having made a complete recovery.

Case Y.—Encephalitis and acute nephritis.

W.W., an ex-soldier, was demobilised in March 1920 as fit, and apparently gave a negative history of any previous illness. On 11th June he consulted a doctor on account of rheumatic pains in extremities which had been troubling him for a fortnight. Medical examination showed an obstinate constipation due to intestinal paralysis; the temperature when first seen was 102° F., rising to 103° F. in the evening, and the pulse rate 80-90; ptosis was noticed, and also lethargy, which increased daily, though the patient could be roused by shouting. At times he recognised people, while at nights there was generally delirium, especially when the temperature was high. The knee jerks were exaggerated, and both Babinski's and Kernig's signs were present, the latter from the commencement. No oedema of face, arms, or leg.

On the 17th the man was admitted to hospital, where it was recorded that: "On admission patient roused with difficulty. Temp. 102° F. Pulse 118, Resp. 36. Sweating a little. Complained of general headache. Subsultus tendinum. Abdomen much distended. Bladder apparently

full. Right base percussion note diminished. Some sticky crepitations heard all over right side of chest. Catheter passed, and 120 oz. of urine withdrawn." On the 18th conditions much as before; 60 oz. of urine were withdrawn, and the specimen showed marked albuminuria. There was no rash, and an examination of the fundus showed no congestion of the discs. Ten c.c. of c.-s. fluid were withdrawn by lumbar puncture; there was no excess of pressure, the fluid was clear, and on being centrifuged showed nothing abnormal. Cultures were sterile after 48 hours. There was no improvement after lumbar puncture, but the temperature rose to 108° F., and the man died on 18.6.20.

At the post-mortem examination signs of acute nephritis and bronchopneumonia were found.

APPENDIX

A. TABLE SHOWING CIRCUMSTANCES OF SOME 50 CASES IN WHICH

Case.	Notification, number.	Initials.	Age.	Occupation.	Condition of woman when attacked with encephalitis lethargica.	Date of onset of E.L.
1	Not notified	?	28	?	Near full term; labour induced 7.4.18.	29.3.18
2	196	K. M.	25	Domestic servant.	Pregnant at time of death; (married 6.4.18).	Last at work 12.11.18
3	9	F. O.	28	Widow	Near full term; confined on 29.12.18.	9.12.18
4	240	E. H.	31	Housewife	Pregnant six months	Jan. 1919
5	37	H. G. R. A.	29	Housewife	Within two weeks of full term; child (2nd) born 16.1.19, three days after onset of encephalitis.	13.1.19
6	121	A. A. E. L.	29	Housewife	Infant born four days after onset.	20.1.19
7	340	Mrs. P.	33	Housewife	Eighth month of pregnancy. Child born six weeks after onset	Jan. 1919

DIX II.

ENCEPHALITIS LETHARGICA WAS ASSOCIATED WITH pregnancy.

Main features of attack.	Result to mother.	Result to child.	Remarks.
Sudden onset	Recovered.	Baby subsequently exhibited mild signs of E.L. and eventually recovered.	Reported by Dr. Wilfred Harris (<i>Lancet</i> April 20, 1918). Cf. instances 23, 31, and 45 in this Table.
Sickness, drowsiness, legs paralysed. Delirium at first, very nervous. Previously anæmic.	Died 24.2.19	—	—
Left side of face drawn up, then partial loss of use of right hand. Severe headache, which increased after delivery. Vomited. History of two fits in street previously. Lethargy. Slight ptosis both eyes.	Died 13.1.19	Infant showed signs of congenital syphilis.	Cerebro-spinal fluid free from micro-organisms.
Headache, drowsiness, extreme lethargy, ptosis. Incontinence, delirium.	Died 2.2.19	Fœtus living until woman's death.	Taken ill at sea while returning from Gibraltar; no similar case on board. Cerebro-spinal fluid normal; Wassermann negative; no albuminuria.
Diplopia, headache, right facial paralysis, stupor, tremors of arms occasionally, speech thick and difficult. Ptosis, squint, and nystagmus. Rash very like erythema multiforme. Cerebral symptoms gradually became worse from 16.1.19.	Died 25.1.19	Child alive and well.	Case seen by Dr. Farquhar Buzzard.
Lethargy, facial paralysis, and nystagmus. Paralysis of larynx at end.	Died 7.3.19	Subsequently suffered from pneumonia; alive and well on 10.10.21.	—
Pain left arm, irritable. Marked lethargy. Ptosis and nystagmus. Choreiform movements of facial muscles (both sides), also of whole body, especially arms.	Slowly recovering.	Child died when 10 days old.	—

APPENDIX II.—A. Table showing circumstances of some 50 cases in which

Case.	Notifica- tion number.	Initials.	Age.	Occupation.	Condition of woman when attacked with encephalitis lethargica.	Date of onset of E.L.
8	Not noti- fied.	A. M. P. -	35	?	Pregnant seven months.	Feb. 1919 -
9	85	E. R. -	18	Draper's as- sistant (out of employ- ment).	Confined 22.2.19, two days before onset of encephalitis.	24.2.19
10	142	B. R. -	31	Housewife -	Confinement Feb. 1919.	Notified 20.3.19
11	493	M. I. J. -	30	Housewife -	Pregnant five months.	10.11.19
12	964	V. S. -	22	Housewife -	"Patient was enceinte and operation was performed which brought on prema- ture birth (six months)."	6.12.19
13	742	Mrs. W. -	17	Housewife -	Pregnant eight months.	11.12.19
14	604	A. J. -	28	Housewife. 'Bus con- ductress ten months pri- or to at- tack.	Near full term; con- fined 12.1.20, three weeks after onset.	22.12.19
15	623	M. M. -	27	Housewife -	? Miscarriage two days before onset.	31.12.19
16	780	D. S. -	24	Factory hand.	Premature confine- ment (followed by curetting) some time previous to onset.	5.3.20

Encephalitis Lethargica was associated with pregnancy—continued.

Main features of attack.	Result to mother.	Result to child.	Remarks.
Began with pain in left arm and "twitching all over." Involuntary movements chiefly involved left shoulder and elbow, also trapezius and pectoral muscles. Unconscious for some days; slight diplopia at one time.	Discharged from in firm ary 6.11.19. Scarcely able to walk and "on the twitch" all day long.	Miscarriage -	Reported by Buzzard and Greenfield (Brain, 1919, xlii, 311).
Headache, irritability, in- creasing lethargy, ptosis, fever, occasional twitch- ings and facial tremors.	Recovered -	Healthy infant -	Labour of short dura- tion, no complica- tions.
Severe cranial and facial neuralgia. Marked leth- argy, dull. Fine tremors of hands. Speech slow and forced.	Quite well on 29.9.21.	Child normal -	—
Insomnia, sore throat, enlarged tonsils, partial coma, tremors, ptosis, spasm of jaw muscles, fever and vomiting. De- lirious towards end.	Died 25.11.19 -	—	—
Pains in stomach, malaise, "sleepy," mouth drawn aside slightly, paresis foot and leg. Semi-conscious for three weeks.	Returned home 29.3.20.	—	Engaged at munitions during 1916.
Sleeplessness few nights. Lethargy marked. Mem- ory defective. Involun- tary rhythmic contractions left arm and leg.	2.2.21 permanent paralysis, tre- mors.	Lived two days	Confinement end of 8th month.
Lethargy and mental ob- tuseness; facial paresis.	Recovered -	Quite unaffected	"Nothing to suggest placental, renal or hepatic toxæmia."
Sudden attack of sleepiness followed by marked leth- argy, fever, ptosis and squint. Some delirium at night, coarse tremors.	Died 19.1.20 -	Miscarriage -	—
Bronchial catarrh; progres- sive lethargy, paralytic motor symptoms arm and hand, squint, ptosis, slurred speech. Transient face palsy.	Improving -	—	—

APPENDIX II.—A. Table showing circumstances of some 50 cases in which

Case.	Notifica- tion number.	Initials.	Age.	Occupation.	Condition of woman when attacked with encephalitis lethargica.	Date of onset of E.L.
17	823	F. G. P.	29	Housewife	Nursing her baby, born 11.1.20.	27.3.20
18	809	A. A.	37	Housewife	Pregnant eight months.	1.4.20
19	1015	Mrs. S.	38	Housewife	Pregnant, nearly full term; confined 5.5.20.	14.4.20
20	899	C. B.	?	Housewife	Pregnant seven months.	17.5.20
21	984	M. B.	33	Housewife	Pregnant . . .	26.6.20
22	1079	M. A. P.	30	Housewife	Pregnant six months	4.8.20
23	1254	E. H.	28	Housewife	Confinement due; child born 28.10.20.	25.10.20

Encephalitis Lethargica was associated with pregnancy—continued.

Main features of attack.	Result to mother.	Result to child.	Remarks.
Typical signs of influenza at first, then symptoms of pneumonia. Loss of taste, fever, lethargy, thickness of speech, delirium, and loss of sphincter control. Coryza and cough, fever, headache. Delirious, som- nolent, facial twitchings. Onset of lethargy 11.4.20; slurring speech. Pain left shoulder and hand. Lethargy on 29.4.20; fa- cial paralysis. Nocturnal delirium.	Died 6.4.20	Child remains healthy and well 3.10.21.	Encephalitis second- ary to pneumonia.
	Died 13.4.20	Unborn . . .	Urine normal.
	Died 6.5.20	?	Slight trace of albu- men; no retinal changes.
Drowsiness, diplopia, weak- ness of legs and tremors. Deviation of tongue to right side 3.6.20. Knee jerks exaggerated.	7.2.21. Has deve- loped myopia. Lack of control; tremors left arm. Insomnia.	—	No albumen, no sugar.
Marked lethargy, diplopia, facial paresis.	Recovered.	—	Troublesome miscar- riage 10 months pre- vious to onset of encephalitis.
Well-marked "lucid som- nolence"; ptosis, slight nystagmus; erythematous eruption on abdomen and chest. Paresis of ocular muscles. Constipation. Occasional tremors.	11.2.21. Diplopia at times. Right eyelid droops when tired, twitching of eyelids, head- ache.	—	Diacetic acid in urine.
Lethargy, headache, squint, facial paralysis, sore throat, diplopia, ptosis, tremors, delirium.	Jan. 1921. Slight ocular paralysis, left side paresis. Clonic spasm right leg.	Died after five weeks, probably from a similar affection.	Baby weighed 7½ lbs. at birth, but the ex- perienced nurse in attendance did not consider it a normal infant, and it was "never strong"; there was no crying. Fed partly by bottle and partly with milk drawn off from mo- ther. First sign was a ptosis of right upper lid together with right-sided par- alysis of face. There followed cyanosis of finger-tips and lips; sweating; loss of weight; general twitchings; uncon- sciousness and death. No meningeal ery, no fever; no rigidity and no history of T.B. (Dr. James Pirie's case.)

APPENDIX II.—A. Table showing circumstances of some 50 cases in which

Case.	Notifica- tion number.	Initials.	Age.	Occupation.	Condition of woman when attacked with encephalitis lethargica.	Date of onset of E.L.
24	1297	V. G.	26	Domestic	Close on full term; confined 6.12.20, one week after onset, in a rescue home.	29.11.20
25	1386	E. S.	25	Charwoman	Pregnant six months	About 1.12.20
26	1397	M. B.	32	Housewife	Confined four weeks previous to onset.	Admitted to hospital 24.12.20
27	1354	R. L.	31	Housewife	Pregnant eight months.	17.12.20
28	1687	F. T.	35	Housewife	Pregnant six months	24.12.20
29	1419	I. M.	22	Housewife	Miscarriage 14 months previous to attack.	25.12.20
30	1915	J. D.	40	Housewife	In last month of pregnancy; confined 2.1.21.	26.12.20
31	1508	L.	35	Nil	In eighth month of pregnancy (4 para.).	26.12.20
32	1479	M. H.	27	Housewife	Puerperium one month before onset.	29.12.20

Encephalitis Lethargica was associated with pregnancy—continued.

Main features of attack.	Result to mother.	Result to child.	Remarks.
Lethargy alternating with excitement. Pupils contracted. Slight int. recti. paresis, occasional twitchings right forearm.	Recovered by 4.2.21.	?	Symptoms more marked after confinement. No albuminuria. Wassermann = 0.
Fever, high and persistent. Lethargy, ptosis, facial twitchings, tremors. Infarct in l. lung.	Died 2.1.21	Unborn	Positive Wassermann. C-s. fluid clear, no cells; albumen in "fairly large amount," trace of globulin, Fehling reduced.
Fever, deep lethargy, ptosis, tremors left side. Albuminuria. Cataleptic spasticity.	Died 26.12.20	Child healthy	Parturition and puerperium normal. Diagnosis of E.L. confirmed at P.-M.
Lethargy from 19.12. Ptosis right eye. Paresis hands and right arm.	Died 7.1.21	Unborn	Wassermann and Nonne-Lange tests negative.
Drowsiness, frontal headache, weakness, nausea. Slight lethargy, squint, nystagmus, tremors, semi-delirium, picking at bed-clothes.	Recovered.	?	—
Headache, lassitude, abdominal pain in left hypogastric region. Delirium, with lethargy by day. Twitching of mouth.	Died 8.1.21	—	Widal = 0.
Severe neuritic pains in right side of body. Unable to sleep for four days after confinement Very "nervy" and drowsy. Lethargy not marked. Definite nystagmus; palsy of (left) tongue.	Discharged from hospital much improved. ? Date.	Infant continues in good health and is thriving.	Wassermann=0. Fundi normal. C-s fluid sterile.
Conjunctivitis and neuritis. Lethargy. Ptosis (double), L. facial paralysis. Twitching left leg and arm. Tremors, delirium, slurring speech.	? Recovered	Male child born 6.2.21 with signs of encephalitis lethargica.	Child became lethargic almost at once and remained so for two weeks; there was also twitching of left arm and leg. (See Table B. Inst. 31.)
History of influenza after childbirth. High temp., headache followed later by apathy and drowsiness. Lethargy passed into semi-coma. Erythematous rash. Much improved by 30.1.21	Gradual recovery, but mental depression remains. Pregnant on 29.9.21 (five months).	Child healthy	Some albuminuria, no acetone; fundi normal.

APPENDIX II.—A. Table showing circumstances of some 50 cases in which

Case.	Notifica- tion number.	Initials.	Age.	Occupation.	Condition of woman when attacked with encephalitis lethargica.	Date of onset of E.L.
33	2231	D. P.	22	Housewife	Two pregnancies con- sidered as predispos- ing causes.	30.12.20
34	1605	G. G. E.	27	Housewife	In eighth month of pregnancy.	10.1.21
35	1566	E. D.	24	Housewife	Miscarriage two months previously.	11.1.21
36	2022	O. S.	19	Printer	Pregnant; labour in- duced 20.2.21, five weeks after onset.	13.1.21.
37	1485	I. O.	24	Housewife	Pregnant four months	24.12.20
38	2317	G. K.	37	Housewife	Confined 1.1.21-	15.1.21
39	2210	G. L. B.	39	Secretary	Miscarriage one month prior to onset.	15.1.21
40	1763	A. M.	32	Unemployed	Confined 10.1.21	27.1.21

Encephalitis Lethargica was associated with pregnancy—continued.

Main features of attack.	Result to mother.	Result to child.	Remarks.
Pain in right eye with dip- lopia. Fever and somno- lence. Mask-like counte- nance. Lethargy deepen- ing into coma; twitching of face and limbs.	Died 7.1.21	—	Diagnosis confirmed at P.-M.,
Lethargy. Pain and twitch- ing in limbs. Ptosis right eye; left facial paresis. Hallucinations at first, then quite sensible when aroused. Pupils sluggish to light.	Confined 7.2.21. Parturition and puerperium nor- mal. Quite recovered by 25.9.21.	Child born heal- thy and has re- mained so.	—
Headache, pain in back of neck and general weakness. Marked lethargy. Incon- tinence; weakness of facial muscles: inability to pro- trude tongue; tremors face and arms.	Slightly improv- ing.	—	—
Headache, vomiting, pains in wrists, shoulders and legs. Choreiform move- ments in various groups of muscles. From 18.1 marked lethargy, deeper in mornings. Ptosis. Pupils dilated. Left facial paraly- sis. Twitchings right fore- finger. Progressive pare- sis, lethargy became more profound. Epileptiform fits. Progressive paresis.	Died 10.3.21	Child stillborn	Case suspected to be chorea. Heart nor- mal; no retinal- changes. Wasser- mann (Blood and C.-s.f.) negative.
Headache for few days, then drowsiness and lethargy. Diplopia. Rigidity of arms and legs. Low muttering, delirium. Deepening coma. Numbness and tingling of fingers.	Died 6.1.21	—	Said to be a "typical case." No sugar or albumen in urine.
Vomiting and severe head- ache. Lethargy. Incon- tinence of urine and faeces. Unconscious at end.	Died 13.2.21	Quite healthy on 1.10.21.	—
Rise of temp. and intense mastoid headache for 14 days. Very drowsy though intelligent. Speech became sluggish.	Died 24.1.21	—	Some doubt as to diagnosis in this case. C.-s. fluid normal in all respects. Blood count normal.
Violent pain in head, vomit- ing, pyrexia, loss of speech and stupor. Slight left facial paralysis. Occasional delirium with stuttering and thickness of speech. Dull and apathetic.	Excellent reco- very by 21.2.21.	Alive and well as far as is known.	Labour normal. Discs pale.

APPENDIX II.—A. Table showing circumstances of some 50 cases in which

Case.	Notifica- tion number.	Initials.	Age.	Occupation.	Condition of woman when attacked with encephalitis lethargica.	Date of onset of E.L.
41	2004	H. D.	40	Wife -	Recovering from Cæ- sarian section on 12.1.21.	12.1.21
42	1719	M. C.	26	Housewife -	Pregnant five months	12.1.21
43	2158	Mrs. M.	25	Housewife -	Advanced stage of pregnancy: con- finement 14.2.21, two weeks after onset.	About 2.2.21
44	2399	R. J. S.	35	Housewife -	Advanced pregnancy. Instrumental de- livery on 28.3.21.	10.3.21
45	2532	E. K. G.	28	Housewife -	Confined 12.4.21. Nor- mal labour.	13.4.21
46	1175	Mrs. M. I.	26	Housewife -	In poor health, due to premature confine- ment 13 weeks pre- viously.	28.6.20
47	1373	R. A. H.	46	Housewife -	Suckling her eleventh child, aged seven months.	13.11.20
48	1329	Mrs. R.	30	Housewife -	Very debilitated from nursing a child for 14 months; baby weaned a fortnight before onset.	10.12.20

Encephalitis Lethargica was associated with pregnancy—continued.

Main features of attack.	Result to mother.	Result to child.	Remarks.
No particulars given -	Recovered with- out any ill-ef- fects except impaired vision, 1.10.21.	Child alive and healthy, 1.10.21	Notified as a case of encephalitis from the Jessop Hospital, Sheffield.
General excitability. Pains all over body, and head- ache. Lethargy during day. Ptosis once. Marked tremors lower limbs. Su- damina and dermatitis. Comatose towards end. Hyperæsthesia, delirious at night.	Died 26.1.21	—	—
2.2. Headache, increasing drowsiness, and disturbed vision. All these got more marked, and were asso- ciated with mild pyrexia; slight squint; some reten- tion of urine.	Gradually re- covered.	Still-born -	Patient was nursing relatives, who were suffering from influ- enza, to within a week of herself suc- cumbing with similar symptoms.
Headache, drowsiness, double vision later. Tem- perature increased after delivery to 104°. Thought to be a case of enteric fever.	Complete re- covery.	Well-developed; quite healthy on 14.10.21. Bottle-fed.	Widal negative on four occasions. No albu- minuria. No rash.
Pyrexia, scarlatiniform rash. Lethargy on 16.4. Dip- lopia. Paralysis of sphinc- ters. Delirium.	Died 18.4.21	Child developed similar rash, with fever and feebleness 14 days after birth, and died 4.5.21, after one week's illness.	Child considered to have suffered from the same affection as its mother.
Headache; then drowsiness, vomiting, paralysis of soft palate; double ptosis, slight coarse nystagmus, pupil reflexes normal, speech nasal.	Died 22.7.20	—	C-s. fluid under pres- sure; clear, no cells.
Headache, right side, with annulus. Lethargic. Diplo- pia, then right ext. strabis- mus. Twitching of facial muscles and some paresis; tremors of tongue. Some delirium, speech slurred. Constipation.	Recovered after slight relapse, 2.12.20.	—	—
Intense headache, slight vomiting. T. 99-101°, rising to 106° later. Lethargy from 15.12.20, also diplopia. Slight herpes lips.	Died 17.12.20.	—	—

APPENDIX II.—A. Table showing circumstances of some 50 cases in which

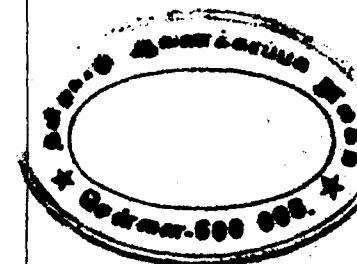
Case.	Notifica- tion number.	Initials.	Age.	Occupation.	Condition of woman when attacked with encephalitis lethargica.	Date of onset of E.L.
49	2,700	W. J. H.	40	Housewife	Pregnant eight months: labour induced on 23.7.21 owing to intractable vomiting and progressive asthenia.	18.7.21
50	2,708	A. G.	24	Housewife	Pregnant three months.	? 16.8.21

B.—TABLE SUMMARISING THE INFORMATION WITH REGARD TO SOME

Instance.	Notifica- tion No.	Initials.	Sex.	Age.	Sanitary District.	Occupation.
1	Not notified	—	F.	28 Infant	—	—
2	Not notified	—	F.	1½	Mansfield	—
			M.	9	"	—
			M.	8	"	—
3	Not notified	C. S.	M.	55	Birmingham	Out-door carter, L.N.W.R.
	228	H. S.	M.	19	"	Deck-hand in sheds, L.N.W.R.

Encephalitis Lethargica was associated with pregnancy—continued.

Main features of attack.	Result to mother.	Result to child.	Remarks.
Intractable vomiting associated with pregnancy. Patient complained of severe pain in lower cervical vertebra, relieved by turpentine fomentation; became very drowsy and lethargic, but vomiting gradually ceased. Kernig's sign and tache cérébrale were marked. Knee J. + No temp. over 99°. Lethargy gradually passed off and she has made apparently a good recovery.	Recovered	Born dead	Hexamine given during acute stage.
Depression of spirits, increasing drowsiness; always sleeping. Muttering for three nights before end. Quite unconscious. During first 10 days complained of nothing but headache. No fever at any time.	Died 21.8.21	Abortion during last hour of life.	—



30 INSTANCES IN WHICH case-to-case infection POSSIBLY OCCURRED.

Connection between cases.	Date of onset.	Type. See footnote p. 55.	Result.	Remarks.
Labour induced; baby born on 7.4.18: subsequently exhibited mild signs of E.L.	1918. 29.3.18	II. + - - -	Both recovered.	Dr. Wilfred Harris' case. (See <i>Lancet</i> , Ap. 20.1918, and Int. J. Pub. H., May-June 1921.)
Three cases in one family.	19.4.18	I. . . .	Died within three days in coma.	—
Brothers and sister	4.5.18 8.5.18	II. + - II. + -	D. . . R. . .	See MacNalty [629f].
Father and son living in same house.	Gradual in April, 1918.	II. - . . .	20. 11. 18. Quite well and at work. No sequelae.	—
—	19.1.19	II. + + - .	26.3.20. Paralysis of both legs and left arm. Tremors of face and arm, lethargic.	Dr. F. J. J. Orton's case.

APPENDIX II.—B. Table summarising the information with regard to some

In- stance.	Notifica- tion No.	Initials.	Sex.	Age.	Sanitary District.	Occupation.
4	111	Mrs. H. .	F.	63	Plymouth, C.B. .	—
	110	N. M. .	F.	39	"	Nurse in Nursing Home.
5	1,257	J. R. W. .	M.	13	Eastbourne, C.B. .	School . .
	1,258	A. W. .	F.	12	"	" . .
6	--	—	M.	—	Bristol C.B. .	—
	—	—	F.	—	"	—
7	92	R. G. .	F.	42	Bath C.B. .	Head school-
	93	E. A. D. .	F.	17	"	mistress. Packer . .
8	181	W. H. .	M.	33	Newton Abbot R. .	Farmer . .
	1,942	L. H. .	F.	25	" "	Nurse, of Leyton- stone.
9	138	C. A. .	M.	20	Walton-on-Thames U.	—
	151	Mrs. A. .	F.	56	" "	Military Officer.

30 instances in which case-to-case infection possibly occurred—continued.

Connection between cases.	Date of onset.	Type.	Result.	Remarks
Patient and nurse; latter, while on night duty, also developed symp- toms of E.L. .	5.12.18 .	I. + — . .	R. . .	Dr. R. Simpson's cases.
—	26.12.18	II. + — . .	R. . .	No other cases re- ported in the Home.
Brother and sister living together and sharing same bed.	1919. 24.1.19 .	II. + — . .	R. . .	Investigated by A. S. M. (M. of H.).
—	7.2.19 .	II. + — . .	R. . .	—
Father and daughter; latter visited home and saw father on the day of his re- moval to hospital.	Indefinite	—	—	Dr. J. O. Symes' cases. (See <i>Bristol Med.- Chir. Jour.</i> , 1920, XXXVII., 30.
—	8. 2. 19. Com- plained of tired- ness.	—	D. 20.2.19 .	Went to work 10th and 11th February. Febrile attack started 12.2.19 and she was removed to Hospital 17.2.19.
Lived 100 yards from each other.	About 2.2.19.	II. + — . .	D. 15.2.19 .	Dr. Fosbery's case.
—	6. 2.19 .	II. + — . .	R. . .	Dr. C. A. Marsh's case. Connection, if any, slight.
Brother and sister; latter home on leave.	16.3.19 .	II. + — . .	R. . .	Dr. A. Coleridge's cases. Investigated by Dr. H. B. Maple- ton and A. C. P. (M. of H.). The home of these pa- tients is an isolated farm on Dartmoor.
—	28.1.21 .	II. + — . .	D. 5.2.21 .	A third case oc- curred in Moreton- hampstead at the time of Miss H.'s illness, and a little later a fourth and fatal case was re- ported from the town.
Son nursed at home by his mother for three or four days; mother then de- veloped the disease.	28.3.19 .	II. + — . .	R. . .	Reported by Dr. F. Buzzard. (See <i>Brain</i> , 1919, LII., 308).
—	14.4.19 .	II. + — . .	R. . .	—

APPENDIX II.—B. Table summarising the information with regard to some

In-stance.	Notifica- tion No.	Initials.	Sex.	Age.	Sanitary District.	Occupation.
10	319	T. J.	M.	15	Acton	Fitter, Willesden Junction.
	1,673	E. J.	M.	18	"	Engineer
11	317	C. H.	M.	9	Farnworth U.	—
	332	F. H.	M.	5	" "	—
12	397	S. A.	F.	17	Derby C.B.	Cleaning and scrubbing.
	398	E. M. W.	F.	34	" "	Assistant to Matron (Member of Staff).
	399	G. P.	F.	18	" "	Laundry maid
	400	A. D.	F.	15	" "	Employed in laundry.
	401	V. S.	F.	17	" "	Laundry maid
	402	M. C.	F.	17	" "	Scullery maid
	403	W. K.	F.	55	" "	Matron
	404	G. W.	F.	30	" "	Assistant to Matron (in charge of kitchen).
	405	N. H.	F.	16	" "	House-work
	406	D. A.	F.	17	" "	"
	407	G. R.	F.	22	" "	"
		L. A.	F.	16	" "	"
13	420	C. D.	F.	2	Salford C.B.	—
	781	J. D.	M.	12½	" "	School
14	472	H. H. W.	M.	19	Wandsworth M.B.	Carpenter's assistant.
	523	A. B.	M.	33	" "	Police constable.

30 instances in which case-to-case infection possibly occurred—continued.

Connection between cases.	Date of onset.	Type.	Result.	Remarks.
Brothers in same house and sharing the same bed.	10.5.19	II.—	D. 27.5.19	Dr. Shadwell's case.
	29.6.20	II.—	Nervous symptoms have cleared up: patient attending hospital for lung disease.	Dr. Jamieson's case. Two other cases in same street: the first in 1918.
Brothers living in same house.	19.5.19	II.+-	D. 21.5.19	Dr. Ramsay's cases
	23.5.19	II.+-	D. 24.5.19	No history of tuberculosis.
Staff and inmates of a girls' home.	14.8.19	II.—	D. 22.8.19	Cases under the care of Dr. Helen Green, Derby.
	14.8.19	III. (Abortive)	Symptoms cleared up after one day's illness.	Outbreak first reported by Dr. Brindley, M.O.H., Derby; further investigated by Dr. MacNalty (M. of H.) and described by him in App. VII to Ann. Rep. 1919-20 of Chief Med. O., Ministry of Health.
—	15.8.19	II.+-	D. 25.8.19	Pathological investigations carried out by Profs. McIntosh and Turnbull and Drs. Helen Ingleby and Da Fano.
	15.8.19	II.+- With transient remission of symptoms.	R.	
—	16.8.19	II.+-	D. 25.8.19	N.B.—It was "case 397" which provided filtered emulsion of brain substance that was inoculated into a monkey and gave rise to typical lesions of encephalitis lethargica.
	16.8.19	II.+-	D. 18.8.19	
—	16.8.19	III. (Abortive)	In bed one day. 17.8.19. Quite recovered on 20.8.19.	Dr. Benington's cases.
	16.8.19	III. (Abortive)	R. by 20.8.19	
—	17.8.19	II.+-	R.	Doubtful instance: tubercular history in family.
	17.8.19	II.+-	D. 26.8.19	
—	19.8.19	III. (Mild)	R.	Slight connection, if any.
	27.8.19	III. (Mild)	R.	
Sister and brother, living at home.	2.9.19	?	D. (Convulsions) 12.9.19.	Discharged from hospital, cured, 10.12.19.
	10.3.20	II.+-	D. 24.3.20	
Patients conversed in street within two or three days of H. H. W.'s return from Cologne.	7.11.19	II.++-	D. 17.11.19	
	12.11.19	II.+-		

APPENDIX II.—B. Table summarising the information with regard to some

In- stance.	Notifica- tion No.	Initials.	Sex.	Age.	Sanitary District.	Occupation.
15	646	A. B.	M.	29	Westminster City	Tailor
16	—	Mrs. F. H.	F.	?	Bournemouth C.B.	—
	745	B. H.	F.	24	Lymington U.	Household
17	709	P. P.	M.	8	Holborn M.B.	School
		W. P.	M.	10	" "	" "
18	938	C. W.	F.	36	Pontardawe R.	Housewife
	937	G. W.	M.	16	" "	Collier's boy
19	1,016	W. R.	M.	49	West Bridgford U.	Nil
	—	A. R.	M.	—	—	—
20	1,095	C. P.	M.	12	Bristol C.B.	School
	1,097	J. S.	M.	14	" "	Errand boy
	1,237	J. H.	M.	64	" "	Bricklayer

30 instances in which case-to-case infection possibly occurred—continued.

Connection between cases.	Date of onset.	Type.	Result.	Remarks.
Patient's brother in hospital with same symptoms. Brothers had worked and slept together for past 18 months. Patient used to visit brother's shop where an assistant had E.L. in 1917.	2.12.19	II. + —	R.	Dr. A. N. L. Thomas' case.
Contracted disease while on a visit to her sister at Lymington.	1920. 13.1.20	—	D. 3.2.20 in Boscombe Infirmary.	Dr. C. W. Kay's cases. P.M. verified diagnosis.
In contact with her sister, who died of E.L.	15.2.20	III.	Discharged from Isolation Building, 1.3.20 R.	
Brothers	30.1.20	II. + —	D. 28.2.20	Dr. M. J. Ryan's case.
—	31.1.20	—	Delirious night of 31.1.20, pain in left side, convergent squint both eyes. 1.2.20, restless and fidgety.	Taken by mother to Charing Cross Hospital, 1.2.20
Mother and son, living together.	20.5.20	II. + —	D. 1.7.20	Dr. Walsh's cases. Son lived with his mother in an isolated cottage on the hillside.
—	7.6.20	II. + —	D. 22.6.20	
Brothers living in same house.	1.6.20	I. —	D. 10.6.20	Dr. Blurton's case.
—	Ill at the same time with somewhat similar symptoms.	—	R.	—
Neighbours living within three doors of each other. All resided in same street.	5.6.20	III.	Discharge from Btl. Gen. Hosp. 12.7.20. R.	Investigated by Dr. Davies and A. C. P. Short, enclosed passage-street peculiarly well adapted for the spread of an infectious disease. This grouping probably indicates something more than coincidence.
—	1.7.20	II. + —	D. 21.7.20	
—	21.9.20	II. + —	R. 26.2.21. Slight paresis left leg.	

APPENDIX II.—B. Table summarising the information with regard to some

In-stance.	Notifica-tion No.	Initials.	Sex.	Age.	Sanitary District.	Occupation.
21	1,152	H. A. C.	M.	18	Lincoln C.B.	Engineer's fitter
	1,207	H. S. S.	M.	18	" "	Engineer
22	1,254	Mrs. E. H.	F.	28	Southam R.	Housewife
	—	Baby H.	—	—	" "	Third child of above.
23	1,578	C. P.	M.	76	Croydon C.B.	Secretary (iron firm).
	2,202	H. H.	M.	18	" "	Student
24	1,722	R., L. J.	M.	19	St. Asaph R.	Teamsman and farmer.
	2,569	M. R.	F.	16	" "	Domestic servant
	Not notified	Two younger children			" "	—
25	1,969	R. D. S. R.	F.	9/52	Boston U.	—
	1,970	D. C. S. R.	M.	8	"	—
	Not notified	Dr. R.	M.	?	"	Medical Officer of Health.

30 instances in which case-to-case infection possibly occurred—continued.

Connection between cases.	Date of onset.	Type.	Result.	Remarks.
Patients worked in same department at engineering works.	1.8.20	II. + -	D. 31.8.20	Dr. Barlow's case.
—	14.9.20	II. + -	D. 2.10.20	Dr. C. J. Coleman's case in City Hospital.
Child born 28.10.20, during mother's illness, and developed E.L.	25.10.20	II. + 1	Jan. 1921. Slight ocular paralysis; left side paresis; clonic spasm right leg.	Dr. Pirie's case. Investigation by Dr. Gibbons Ward and A. C. P.
—	—	II. + 1	Died after living five weeks.	—
	1921.			
Friends. Attended same church. H.H. sat up in church all night with coffin of C.P.	7.1.21	II. + -	D. 22.1.21	Dr. H. D. Everington's case.
—	3.2.21	III.	Sleeping for three days, after which symptoms cleared up.	Dr. Brooke's case.
Farmer's children and a maidservant "living in."	Definite onset in all four cases on 8.1.21, but the son had been ailing for a fortnight previously.	I. -	Sleepy for 18 days, slowly recovered.	Cases under care of Drs. Jones and Morris. Circumstances investigated by Dr. Henry Lloyd and A. C. P. House in a lonely farmstead among Welsh hills. The son had recently been to London, where he very possibly became infected.
—		II. + -	First attack of sleepiness lasted two days; remissions with other symptoms three weeks later: slowly recovering.	
—	—	III. both	Both recovered in a few days.	
Sister and brother	14.2.21	I. + -	—	—
—	17.2.21	I. -	—	—
Father of children above.	—	III.	R. in 48 hrs.	—

APPENDIX II.—B. Table summarising the information with regard to some

In-stance.	Notifica- tion No.	Initials.	Sex.	Age.	Sanitary District.	Occupation.
26	2,112	D. S.	F.	9	Bristol C.B.	School
	Not notified	C. S.	M.	12	" "	"
	"	I.S.	F.	15	" "	"
	"	Mrs. S.	F.	—	" "	Housewife
27	2,422	J. R.	M.	26	Whitchurch U.	Railway goods porter.
	2,594	A. J.	M.	19	" "	Railway goods clerk.
28	2,193	M. C.	F.	42	Hornsey B.	Trained private nurse.
	2,194	A. C.	F.	38	" "	Typist.
29	Not notified	.	M.	58	Leicester C.B.	Bus driver
	"	.	F.	A few years younger than above.	" "	House duties
30	2,532	E. K. G.	F.	28	Bromley B.	Housewife
	2,635	C. G.	F.	22 days	" "	—

30 instances in which case-to-case infection possibly occurred—continued.

Connection between cases.	Date of onset.	Type.	Result.	Remarks.
Familial cases	21.2.21	I. + —	22.3.21. Re-covering.	—
—	Sleepy, sore throat early in February.	III.	R. in two or three days.	Investigated by Dr D. Davies, M.O.H., Bristol, and the School Medical Officer.
—	Vomiting and sleepiness March 3rd.	III.	Ditto.	—
—	Nausea, sore throat, sleepiness on Mar. 4th.	III.	Ditto.	—
A. J. replaced J. R. at work when latter was taken ill. Work consisted in unloading sacks of grain.	27.2.21	II. —	30.5.21. Still in hospital.	Dr. Perram's case.
—	20.5.21	II. —	D. 6.6.21	Dr. Newcombe's case. Investigated by Dr. Gepp, M.O.H., S. W. Salop, C.D., and A. C. P. Clinical notes supplied by the late Dr. Elliott, of Chester.
Sisters living together.	8.3.21	II. + — —	—	Dr. Gittens' cases.
—	21.3.21	III.	—	—
Man and wife	?	I. + . . .	Improvement very slow.	Reported by Dr. M. Dixon, Melton Mowbray.
—	?	II. — — + .	Weakness persistent.	—
Mother and newly born child—bottle fed.	13.4.21	II + —	A scarlet fever-like rash common to both	D. 18.4.21
	26.4.21	I — .		D. 4.5.21
				Dr. R. Montgomery's cases. Mother confined normally on 12.4.21. Child quite well for first 14 days, then rash appeared with severe constitutional symptoms.

APPENDIX II.—B. Table summarising the information with regard to some

Instance.	Notification No.	Initials.	Sex.	Age.	Sanitary District.	Occupation.
31	1,508	L. . .	F.	35	Shifnal R. . .	—
		Baby			" " . .	—
32	2,754	T. S. .	M.	55	Chesterton R.D. .	Farmer .
	1,675	D. S. .	F.	21	" " . .	Domestic servant .

30 instances in which case-to-case infection possibly occurred—continued.

Connection between cases.	Date of onset.	Type.	Result.	Remarks.
Mother and newly-born infant.	26.12.20	II. + — . .	R., after critical confinement.	Dr. D. J. M. Leggo's cases.
—	26.2.21	II. + — . .	R., after three months.	Child cried lustily at birth, but almost at once became abnormally quiet and then sleepy. For first fortnight of life the child never opened its eyes, and normal suckling had to be re-inforced by spoon feeding (mother's milk) because the patient could not be wakened to take the breast. There was twitching of left leg and arm similar in character and distribution to that which occurred in the case of the mother.
Father and daughter	6.12.20	II. — . . .	D. 1.9.21	Dr. Robert Ellis' cases.
—	30.8.21	II. + — . .	D. 16.12.20	Father, an alcoholic subject, suffered from general symptoms, lethargy and ptosis. Case of daughter marked by hyperpyrexia, deepening stupor, eye palsies, tremors, <i>flexibilitas cerea</i> and a papular rash on chest.

APPENDIX II.—C. TABLE GIVING LIST OF CASES (265) IN WHICH LUMBAR PUNCTURE WAS PERFORMED AND WHICH SHOWS FOR EACH CASE THE REPORTED CHARACTERS OF THE CEREBRO-SPINAL FLUID.

Note.—Abbreviations: N = normal; + = present, positive; ++ = increased in degree or amount; +++ = excessively increased; O = negative, absent; — = no statement made in report; L.P. = lumbar puncture; T.B. = Tubercle bacillus; E.L. = Encephalitis lethargica; D = fatal case: where cell contents are mentioned the figures refer to the number of cells per c.m.m. The details given in this table are transcribed from the original case reports, and it is obvious, therefore, that these details, supplied, as they are, by so many different observers—adopting, perhaps, different standards—have only a limited value for comparative purposes, except in cases where actual figures are given or methods are described. The characters of a "normal" cerebro-spinal fluid are discussed on page 146 of this report.

Notification number.	Physical characters.	Chemical content.	Microscopical features.	Bacteriology.	Remarks. (Pathologists' remarks enclosed in inverted commas.)
21	—	Reduction N. —	Practically normal.	—	"Suggestive of E.L." D.
526	—	{ Reduction N. — Noguchi O. —	Lymphocytosis	—	Wass. O.
659	—	—	Organisms O.	—	"Character of fluid and negative findings indicate, probably, E.L." D.
503	P. low	—	—	Sterile.	—
6	—	{ Alb. trace — Reduces Fehling — Faint globulin reaction —	{ Cells N. (lymphos.) — Organisms O. — Cells N. —	Sterile	D.
?	P. +	N.	—	N.	"Does not suggest syphilis."
179	P. + + +	—	—	—	D.
412	P. + + +	—	—	—	Negative. No relief after puncture.
78	—	—	—	—	D.
306	P.N., clear	—	—	—	"Negative." D.
660	—	Glob. N. —	Cells N.	—	D.
533	Deposit. Blood-stained.	Alb. + +	Lymphocytosis Organisms O.	—	D.
592	—	—	—	—	"Negative."
141	P. + +, clear	—	Lymphocytosis Cells 3—4	—	—
456	—	—	—	—	—
545	—	Reduces Fehling	Lymphocytosis Cells 3—4	Sterile.	—
45	P.O., clear, no clotting	Protein N. —	—	—	—
88	—	—	—	—	—

290	Clear.	—	Cells N.	Wass. O.	D.
325	Clear	—	—	—	D.
472	P.O.	—	Slight leucocytosis	Wass. O	D.
523	—	—	—	L.P. brought patient out of trance.	D.
524	—	—	—	D.	—
390	—	—	—	—	—
518	P.O., clear.	—	Slight lymphocytosis	—	—
485	P.O., clear.	—	Lymphocytes. Organisms O	—	—
147	P.O.	—	—	—	—
470	P. + + +, fluid opalescent.	—	Cells O, Organisms O.	—	—
548	P. +	—	—	—	—
240	P. +.	—	—	—	—
415	—	—	—	—	—
261	P. +, fluid in excess.	—	—	—	—
453	P.N., clear.	—	—	—	—
331	P. +, clear.	—	—	—	—
509	P. +.	—	—	—	—
12	P.O., clear	—	—	—	—
26	P.O., clear	—	A few cells. Poly. 10 %, Mono. 70 %, Endothelial 20 %.	—	—
27	Clear	—	Very few cells	—	—
104	—	—	Practically no cells	—	—
220	—	—	—	—	—
408	P. +, clear	—	Cells, mostly lymphocytes	—	—
9	—	Glob. 0.05 %. Reduction N.	Many lymphocytes	—	—
324	Yellowish	—	Moderate number of leucocytes	—	—
563	—	—	Organisms O.	—	—
					Scanty deposit of red and white cells on centrifuging.

APPENDIX II.—C. Table giving list of cases (265) in which lumbar puncture was performed and which shows for each case the reported characters of the cerebro-spinal fluid—*continued*.

Notifi- cation number.	Physical characters.	Chemical content.	Microscopical features.	Bacter- iology.	Remarks. (Pathologists' remarks enclosed in inverted commas.)
61	—	Marked reduction of Feh- ling.	Leucocytosis. Organisms O.	—	—
99	—	—	—	Sterile	—
309	N.	N.	N.	N.	“Practically normal.”
517a	P. +	Alb. N, Sugar N, Glob. O.	Cells 2.5	—	First puncture.
517b	—	Alb. N, Sugar N, Glob. trace.	Cells 22.5, lymphocytes and endo- thelial.	—	Second puncture.
517c	P.O. clear	N.	N.	N.	Third puncture.
445	P. +, clear	—	Cells N, Organisms O.	—	—
292	—	—	—	Sterile	“Negative.” D.
481	Clear	—	Considerable leucocytosis	—	Clinically, a case of E.L.; athetosis and paresis of arm as sequels.
359	—	—	—	—	Negative.
92	P. +, clear	—	Slight leucocytosis (lymphos. 95 %)	Sterile	D.
360	P. N.	Alb. slight increase	—	—	—
343	P. +, clear	—	—	—	—
249	—	—	—	—	Negative.
421	—	—	—	—	Negative.
539	—	—	—	—	Negative.
279	Clear	—	—	Sterile	—
278	Clear	—	No meningococci.	—	Negative.
167	Clear	—	—	—	—
298	Clear	—	—	Sterile	D.
483	—	N.	N.	Sterile	D.
271	—	—	—	Sterile	—

497	P. + + + +, pale and clear.	—	—	Sterile	—
358	P. +, clear	—	—	—	—
511	Amount greatly in- creased, clear and colourless.	Alb. 0.01 %. Glucose 0.1 %.	Cells 36 (mono-nuclears)	Sterile	—
37	—	—	—	Sterile	D.
247.	N.	N.	N.	N.	D.
J. & A. W.	P.O. clear	—	Few lymphocytes; traces of bacilli and cocci, latter just gram- negative. No T.B.	—	—
588	Clear	N.	T.B. O, meningococci O	N.	—
488	—	Alb. in excess. Reduction active.	N.	Sterile	—
465	P. +, clear	—	Occasional endothelial cell. Or- ganisms O.	Sterile	—
513	P. +, clear	—	Lymphocytes a few, polymorphs, a few, T.B. O.	Sterile	D.
560	Clear	—	—	Sterile	—
229	Clear	—	—	—	—
396	—	—	—	—	“No sign whatever of a meningitis either chem- ically, histologically or bacteriologically.” D.
132	N.	—	—	Sterile	D.
148	P.N, clear	No fluid	obtained.	—	Two punctures.
329	N.	Glob. reaction faint	Cells 32	Wass. O.	D.
245	N.	N.	N.	N.	D.
151	P. + +	Chlorides increased.	N.	N.	D.
138	P. + +	Glucose increased.	N.	N.	—
1210	—	—	—	—	—
1273	Clear	Proteins, slight increase.	Cells 8.	—	Son of above (151).
1144	P.O. clear	Alb. trace. Sugar un- diminished. Globulin O.	Cells 74 (all lymphos)	Negative.	D.
	—	—	—	Sterile	—

APPENDIX II.—C. Table-giving list of cases (265) in which lumbar puncture was performed and which shows for each case the reported characters of the cerebro-spinal fluid—*continued*.

Notifi- cation number.	Physical characters.	Chemical content.	Microscopical features.	Bacter- iology.	Remarks. (Pathologists' remarks enclosed in inverted commas.)
1845	Clear, no clot .	Alb. increased, Sugar N.	Cells 20. Organisms O .	Sterile	Negative.
574	—	—	—	—	Three attempts. D.
2033	—	No fluid	withdrawn.	—	D.
2218	P.N., clear .	Sugar 0.1 % .	Pneumococci O. Meningococci O.	Sterile	D.
1531	—	—	Organisms O.	—	D.
1945	Slightly turbid .	—	Leucocytes a few. Micrococci a few	—	Diagnosis after consulta- tion with Prof. Murray
1967	—	—	—	—	Liverpool. D.
1466	P. +, clear .	—	Organisms O. .	N.	—
1682	N.	N.	withdrawn.	—	—
1532	—	No fluid	(T.B. O, Meningococci O).	—	—
1673	—	—	Slight leucocytosis. Organisms O.	—	—
1425	P.O., clear.	—	Occasional lymphocyte. Organisms	—	—
1435	Clear, no flocculi .	Glob. N. Reduction N.	O.	Sterile	—
1358	P. + +, clear.	—	—	—	Negative.
1369	—	—	No excess of cells.	—	D.
1397	P.O., clear .	—	Cells 342 (Polymorphs. 47 %)	—	Patient eight months pregnant. D.
1354	—	Nonne O. Lange O .	Cells increased. Organisms O.	—	—
1321	Clear .	Globulin O. .	N.	N.	—
1323	N.	N.	—	—	D.
1326	P.O., clear .	—	Lymphocytosis	—	—
1333	Clear .	—	Cells 15--156 (chiefly lymphos)	Sterile.	D.
1336	—	Alb. increased	—	—	Doubtful case. D.

1337	—	Alb. increased	Cells 13--1600, no bacteria .	—	Negative.
1254	—	—	—	Sterile	D.
1153	—	—	—	—	D.
1154	P. +, clear .	—	N.	N.	—
1261	N.	—	Organisms O.	—	Negative. D.
1262	—	—	—	—	D.
1267	—	—	Leucocytosis (mono-nuclears)	—	Negative. D.
1276	—	—	—	—	D.
1278	—	—	Few lymphocytes, no polymorphs	Sterile	D.
128	P.O., clear .	—	N.	N.	Contaminated with sarcinae.
1284	Clear .	—	Red cells 5. White cells 20. (lym- phocytes and a few endothelial).	—	D.
1292	—	—	Cells O, organisms O .	—	Negative. D.
1295	N.	—	N.	—	Negative.
1297	Clear, no clot .	Alb. +, Sugar N. .	—	—	D.
1229	P. + + +	—	—	N.	—
1241	N.	N.	—	Sterile	D.
1247	—	—	—	—	D.
1250	—	Globulin + +, Sugar + +.	—	Sterile	D.
1196	—	—	Cells O.	Sterile	—
1202	Clear	—	Organisms O.	—	—
1205	—	—	Lymphocytosis, no meningococci .	Sterile	—
1207	—	—	Lymphocytes +, organisms O.	—	D.
1208	—	—	Lymphocytosis, organisms O.	—	D.
1211	P.O., clear, no clot .	Glob. O, Sugar + .	—	—	—
1214	Clear	—	Slight lymphocytosis. T.B. O; meningococci O.	Sterile	—
1216	—	Glob. + + .	Cells O, organisms O.	—	D.
1223	—	—	Organisms O.	—	D.
1224	—	—	Lymphocytes +, organisms O.	—	D.
1175	Clear	—	Cells O.	—	D.
1186	P. + +, clear	—	T.B. O; meningococci O.	—	D.
1188	—	—	Organisms O.	—	D.
1195	—	—	No organisms.	—	D.

APPENDIX II.—C.—Table giving list of cases (265) in which lumbar puncture was performed and which shows for each case the reported characters of the cerebro-spinal fluid—continued.

Notifi- cation number.	Physical characters.	Chemical content.	Microscopical features.	Bacter- iology.	Remarks. (Pathologists' remarks enclosed in inverted commas.)
1138	P. + +, clear	—	Cells O, organisms O.	—	D.
1150	—	Glob. + +, Glucose de- creased.	Few lymphocytes.	—	D.
1160	P.O.	Glob. +, Sugar N.	Relative lymphocytosis.	Sterile	D.
1166	P. +, clear	—	Lymphocytes a few; organisms O.	—	—
1120	—	—	N.	N.	D.
1123	N.	—	—	—	D.
1124	Clear.	—	No increase of cells	Sterile	40 c.c. removed. D.
1091	P. +, no deposit	Alb. N. Reduction slight.	Organisms O.	—	D.
1093	—	N.	N.	N.	D.
1097	N.	N.	N.	N.	D.
1098	N.	N.	N.	N.	D.
1103	Almost clear, no clot	Alb. diminished. Glob. O.	Cells 56 (lymphos. 80 %) (Endoth. 20 %).	Sterile	—
1057	—	—	No excess of cells	—	Negative. D.
1063	Clear	—	Cells 30.	Sterile	D.
1071	—	—	—	Sterile	—
1078	P. + + +, clear, colourless.	—	—	—	—
1027	N.	N.	N.	N.	D.
1030	P. N.	—	—	Sterile	Blood also sterile.
1032	—	—	—	—	Several punctures.
1035	P. + +	—	—	—	Negative.
1037	—	—	Few lymphocytes	Sterile	L. P. 8th day. D.
1040	N.	N.	N.	N.	Two L.P., which gave relief.

1043	—	—	—	—	Negative—Blood showed a leucocytosis (large lymphos). D.
1050	P. + +, clear	—	Cells increased	—	D.
1051	P. + +	—	—	Sterile	—
1008	Clear.	—	N.	N.	—
1015	P. +	N.	—	—	“Results compatible with E.L.” D.
1016	—	—	—	—	Negative. D.
1017	—	—	—	—	Four punctures.
941	—	No fluid	withdrawn.	—	—
945	P. + + +, clear	—	Cells O, organisms O	—	—
955	P.O.	N.	N.	N.	—
956	P. +, clear	—	—	Sterile	—
962	P.O.	—	—	Sterile	—
976	—	—	—	Sterile	Negative. D.
977	—	—	—	Sterile	No effect when injected into 8 guinea-pigs.
987	—	—	—	Sterile	D.
911	Clear.	—	Few leucocytes	Sterile	D.
916	—	—	Cells N.	Sterile	D.
919	P. +, clear	—	Leucocytosis (polymorphs. and lymphos).	Sterile	D.
927	—	—	Lymphocytosis	—	Wass. slightly positive.
880	N.	N.	N.	N.	—
882	N.	N.	N.	N.	—
890	N.	N.	N.	N.	—
821	Clear.	Alb. a trace. Sugar in- creased.	—	Sterile	—
827	P. + +	Lange's reaction doubtful.	Cells 300 { Endoth. 10 % Lympho. 40 % Polymorph. 50 % }	—	Wass. + in 0.5 c.c.; in blood negative.
833	—	—	Organisms O.	Sterile	Pneumonia a complica- tion. D.

APPENDIX II.—C. Table giving list of cases (265) in which lumbar puncture was performed and which shows for each case the reported characters of the cerebro-spinal fluid—*continued*.

Notification number.	Physical characters.	Chemical content.	Microscopical features.	Bacteriology.	Remarks. (Pathologists' remarks enclosed in inverted commas.)
838	P. + +, clear	—	Marked leucocytosis	—	D.
841	Clear.	—	Slight leucocytosis.	—	Wass. O. Clinically a case of E.L.
845	—	Alb. + +. Sugar diminished.	—	Sterile	Negative. D.
847	P.O., clear.	—	—	—	—
800	N.	Alb. trace.	N.	N.	D.
802	P.O.	—	Few cocci and a very few diplococci.	—	D.
804	—	—	Lymphocytosis	—	Negative. D.
807	P. + +, clear.	—	—	—	2 oz. withdrawn. D.
812	P. + +, clear.	—	Small lymphos. few pus cells	Sterile	D.
764	—	—	—	—	Negative.
776	P. + +, clear.	—	—	Staphylococcus aureus.	? Contaminated.
777	—	—	—	—	D.
781	—	—	Mono-nuclear leucocytosis.	—	D.
789	—	—	O.	Diphtheroid bacilli grown.	Pat. suffered from sore throat. D.
740	P. + +, clear	—	Lymphocytosis.	—	—
760	—	Alb. + +	Slight lymphocytosis	—	Wass. O.
708	—	—	T.B. O, meningococci O	—	Wass. O. D.
723	P. + +, clear	Alb. + Glob. O. Reduction N. Urea N.	Cells 40 (95% = small lymphocytes)	Sterile	Wass. O. D.

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682	—	—	Diplococci in pairs found in deposit.	—	—
697	—	—	Few cells, chiefly small lymphocytes.	—	Negative. Sugar in urine.
698	P. + +, clear	—	—	Sterile	D.
701	P. + +, clear	—	Few lymphocytes	—	Wass. O. D.
652	P.O.	—	—	—	—
655	N.	N.	N.	N.	—
619	Clear.	—	—	—	Six punctures; first followed by a fit and the 6th by return of consciousness.
632	Clear	—	Gram-positive diplococci.	Sterile	—
635	Clear.	—	—	—	Blood also sterile. D.
647	—	—	—	—	Negative.
648	—	—	—	—	51 c.c. withdrawn.
579	—	—	—	—	2nd puncture (5th day).
602	—	—	N.	Sterile	—
603	P. + +, clear	—	—	Sterile	—
611	P. + +, clear	—	Cells O.	—	—
1345	P.O., clear	—	Slight lymphocytosis	Sterile	—
1347	—	Alb. + +	—	Sterile	—
1408	Clear, no clot	Alb. 0.01%. Glob. + +. Dextrose a trace.	Small number of lymphocytes. Organisms O.	—	—
1497	—	—	Meningococcus O. Gram-positive cocci +; Gram-negative bacilli +. Slight lymphocytosis. T.B. = O.	—	—
1163	P. + +, clear.	—	11th day cells = 173	—	D.
1199	—	Protein + + +	18th " " = 104	—	Blood count — W.B.C. 3,400; poly. 78%.
1302	N.	N.	20th " " = 88.	—	lymphos. 17%. D.
1303	P. + +, clear.	—	Organisms O.	N.	—
1338	P. + +, clear.	N.	—	N.	—
1319	Clear.	—	—	—	—

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APPENDIX II.—C. Table giving list of cases (265) in which lumbar puncture was performed and which shows for each case the reported characters of the cerebro-spinal fluid.—*continued.*

Notifi- cation number.	Physical characters.	Chemical content.	Microscopical features.	Bacter- iology.	Remarks. (Pathologists' remarks enclosed in inverted commas.)
1318	—	—	Cells N, Organisms O.	—	D.
942	—	—	Lymphocytosis	—	Negative.
924	—	—	N.	—	—
974	P. + +	Alb. N., Glob. + +	Cells 57 (lymphos.)	N.	L.P. 10th day.
978	P. N.	Sugar diminished.	—	—	Wass. O. twice.
1089	P. reduced	Protein + +	Cells 7.	—	D.
982	P.O., clear	—	N.	Sterile	L.P. on 10th and 14th days.
990	P. +	N.	—	Sterile	—
1143	P. N.	—	No pus	—	Wass. O.
855	Bloodstained	—	Cells 8	—	Wass. O.
925	—	—	Cells 19, mostly small mono-nu- clears.	—	—
948	P. + +	Alb. 0.05%. Nonne- Apelt definitely +.	O.	—	—
934	Clear, colourless, no clot.	Chlorides 0.75%. Glucose 0.7%.	—	—	—
1006	—	—	Cells 12, mono-nuclears only	—	Negative.
949	—	Alb. 0.001%, Glob. O.	—	—	Negative.
997	P. + +	Reduction N.	N.	—	D.
931	N.	N.	N.	N.	—
939	N.	N.	Cells N	N.	D.
1010	—	Glob. (Noguchi) O. Sugar +.	—	—	—

	P. + +, no clot	Alb. slight increase. Dex- trose +.	Slight lymphocytosis.		D.
709	—	Proteins increased	Cells 9.	—	—
765	—	N.	—	Sterile	—
842	N.	—	N.	N.	D.
820	Bloodstained	N.	N.	Sterile	D.
782	N.	—	No polymorphs.	—	—
946	P.O.	—	Small lymphocytes	N.	—
905	P. N., clear	N.	Slight leucocytosis.	Sterile	D.
837	N.	—	—	—	—
596	N.	—	—	—	—
629	Clear.	—	—	—	—
599	P. + +	Alb., slight increase. Sugar N.	—	—	—
825	P. +	N.	N.	N.	D.
637	N.	N.	—	N.	—
675	N.	—	—	N.	—
712	N.	—	—	—	—
625	—	Alb. slight excess	—	Sterile	—

APPENDIX II.—D. TABLE GIVING THE REPORTED *post-mortem findings*

Notifi- cation. number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
644	F.	44	Vague, with general pains. Treated for indigestion.	Progressive lethargy, deepening to coma at last; moderate temp. Ptosis, but no other palsies.	24 days
290	M.	47	Epistaxis, with headache and vomiting.	Irritable, then lethargic; temp. subnormal till day of death, when it rose to 99.5. Pupils unequal and sluggish to light. Mask-like face.	24 days
472	M.	19	General malaise and diplopia.	Lethargy broken by convulsions; high temp. Squint and ptosis; oedema of discs. Both sphincters paralysed at last.	10 days
453	F.	59	Sudden, with attack of unconsciousness.	Deepening lethargy, alternating with delirium. Persistent vomiting on one day and obstinate constipation throughout. No eye or face palsies, but general paresis with retention and overflow incontinence of urine. Temp. remittent.	15 days
26	F.	16	Fairly rapid; headache and delirium.	Fever, lateral nystagmus and lethargy proceeding to coma.	11 days

IN THOSE CASES WHERE AN AUTOPSY WAS PERFORMED.

Post-mortem findings.			Remarks.
Naked-eye lesions.	Microscopical appearances.	Experimental results.	
Few small patches of very slight red staining in sub-arachnoid. No naked-eye changes in substance of brain or cord.			
Brain = 1320 grammes. Dura and pia moderately congested. Many small hæm. near pons; no inflammatory reaction around hæm. Lungs and kidneys congested.			
Meninges generally congested and brain substance also. No appearance of tuberculous or other form of meningitis.			
Both hemispheres engorged; pia-arachnoid space deeply congested. Some flattening of convolutions from cerebral oedema. In region of left temp-sphenoidal fissure the pia-arachnoid space was filled with blood-stained fibrin and fluid: no pus. Meninges at base apparently normal. No microscopic changes in brain substance. Lungs showed red hepatisation at bases behind.			
Superficial cortical veins turgid; some hæmorrhage in pia-arachnoid.	Few scattered minute hæmorrhages, with peri-vascular infiltration of mono-nuclear cells in cortex of ant. and post. cerebral lobes. Nerve cells unaffected, no proliferation of neuroglia; in floor of III. and IV. vent., pons, medulla and spinal cord there were numerous peri-vascular cell-infiltrations with occasional hæmorrhages. Superficially a layer of mono-nuclear cells and irritated blood vessels. Antr. horns of c. cord showed many mono-nuclear peri-vascular foci. Some diminution of nuclei and alteration of their position in the	—	Dr. I. Walker Hall, Bristol.

APPENDIX II.—D. Table giving the reported post-mortem findings

Notifi- cation number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
12	M.	3	Fairly sudden with headache and vomiting and then irritability.	Drowsiness and later partly unconscious. Abd. scaphoid, legs curled up. Towards end, left ophthalmoplegia, left facial paralysis and nystagmus right eye. Temp. low until just before death.	19 days

in those cases where an autopsy was performed—continued.

Post-mortem findings.			Remarks.
Naked-eye lesions.	Microscopical appearances.	Experimental results.	
Congestion of lower lobes of lungs the only gross change.	nerve cells. No proliferative foci of neuroglia. <i>Heart.</i>—Much peri-vascular irritation and cloudy swelling of muscle cells. <i>Lungs.</i>—Bronchiolitis and oedema. <i>Liver.</i>—Fatty infiltration. <i>Kidneys.</i>—Early catarrhal nephritis. (Extracted from the <i>Bristol Med. Chir. Journ.</i>, 1902, xxxvii., 33.) General oedema and a few hæm. in cortex of ant. and post lobes. Peri-vascular infiltration of a few blood vessels by mono-nuclear cells. Here and there a capillary was thrombosed. Nerve cells unaffected; no proliferation of neuroglia. In floor of III. and IV. vent., pons, medulla and s. cord hæmorrhages more numerous and arranged superficially; blood vessels more generally infiltrated by mono-nuclears, and around some were definite foci of these cells. Tissue immediately beneath floor showed layer of mono-nuclears, and peri-vascular tissues were thickly infiltrated. Definite foci of mono-nuclears in ant. horns of cervical part of cord. Nuclei of occasional nerve cells showed shrinking and eccentricity. Chromotolysis not a general feature. No foci of proliferation of neuroglia. <i>Heart.</i>—Cloudy swelling and evidence of irritation of peri-vascular tissues. <i>Lungs.</i>—Catarrh of small bronchi and general oedema. <i>Kidney.</i>—Cloudy swelling. <i>Liver.</i>—General fatty infiltration. (Extracted from <i>Bristol Med. Chir. Journ.</i>, 1920, xxxii., 32.)	—	Dr. I. Walker Hall.

APPENDIX II.—D. Table giving the reported post-mortem findings

Notifi- cation number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
273	F	20	Gradual, with change of temperament, and alteration in speech and headache.	Remittent temperature; ptosis, unequal pupils, palsy of face and tongue. Stupor alternating with delirium, dysphagia, incontinence of urine and f. and paralysis of chest muscles near end of illness.	14 days
341	M.	15	General malaise, with vomiting and headache.	Apathetic, progressive drowsiness. No ocular or other palsies. Plantar reflexes increased.	14 days
449	F.	20	Sudden, with left facial paralysis and left ptosis.	Lethargy began 6th day, increasing eventually to coma. Squint disappeared 10th day, replaced by nystagmus. Fine tremors eyelids and hands; plantar reflex at first flexor, then extensor. At first apyrexia; fever began 25th day of disease and rose to 106°, reaching 108·6° on day of death. Sphincter control lost last 3 days.	27 days
511	F.	16	Gradual, with apathy and amnesia.	Well marked lethargy, but mind clear until just at end. Slight double ptosis and face mask-like. General catatonic condition of muscles, slight twitching of legs. Retinal vessels slightly dilated. Speech monotonous and nasal. Temp. variable.	24 days
460	M.	47	Shivering attacks, followed by diplopia.	Semi-unconscious all the time. Temp. 101° average. Enlargement of both parotids.	8 days

in those cases where an autopsy was performed—continued.

Post-mortem findings.		Experimental results.	Remarks.
Naked-eye lesions.	Microscopical appearances.		
Very acute congestion over whole cerebrum, meningeal and cortical, with fine capillary hæm. Excess of fluid which escaped on opening cranium. Congestion of all vessels at base of brain and over pons and medulla. Fine hæm. into pons and medulla; lat. vent. distended.	Microscopically six sections of material taken from the floor of the IV. ventricle show, though not equally, the changes typical of enc. lethargica.	—	Prof R. F. C. Leith, Birmingham.
"Microscopically it has all the appearance of a healthy brain."	(1) The degenerative changes in the nerve cells (nuclei of certain cranial nerves) are not so pronounced as is often the case. (2) The congestion of the vessels is moderate. (3) The infiltration of the perivascular lymph sheaths with leucocytes is well marked. (4) There is a slight infiltration of the surrounding brain substance with similar leucocytes. (5) No meningitis.	—	
	Typical changes—see Dr. Deas' case, Appendix I., Case A, No. 449/1919.	—	Dr. J. G. Greenfield, London.
Surface of whole brain very pink ("rose brain"); numerous sub-arachnoid hæmorrhages of small size (5-1 m.m.). Congestion of cortical vessels of cord.			
—	"Brain shows everywhere fairly severe changes of the kind indicated in my recent paper, with Dr. Buzzard (Brain, 1919,	—	Dr. J. G. Greenfield, London.

APPENDIX II,—D. Table giving the reported post-mortem findings

Notifi- cation number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
247	M.	38	Somewhat sudden, with dimness of vision, diplopia.	Insomnia at first due to neuralgia in right arm, then became drowsy. Dysphagia, constipa- tion, paresis right face, bleph- arospasm. Death due to inhala- tion pneumonia.	1 month
229	M.	48	Those of influenza.	Drowsiness, increasing eventually to coma, but at first noisy and delirious. Double ptosis, some dysphagia. Some retraction of head; vomiting on one occa- sion. Max. temp. 103°.	10 days
245	F.	28	With headache, vertigo and drow- siness.	Irregular fever, 104° just before death. Lethargy deepened to coma. Incontinent through- out; constipated; reflexes di- minished. Slight ptosis, other- wise no focal symptoms. De- veloped pneumonia.	1 month
1210	M.	47	Vomiting and ab- dominal colic.	?	2 days
402 (No. 6).	F.	17	"Eyes going fun- ny." Vomiting.	Restless and emotional, with widely dilated pupils reacting sluggishly to light and accom- modation. Inability to swallow. Voice husky and deepening lethargy. Died in coma.	3 days
397 (1).	F.	17	Suddenly, with epi- staxis, nausea and stiffness of throat.	Rapidly became stuporose; ex- treme degree of ptosis and oph- thalmoplegia, pupils being wide- ly dilated. Died in coma.	8 days
399 (3).	F.	18	Nausea and dry throat, with dim- ness of vision.	Double ptosis, dilated pupils, photophobia; lethargy became profound; limbs spastic. K.J. exaggerated; general tremors.	10 days
401 (5).	F.	17	Dim vision; pain in throat and nausea.	Lethargy profound and progress- ive with double ptosis. Champ- ing movements of jaw; speech slow and slurred. Died in coma.	9 days

in those cases where an autopsy was performed.—continued.

Post-mortem findings.		Experimental results.	Remarks.
Naked-eye lesions.	Microscopical appearances.		
Edema surface of brain. Left lung gangrenous; healed tubercle left apex.	XLII, 305-338). There is a small hæmorrhage very near the dentate nucleus. Cortex shows fairly severe changes in the way of excess of small cells in the substance of the brain and a consider- able amount of neuronoph- agy." The histopathology of this case is considered in con- junction with that of two others in a series speci- ally investigated by Dr. C. Da Fano and Dr. H. Ingleby. (Vide Bibl. I. 252.)		
Meninges congested. Slight thin opalescent exuda- tion over whole upper surface of brain; mem- brane adherent for some distance along each side of central sulcus.			
Congestion of cerebral vessels.			
Post-mortem confirmed diagnosis of encephalitis lethargica.			
No sign of irritant poison- ing in stomach or intes- tines. No meningitis. Surface of brain univer- sally and deeply con- gested.	Congestion and edema, but little else.	—	Prof. J. McIntosh and Dr. H. M. Turnbull, Lon- don.
Some congestion of brain, but very little abnormal to the naked eye. Spleen somewhat enlarged.	—	Filtered emulsion inoculated into a mon- key gave rise to typ- ical lesions of E. L. in the mon- key.	Prof. J. McIntosh and Dr. H. M. Turnbull, Lon- don.
No macroscopic changes apparent.	Doubtful	—	Dr. C. Da Fano and Dr. H. Ingleby, London.
No macroscopical changes apparent.			Dr. C. Da Fano and Dr. H. Ingleby, London.

APPENDIX II.—D Table giving the reported post-mortem findings

Notifi- cation. Number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
406 (10).	F.	17	Nausea, abdominal pain, sore throat, drowsiness.	Progressive ptosis and ophthal- moplegia; lethargy at first diminished and general condi- tion improved, but coma event- ually supervened.	9 days
637	M.	23	Headache, drowsi- ness, asthenia and abdominal colic.	Increasing lethargy with coma- vigil; squint, diplopia and paresis right arm and leg. Suffered from delusions, photo- phobia and tremors of hands. Discs congested; c.-s. fluid under pressure. Temp. sub- normal during four days before death.	21 days
589	F.	37	Pain in head and neck, nausea and giddiness.	Temp. average 101°, rose to 104° just before death. No eye or face palsies, but twitching of face and arms.	12 days
782	M.	14	Very acute, with headache and sudden uncon- sciousness.	Rapidly comatose; dilated pupils but no other signs.	1 day
879	F.	19	Hysteria and me- norrhagia; "typi- cal chorea" in 48 hours and last- ing 3 days.	Temp. raised during irritative phase and subnormal during lethargy. Ptosis, squint and nystagmus; incontinence of urine and faeces.	17 days
939	F.	5	Maniacal.	High temp. Mania gave way to lethargy at end; dysphagia, incontinence of urine and faeces.	12 days
1209	F.	37	Sudden, with head- ache.	Very restless, no pupillary re- action, no lethargy, knee jerks exaggerated.	2 days
1086	M.	62	Giddiness and head- ache.	No fever. Increasing lethargy with palsy of mouth and tongue and incontinence of urine and faeces at last. Convulsion two days before death.	
982	M.	27	Headache, vomit- ing and twitch- ings.	Fully conscious up to 24 hours before death. Ptosis the only palsy. Twitching of head and shoulders and spastic condition of limbs.	9 days
1294	M.	25	Suddenly, with frontal headache.	Moderate fever; speech slow and slurred; ptosis; tremors of ab- dominal muscles. Incontinence of urine and faeces.	4 days

in those cases where an autopsy was performed—continued.

Post-mortem findings.			Remarks.
Naked-eye lesions.	Microscopical appearances.	Experimental results.	
No macroscopical changes apparent.	Doubtful	(The five pre- ceding entries refer to those patients who died dur- ing the outbreak in a girls' home, Derby.)	
Many punctiform hæmor- rhages in white and grey matter, chiefly white; thrombosis of veins of Galen and in longitudinal sinus.			
Œdema of meninges.			
Considerable swelling of brain as a whole.			
Post-mortem findings consi-	sonant with diagnosis of encephalitis lethargica.		
Congestion of brain; bron- cho-pneumonia.			
Diagnosis of E.L. confirmed	by P.M.		
Examination of brain showed	death to be due to E. L.	—	Dr. B. H. Spils- bury, London.
Considerable external hy- drocephalus and slight atrophy of cerebral con- volutions.			
No gross lesions. Congestion of pia-arachnoid. Capil- lary congestion and small punctate hæm. freely scattered throughout white matter of hemi- spheres and basal ganglia. Much congestion of cho-			Dr. J. G. Forbes, London.

APPENDIX II.—D. Table giving the reported **post-mortem findings**

Notifi- cation number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
1336	F.	40	Drowsiness and tingling in R. ear.	Increasing lethargy; delirium; fever slight. Squint and diplopia with general paresis and immobility. Slight general tremors. Cerebro-spinal fluid normal.	23 days
1347	M.	12	—	Cerebro-spinal fluid sterile	6 days
1357	F.	20	Gradual onset of drowsiness.	Continuous fever, extreme lethargy and toxæmia, asthenia. Some facial paralysis and incontinence of urine. Slight tremors of limbs and myoclonus. C.-s. fluid normal. Blood showed a polymorpho-nuclear leucocytosis.	24 days
647	M.	16	Sudden, while at work.	Fainting attack; then delirious at night, with day lethargy. Slight ptosis, otherwise no focal symptoms. Blood and c.-s. fluid normal.	5 days
700	M.	32	Sudden, with headache and vomiting.	Nervous symptoms, both general and local, were negative, but some delirium occasionally.	22 days
708	M.	32	Apoplectic fit after some weeks of headache and lethargy.	Lethargy deepening rapidly to coma. Ptosis, squint, complete facial palsy, and partial of left arm. Irritable, photophobia, contracted pupils; twelve convulsive attacks and tremors of face.	2 days
783	F.	36	Paralysis of little finger, then left arm.	Wass. = 0. C.-s. fluid sterile. Hyperpyrexia (max. 107.8°); screaming fits and deepening lethargy. Early papillary oedema, ptosis, and conjugate deviation. Rigidity of left arm, Babinski, and exaggerated K.J. Temp. persistently subnormal. Pin-point pupils, dysphagia and retention of urine.	5 weeks
784	M.	5 weeks	Increasing drowsiness.		6 days

in those cases where an autopsy was performed.—*continued.*

Post-mortem findings.			Remarks.
Naked-eye lesions.	Microscopical appearances.	Experimental results.	
roid plexus and ependyma lining both lateral ventricles, resulting in hæmorrhagic discolouration of the underlying brain, but no evidence of actual hæmorrhage into ventricles. Mid-brain and medulla congested, also deeper parts of cerebellum, but no definite hæm. visible to naked eye. Changes observed are consistent with a diagnosis of E. L.			
Punctate hæmorrhages around fissure of Sylvius and some oedema of upper pons.			
Marked congestion of meninges.			
—	Brain microscopically shows typical lesions of E. L.		
Multiple small subserous hæmorrhages in lungs, heart and brain. Spleen slightly enlarged.			
Congestion of superficial vessels, especially near pons and bulb. No meningitis.	Sections showed acute encephalitis.	—	Dr. H. St. Arnaud Agate.
Intense congestion convex surface brain and cerebellum; less marked at base. No signs of tubercular lesions. Punctate hæmorrhages in upper layers of brain. No sign of any congestion of pons. Brain intensely congested.			
Rust coloured patches on brain, vertex and base, but most marked at base. Brain very soft.			Prof. E. E. Glynn, Liverpool.

APPENDIX II.—D. Table giving the reported post-mortem findings

Notifi- cation number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
812	M.	3	Twitching of lower lip, then difficulty in speech, and dribbling.	Progressive lethargy, inability to articulate, dysphagia and paralysis of tongue. Right side parietic. General convulsions and extension of twitchings to left face, right face and right arm. C.-s. fluid clear, pressure increased.	4 days
882	M.	48	Drowsy, with occipital headache.	Fever only at onset; lethargic by day and delirious at night. Some squint and facial paresis. Fundi and c.-s. fluid normal. Developed lobar pneumonia.	5 months
941	F.	27	Gradual, with drowsiness.	Twitchings of arms and thumbs and, later, all limbs; pupils first contracted then dilated. Reflexes at first exaggerated, then lost. Flaccid palsy of all limbs at last with deepening stupor. Urine and blood normal. No fluid on lumbar puncture.	—
945	F.	27	Drowsiness following pneumonia.	Severe occipital headache; temp. variable; frequent vomiting and constipation. Bright at times and then lethargic. Speech slow and deliberate; no eye palsies, but paresis of legs. Babinski, loss of K.J. and abdominal reflex. C.-s. fluid clear and under great pressure.	42 days
1037	F.	35	Gradual, with malaise for 3 weeks.	Increasing lethargy, with intervals of wakefulness; great asthenia, hyperpyrexia and face flushing. No eye palsies; twitching of lips and slight rigidity of neck.	9 days
1038	F.	54	Gradual, with general malaise, headache and fever.	Increasing lethargy, mind clear when awake; transient ptosis and squint and incontinence of urine. Some twitching of lips and tremors of fingers.	7 days
1088	M.	37	With abdominal pain and vomiting.	No lethargy. Delirium, with pain in chest and abdomen and spasticity of muscles. Ptosis and squint. C.-s. fluid sterile.	17 days
1138	M.	32	Myoclonus and pain in abdominal muscles, diplopia.	Delirium at first and then increasing lethargy. Fundi normal, marked leucocytosis of blood; c.-s. fluid normal.	12 days
1152	M.	18	Pain and discharge from R. ear, fever and headache.	Progressive lethargy preceded by delirium and hallucinations. Ptosis and squint late and incontinence of urine and faeces for last fortnight. Widal = 0.	20 days

in those cases where an autopsy was performed—continued.

Post-mortem findings.		Experimental results.	Remarks.
Naked-eye lesions.	Microscopical appearances		
Brain oedematous and congested. Meninges congested; a little sero-purulent matter in fissures.	—	—	Doubtful case.
Meningitis of vertex; much clear fluid, lymph and adhesions. Pia dotted with opacities. Substance of brain normal to naked eye.			
All organs apparently healthy, but cerebro-spinal fluid very scanty.			
Pia congested; no clear evidence of meningitis, but thickening of membranes at vertex. Numerous small congested vessels in grey and white matter. No tumour present. Good deal of greenish and purulent exudate.	No. T.B. No growth.	Exudate from ventricles injected into g. pig produced no lesions of tubercle.	
Nothing macroscopical except apparent congestion of pituitary.	"No evidence of peri-vascular or other changes which have been described as characteristic of E. L."		
—	Very marked peri-vascular infiltration, with small round cells; congestion. Few glial cells.		
Lesions of encephalitis lethargica are present and typical.		—	Clinical Research Laboratory.
Injection of pia; one or two patches of sub-pial hæmorrhage.			
Brain very congested.			

APPENDIX II.—D. Table giving the reported **post-mortem findings**

Notifi- cation number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
1160	M.	16	Sudden, with epis- taxis, restlessness and headache.	No pyrexia, but great thirst and tachycardia. Roused with diffi- culty. Epileptic seizures, myo- clonic spasms R. side of body and fibrillary twitchings.	15 days
1097	M.	14	Headache, delirium and choreic move- ments.	Progressive lethargy; speech slow and slurred; ptosis. Fundi and c.-s. fluid normal.	20 days
1098	F.	45	?	Cerebro-spinal fluid normal. Pro- gressive lethargy.	17 days
1207	M.	18	Sharp, with head- ache, rigors and vomiting.	Increasing lethargy; constipa- tion; aphasia; no eye or other palsies, but general flaccidity. Hypostatic congestion of lungs; c.-s. fluid showed lymphocy- tosis.	18 days
1222	F.	32	Sudden onset, with headache, vomit- ing and diplopia.	Temp. subnormal. Comatose al- most at once. R. face and R. arm paralysed, R. leg partly. Some jerky movements of body generally.	2 days
269	F.	16	Neuralgia R. face and head. Rigor.	Moderate lethargy; double ptosis and bilateral facial palsy. Pro- gressive asthenia. Some tremor in left leg.	6 days
276	M.	30	Severe headache, vomiting and dip- lopia.	Fever slight; became lethargic on 7th day and later comatose. No optic neuritis, divergent squint. Mask-like facies and "an effigy in aspect and pos- ture." Arms spastic. Profuse sweats and flushing.	21 days
1290	M.	44	Gradual, with gen- eral malaise.	Nervous, with sense of impending calamity. Fever marked; temp. reaching 110° just before death. Erythema on arms. Deep stupor, double ptosis and in- continence of urine and faeces. Twitching of L. face and later of arms. K.J. exaggerated, no Babinski.	4 days
1295	M.	29	Gradual, with lan- guor, headache and anorexia.	Lethargy, difficulty in articula- tion, ptosis. Fundi normal, also c.-s. fluid.	1 month
1336	F.	40	?	Wass. = 0. Cerebro-spinal fluid contained 15-156 cells per c.m.m., chiefly lymphocytes. Albumen present.	

in those cases where an autopsy was performed—*continued.*

Post-mortem findings.		Experimental results.	Remarks.
Naked-eye lesions.	Microscopical appearances.		
Left Rolandic area intensely congested; right less so.			
No gross lesions. Abscess in one tonsil.			
No gross lesions in brain or cord.			
Brain very congested, ex- cess of fluid in ventricles. Vascular engorgement in sheaths of small arteri- oles. No actual hemor- rhages seen.			
No changes found.			
Hæmorrhage in pons and floor of IV. ventricle. Small hæmorrhages in the white matter and conges- tion of all surface vessels.	"Histopathological ap- pearances typically those of E.L."	—	Dr. C. De Fano.
Negative.			
Marked congestion of men- inges; no meningitis. Recent inflammation of <i>liver, spleen and kidneys.</i> Advanced chronic <i>nephri- tis.</i>	Infiltration and necrosis of the nerve cells and fatty degeneration of neuroglia. Diplococci, in all respects like Rose- now's, present. Appear- ances are in keeping with a diagnosis of E. L.	---	"Virolog" labora- tories.
Numerous minute hemor- rhages in hemispheres, pons and medulla.			
Venous congestion of brain.			

APPENDIX II.—D, Table giving the reported post-mortem findings

Notifi- cation number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
1397	F.	32	Gradual, with gen- eral malaise and apathy.	Stuporose; temp. averaged 100° but rose to 106° just before death. Double ptosis, inconti- nence of urine. Marked weak- ness of trunk muscles with tremors on left side and general cataleptic spasticity. Some al- buminuria; discs normal. C.-s. fluid normal.	1 month
1425	F.	47	Malaise and low delirium.	Increasing lethargy replaced early by delirium; double ptosis, but no other palsy. Some tremor. Wass=O. C.-s. fluid normal.	34 days
1466	F.	24	Somewhat sudden, with twitching of muscles.	At first sleepless, then overcome with increasing stupor; temp. remittent. Double ptosis and slight squint, fundi normal. Nothing abnormal in C.-s. fluid.	12 days
412	M.	5	Vomiting and con- vulsions.	Marked lethargy; ptosis and quint, varying from day to day; paresis shoulder and fore- arms. Irritable and delirious at times.	11 days
306	M.	38	Gradual, with lassi- tude and depres- sion.	No delirium, marked and increas- ing lethargy and ptosis, slight facial palsy, slight paresis left side; temp. rose to 105° before death. Peculiar purplish blue colouration of face.	
660	M.	24	Gradual general malaise.	Continuous lethargy with paresis of L. side L. face, marked con- stipation. Optic neuritis, nys- tagmus. Tendon reflexes in- creased. Babinski left side.	4 months

in those cases where an autopsy was performed—continued.

Post-mortem findings.			Remarks.
Naked-eye lesions.	Microscopical appearances.	Experimental results.	
No oedema of brain; corti- cal vessels engorged. A few cortical hæmor- rhages but no special con- gestion of basal ganglia.			Dr. J. R. Perdrau.
Cerebral veins engorged. No manifest lesion.			
Injection of vessels and signs of encephalitis. All other organs normal.			
Brain and meninges in- jected, especially the pia- arachnoid. Punctiform hæmorrhages in cortex and also, to a less extent, in the basal ganglia.			
Sub-dural hæmorrhages over night ascending frontal and parietal, and superior and middle frontal lobes. Ext. hydro- cephalus; blood-stained int. hydroceph. Marked congestion of medulla and brain; punctiform hæm. of mid-brain. Blood in intradural space. Cord swollen and oedematous. Liver and lung bases congested. Slight paren- chymatous degeneration of kidneys.			
No naked eye changes. Cerebral cortex rather acutely inflamed; good deal of blood between pia and arachnoid. Cere- bellum much less affect- ed. Intense engorgement of grey matter generally; cortex affected more than pons and medulla. Ap- pearances rather like a typical cer. hæm. from ruptured aneurysm, but arteries healthy and no aneurysm seen. Blood universally distributed in a thin layer.	Those characteristic of E. L. in pons and me- dulla. Nothing in cere- bellum. Slight infiltra- tion of meninges. Brain under preparation for section cutting.	—	

APPENDIX II.—D. Table giving the reported **post-mortem findings**

Notifi- cation number.	Sex.	Age.	Type of onset.	Main clinical features.	Duration of illness.
592	M.	56	Diplopia	Frontal headache, restlessness followed by great drowsiness. No eye or face paralysis, but definite paresis of respiratory muscles. Some spasticity lower limbs. Coma at last.	41 days
45	M.	28	Irritability, pain and stiffness of limbs, headache.	Lethargy, with ptosis, facial palsy, paralysis of right tongue and incontinence of urine. Temp. declined towards death.	

in those cases where an autopsy was performed—*continued.*

Post-mortem findings.			Remarks.
Naked-eye lesions.	Microscopical appearances.	Experimental results.	
Cortical vessels extremely congested; localised patches of slight meningitis; patches of lymph on posterior part of cerebellum and a few on cerebrum. Tiny pin-point hæm. in Sylvian fissures and on upper surface of temp. lobes. Recent filmy adhesions to the longitudinal Sylvian fissures. Spinal cord congested in less degree. Some atheroma of thor. aorta.	Films from patches showed lymphocytes and a few polymorphs, but no organisms.		
Blood-stained fluid in cisterna magna. Some lymph exudate and venous engorgement — capillaries over cerebellum congested.	Brain being prepared for section cutting.		

APPENDIX II.—E. TABLE SHOWING THE VARIOUS *sequelæ* REPORTED AS OCCURRING IN THE CASE OF 271 PATIENTS ATTACKED BY ENCEPHALITIS LETHARGICA.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
523	33	11.11.19	II + -	—	Slight paresis L. ext. rectus.
13	41	18.12.18	II + -	—	Squint for 4 months.
587	12	19.11.19	II + -	24.2.20	Slight squint; twitching during sleep; very nervous and excitable; knee jerks exaggerated.
389	68	27.7.19	I -	—	Partial paralysis; impaired mental condition.
4	20	21.12.18	II + -	21.3.20	Has to wear glasses; right arm tremulous and weak; irritable and passionate; "breaks china and threatens murder."
49	56	12.1.19	II -	9.3.20	Persistent squint; irritability and shortness of temper.
567	54	14.12.18	II + 1	9.3.20	Requires glasses; general weakness.
545	12	16.11.19	II + -	Early 1920.	Ciliary muscles weak; difficulty in accommodation.
88	13	Dec. 1918	II -	Early 1920.	Mentally defective.
18	47	6.1.19	II + + + -	4.4.19	Practically recovered; light work.
505	33	20.11.19	II + -	Early 1920.	Tremors; slight athetosis.
307	16	2.2.19	II + -	Early 1920.	Slight tremors; apathy.
392	59	13.6.19	II + -	Early 1920.	Lack of concentration.
154	12	16.1.19	II -	Early 1920.	Very excitable and violent with his mother; to be put under restraint.
126	14	23.2.19	II -	Early 1920.	Definite chorea, slow speech.
426	23	13.8.19	II -	Early 1920.	Slight blurring of vision; dull.
549	35	30.4.19	II -	8.12.19	Speech, hearing and mental condition good, but is quite blind.
218	32	15.2.19	II -	1920	Slight drawling of speech.
350	4½	11.6.19	II -	Early 1920.	Paralysis of neck muscles.
432	17	21.8.19	II -	March 1920.	Paresis left leg and left arm; can walk, but cannot board a train.
256	11	4.3.19	II -	28.2.20	Recovery very slow; legs still paralysed.
168	11	11.2.19	II -	1920.	Memory very defective; easily frightened and not able to walk far.
459	40	20.9.19	II -	1920	Mentally dull.
60	2	7.2.19	II + -	3.3.20	Recovery protracted but complete.

APPENDIX II.—E. Table showing the various *sequelæ* reported as occurring in the case of 271 patients attacked by encephalitis lethargica—continued.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
51	37	12.12.18	II + -	25.3.20	Still under treatment: twitching of limbs; athetosis. Slight ocular palsy; mental condition good.
27	16	11.1.19	II + -	18.3.20	Difficulty in reading.
74	31	4.1.19	II -	19.3.20	Slight paresis of legs.
144	48	28.3.19	II + -	1920	Delusions.
353	21	Mar. 1919	II + -	2.5.19	Physically and mentally weak.
549	16	12.12.19	II + -	18.3.20	In bed with chorea; athetosis.
573	35	16.12.19	II + -	1.4.20	Vision impaired; speech slow and deliberate.
550	4	23.12.19	II -	1920	Slight ocular palsy.
166	16	20.12.18	II -	1920	Feeble-minded.
171	19	26.6.19	II -	1920	Palsy of legs; arms stiff; melan cholic.
175	58	Jan. 1919	II -	1920	Coarse tremors right hand, persistent during sleep. Mentally dull, though once a very capable salesman.
408	16	10.8.19	II -	1920	Nystagmus for several months after discharge.
10	29	24.12.18	II -	1920	Slight palsy right face.
54	43	23.12.18	II + -	1920	Slight palsy face.
82	47	Jan. 1919	II + -	1920	Spasticity of some muscles; bladder still weak.
502	19	19.11.19	II -	1920	Gait stiff, mentally dull, speech affected.
438	15	16.1.19	III	Early 1920.	Slight drowsiness and staccato speech.
36	39	19.1.19	II + -	27.3.19	Double ptosis marked, and palsy ext. rect.
388	70	28.7.19	II -	1920	Lack of mental grip.
157	30	27.12.18	II -	1920	Insomnia.
480	13	29.10.19	II + -	1920	Some eye palsy; frequent frontal headache; memory impaired; irritable.
517	15	2.12.19	II + -	1920	Very weak; frequent and severe headaches.
172	11	22.12.18	II + -	April 1920.	Some palsy left face and left leg.
379	14	11.7.19	II + -	March 1920.	Legs spastic; speech very defective; dull mentally.
481	30	25.10.19	II + + + -	March 1920.	Paresis right arm; some athetosis.
223	12	9.2.19	II + -	March 1920.	Headaches; still away from school.
359	42	July 1919	II + -	April 1920.	Impaired memory.
249	18	1.2.19	II + -	10.4.20	Partial palsy muscles right eye. Walks with a stick; improvement mentally.

APPENDIX II.—E. Table showing the various sequelæ reported as occurring in the case of 271 patients attacked by encephalitis lethargica—*continued*.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
236	58	10.2.19	II —	April 1920.	Slight ocular palsy.
422	35	25.8.19	II + —	1920	Right ptosis persists.
279	11	26.12.18	II —	1920	Loss of memory; mentally dull.
286	48	1.1.19	II —	—	Removed to Bethlehem Hospital for a period.
284	50	20.1.19	II —	1920	Face and eye palsies slowly clearing up.
578	11	—	II + —	22.4.10	Still has diplopia.
120	22	11.3.19	II + —	1920	Slight palsy face.
297	30	20.12.18	II + —	1920	Slight ptosis.
295	38	23.1.19	II + —	1920	Leg palsied; absent-minded.
296	59	20.12.19	II + + —	1920	Diplopia; slight ptosis.
298	64	Jan. 1919	II + + —	1920	Slurring of speech; inco-ordination legs and arms; mentally dull.
512	39	22.11.19	II + —	1920	Wasting muscles.
541	39	13.12.19	II + —	1920	Eye palsies remain.
561	20	12.12.19	II —	1920	Diplopia.
534	10	13.12.19	II —	1920	Mentally weak.
192	50	31.12.19	II + —	1920	Eye palsy persists; marked tremors.
581	12	11.12.19	II + —	1920	Some palsy right arm.
55	43	17.1.19	II + —	1920	Tremor in one leg.
558	30	22.11.19	II —	April 1920	Some eye palsy; partial palsy of legs; mentally dull.
252	13	12.1.19	II + —	30.5.20	Mental powers impaired; speech and movements slow.
434	32	18.9.19	II + —	23.4.20	Dull and lethargic.
214	7	23.1.19	II + —	21.4.20	Good physical recovery, but is thievish.
501	22	15.11.19	II + —	15.4.20	Not so alert; speech hesitating; easily tired.
554	21	22.11.19	II + —	April 1920.	"Not the same girl as before"; dull, apathetic and sometimes drowsy.
226	6	18.1.19	II —	1920	Physically well, but mentally changed. Irresponsible; steals; ill-treats younger child; irritable.
228	19	19.1.19	II + + —	26.3.20	Palsy both legs and left arm; tremors face and arm; lethargic.
527	16	11.12.19	II + —	19.4.20	Some palsy face; apathetic.
555	15	15.12.19	II + —	26.4.20	Strange in mind and manner; has delusions.
583	12	24.12.19	II + —	26.4.20	Squint; some weakness of legs; twitchings of face; excited and irritable.
322	18	25.5.19	II + —	16.4.20.	Slight internal squint.

APPENDIX II.—E. Table showing the various sequelæ reported as occurring in the case of 271 patients attacked by encephalitis lethargica—*continued*.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
557	30	10.11.19	II + —	1920	Slight squint right eye; some diplopia; slight tremor.
83	41	7.2.19	II + —	29.4.20	Paresis arms and legs; "still strange" and depressed.
587	12	6.11.19	II + —	18.1.21	Right eye bulges at times; nervous at night; general health fair.
629	27	28.11.19	II + —	18.1.21	Paralysis L. arm and side.
675	37	22.12.19	II + —	22.1.21	Very nervous and excitable.
712	34	10.1.20	II + —	21.1.21	Still under treatment; partial paresis legs and left hand.
722	37	29.1.20	II + —	2.2.21	Memory impaired; exaggerated reflexes.
765	20	12.2.20	II —	21.1.21	Abnormally fat. Attacks of <i>petit mal</i> .
936	11	30.2.20	II + —	21.1.21	Drowsy, weak and dull mentally; some athetosis.
946	57	10.4.20	II + —	15.2.21	Diplopia and giddiness.
915	16	12.4.20	II + —	8.2.21	Nystagmus. Right arm rigid and weak; tremors right arm; general movement slow and difficult.
854	18	15.4.20	II + —	1920	Athetosis right hand and leg.
905	29	19.4.20	II + —	26.1.21	Attending hospital for neurosis.
837	54	9.4.20	II + —	21.2.21	Slight paresis limbs.
925	20	19.5.20	II —	21.2.21	Unable to work, drowsy by day, sleepless at night.
935	16	27.5.20	II + —	14.2.21	Still attends hospital; drowsy at times.
1006	14	29.5.20	II + —	25.4.21	Marked lateral nystagmus; some facial paralysis; paresis L. arm, heavy and generally weak. Incontinence of urine.
999	14	31.4.20	II + —	21.1.21	In <i>statu quo</i> .
1005	16	14.6.20	II + + —	19.1.21	Attending hospital for fits.
942	36	16.6.20	II + —	21.1.21	Still in hospital.
990	10	1.7.20	II + —	23.12.20	Still appears a "mental defective"; tendency to tremor; little control of emotions.
1143	39	25.7.20	II + —	11.2.21	Still confined to bed in hospital. General weakness and marked constipation. Some athetosis; irritable and mentally dull.
1230	41	2.8.20	II + —	21.2.21	Still in bed; tremor of face; emaciated.
1173	35	3.9.20	II + —	14.2.21	Still attends hospital. Drowsy at times.
1302	61	1.11.20	II + —	17.12.20	Still under treatment.
1303	33	15.11.20	II + —	21.2.21	Still in hospital.
1300	13	28.11.20	II —	5.2.21	Eye palsies persist; loss of intellectual tone.

APPENDIX II.—E. Table showing the various sequelæ reported as occurring in the case of 271 patients attacked by encephalitis lethargica—*continued*.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
1319	18	26.11.20	II + -	22.1.21	Still under treatment for headache and ocular pain; tremors of hand.
1339	63	30.11.20	II + -	21.2.21	Still in bed and under treatment.
1348	14	10.12.20	—	14.2.21	Subject to fits.
1355	41	22.12.20	II + -	18.1.21	Still under treatment.
1365	36	24.8.20	II + -	1921	Some permanent paralysis.
594	6	4.12.19	II + + -	1.2.21	Still attends hospital for nervousness and insomnia.
601		8.1.20	II + -	4.2.21	Slight tremors of legs after walking.
606	41	6.1.20	II + -	7.2.21	Insomnia; tremors right arm; myopic.
608	42	17.1.20	II + -	4.4.20	(Died of malignant disease of liver.)
610	56	7.1.20	II + -	21.1.21	Mentally dull and irritable.
619	9	19.12.19	II + + -	4.2.21	Very dull.
620	13	18.1.20	II + -	4.2.21	Left squint; palsy of face; spastic gait. Mentally feeble; fits of temper.
627	17	12.1.20	II + -	8.2.21	Very neurotic and feeble.
628	45	14.1.20	II + -	7.2.21	Severe headaches.
635	28	24.1.20	II + -	7.2.21	Confined October, 1920; speech difficult when tired and mouth muscles twitch.
639	56	27.1.20	I -	—	(Subsequently—10.8.20—died of cancer of colon.)
642	44	20.1.20	II -	22.1.21	Difficulty in reading; some loss of memory; "feels like walking on air."
643	22	20.12.20	II + -	Feb. 1921.	Some paresis of legs.
650	58	24.12.19	II + -	28.1.21	Abnormally drowsy; "light duty" only.
652	25	8.1.20	II + -	25.1.21	Neurasthenic.
653	49	10.12.19	II + -	25.1.21	Mentally dull.
655	14	11.1.20	II + -	5.2.21	Mentally more childish than before.
658	7	26.12.19	II + + + -	Feb., 1921.	Marked chorea; also enlarged thyroid.
676	37	21.1.20	II + -	22.1.21	Lack of facial expression.
678	19	25.12.20	II -	26.2.21	Blind right eye; general paresis slightly improving.
679	29	11.11.19	II + -	3.2.21	Some eye palsy; transient somnambulism.
682	65	Jan. 1920	II -	25.1.21	Diplopia.
692	12	16.1.20	II + + + -	Jan. 1921.	Slight palsy L. eye.
705	62	29.12.19	I -	25.1.21	Emotional. Cannot walk along a straight line.

APPENDIX II.—E. Table showing the various sequelæ reported as occurring in the case of 271 patients attacked by encephalitis lethargica—*continued*.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
711	49	26.1.20	II -	27.1.21	Palsy right eye persists; much insomnia.
713	63	6.2.20	II + -	12.2.21	Has resumed medical practice, but not as fit as usual.
714	46	25.11.19	II + -	12.2.21	Slight tremor left hand.
715	53	12.1.20	II + -	5.2.21	Still dribbles. Tremors of hands; insomnia.
732	25	29.1.20	II -	4.2.21	Gets easily over-tired. Memory improved.
736	49	4.1.20	II + -	26.2.21	Very weak.
742	17	18.12.19	II + -	2.2.21	Permanent paralysis with tremors.
752	37	26.1.20	II + -	2.2.21	Neurasthenia.
757	51	18.2.20	II + -	1921	Disposition changed; previously jolly with his children, now quiet and does not speak. Sleeps badly; tremors of face when asleep but not when awake. Very fat.
758	11	13.2.20	II + -	Jan. 1921.	Abnormally passionate.
759	23	30.12.20	II -	Jan. 1921.	Very emotional.
760	45	Dec. 1919	I + -	Feb. 1921.	Mentally defective; speech slow. Slight facial palsy.
767	7	31.1.20	II -	4.2.21	Very nervous.
771	28	25.11.19	II + -	2.2.21	Mentally dull; memory impaired.
792	11	1.3.20	II -	—	(Died of appendicitis 5.11.20.)
793	2½	2.1.20	II + -	8.2.21	Mental condition bad; very irritable, can only squeal. (This state not consequent upon, but aggravated by, illness.)
796	3 months	23.2.20	I -	20.1.21	Ext. squint R. eye; general weakness; mentally backward.
798	29	10.2.20	II -	9.2.21	Slight mental dulness.
814	46	Feb. 1920	II + -	—	Apparent recovery: eventually the patient died in an attack of epilepsy, to which condition she had become subject.
826	31	24.3.20	II + -	12.2.21	Memory impaired.
827	18	4.3.20	II + -	11.2.21	Night sweats.
831	33	26.2.20	II + -	21.1.21	General weakness; tremors and diplopia.
843	33	1.4.20	II + -	Feb. 1921.	Paresis right arm and hand; articulation difficult.
845	16	March 1920	II + -	8.2.21	Eye palsy persists; abdominal reflex absent; knee jerks exaggerated.
860	55	—	II + -	10.2.21	"Not the woman she was."

APPENDIX II.—E. Table showing the various sequelæ reported as occurring in the case of 271 patients attacked by encephalitis lethargica—*continued*.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
864	58	25.4.20	II —	27.1.21	Pupils very sluggish to light; slight paresis of fingers. Much asthenia.
870	11	30.3.20	II + —	4.2.21	Mentally dull.
881	59	30.4.20	II —	8.2.21	Squints when tired; weak; neurotic and irritable.
889	25	11.4.20	II —	8.2.21	Apathetic, irritable and self-willed. Sight impaired.
890	37	Feb. 1920	II + —	8.2.21	Irritable.
899	—	17.5.20	II + —	7.2.21	Now myopic. Tremors left eye. Psychic disturbances, (reading of murders tempts her to commit such).
902	43	1.4.20	II + —	20.2.21	Tremors; mentally feeble.
911	50	12.4.20	I —	4.2.21	Chronically ill. General asthenia; head and body bent; shuffling gait; limbs stiff; face expressionless.
916	16	May 1920	II —	11.2.21	General muscular weakness, left side especially; mouth kept half open; tired on least exertion. Extremely passionate; severe headache; insomnia.
917	23	8.6.20	II + —	11.2.21	Still very weak, slow and apathetic.
920	20	7.5.20	II + + —	4.2.21	Weak left arm; both arms hypertonic; shoulders bent; melancholy.
929	21	7.5.20	II + —	Feb. 1921.	Palsy right leg; mentally sluggish.
930	20	2.6.20	II + —	Feb. 1921.	Eye palsies; paresis right arm; tremors. Mentally sluggish.
955	10	8.6.20	II + —	Feb. 1921.	Ocular signs persist, also face palsy; tremors.
956	48	18.5.20	II + —	26.2.21	Blurring of vision. Palsy left arm and leg; slowly improving.
960	27	13.6.20	II + —	Jan. 1921.	Very nervous.
962	16	22.6.20	II + —	2.2.21	Eyes protrude every three days. Right foot paralysed.
979	41	23.5.20	II + —	10.2.21	Irritable and melancholy.
980	14	23.6.20	II + —	10.2.21	Nearly normal, but still attends hospital at intervals.
985	11	13.6.20	II + —	28.2.21	Tremors in fingers and tongue. Fidgets at times; stuporose; subject to headaches and pain behind eyes. Nodding of head; right pupil sluggish to light.
1602	12	4.5.20	II + —	21.1.21	Occasional tremors. Temper bad; weak bladder.

APPENDIX II.—E. Table showing the various sequelæ reported as occurring in the case of 271 patients attacked by encephalitis lethargica—*continued*.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
1013	39	7.5.20	II + —	18.2.21	Squint, ptosis, diplopia, slight facial palsy; paresis legs.
1021	50	Xmas. 1919	II + —	8.2.21	Memory impaired.
1027	50	27.6.20	II —	16.2.21	Right ptosis; double squint and diplopia; paresis right deltoid; tremor right arm. Mentally slow.
1028	16	10.6.20	II + — —	16.2.21	General tremors; mentally dull.
1029	9	May 1920	II + — —	12.2.21	Palsy left hand; tremor left hand and arm; very dull.
1030	38	8.4.20	II + —	21.1.21	Tired after reading.
1052	48	11.7.20	II + —	21.2.21	Tremor after exertion.
1060	10	12.7.20	II + + —	11.2.21	Nystagmus; mentally slow? drowsy; insomnia.
1068	73	23.6.20	II + + —	7.2.21	Persistent headache.
1081	26	3.6.20	II + —	8.2.21	Diplopia at times; facial palsy; tremors both arms. Dull and irritable at times; cannot see to read, speech slow.
1095	12	5.6.20	III	26.2.21	Lack of energy.
1096	66	29.6.20	I	26.2.21	Eye palsies persistent. Still drowsy and mentally deficient.
1099	49	4.5.20	II —	26.2.21	Frequent vertigo.
1105	12	July 1920	II + —	11.2.21	Tremulous, lethargic, hot-tempered.
1107	19	25.7.20	II + + —	25.1.21	Occasional attacks of drowsiness.
1111	43	14.6.20	I + —	—	Very slow convalescence.
1113	18	6.6.20	II + —	Jan. 1921.	Very excitable and childish.
1120	13	8.7.20	II +	—	Nervous; excited; amnesia and stammering during convalescence. Practically recovered Jan. 1921.
1136	57	4.8.20	II + —	11.2.21	Some palsy both legs; tremors hands and feet.
1149	52	22.7.20	II + +	3.2.21	Very nervous and weak.
1161	38	9.8.20	II + — —	11.1.21	Recovering; still very weak.
1165	27	22.7.20	II	8.2.21	Marked contraction of leg and face muscles.
1166	16	3.7.20	II	11.2.21	Twitching of face.
1180	14	29.8.20	II + —	4.2.21	General tremors; irritable; diplopia.
1186	12	16.5.20	II + —	5.2.21	Pains in legs and arms.
1187	46	May 1920	II + — —	5.2.21	Lack of energy: headache; tremors; insomnia.
1191	23	18.6.20	II + —	5.2.21	Sleepy at times: very silent; tremors.
1192	46	June 1920	II + + —	5.2.21	Impaired vision.
1193	39	14.6.20	II + —	5.2.21	Vision impaired; muscular twitchings; insomnia.

APPENDIX II.—E. Table showing the various *sequelae* reported as occurring in the case of 271 patients attacked by encephalitis lethargica—*continued*.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
1196	44	28.9.20	II + —	25.1.21	Weakness; headache; unable to work.
1198	13	4.8.20	II + —	11.2.21	Weakness and pain in hamstrings.
1201	30	28.8.20	II + —	4.2.21	Great debility.
1211	24	15.7.20	II —	13.2.21	Twitching of face; not normal mentally.
1213	45	17.7.20	II + —	8.2.21	Tremor upper lids; persistent frontal headache; not so alert mentally.
1216	19	27.9.20	II + —	11.2.21	Abnormally sleepy.
1229	17	26.9.20	II + —	1.2.21	Slight tremors face.
1233	13	26.7.20	II + —	7.2.21	Mental impairment.
1234	41	21.5.20	II + —	5.2.21	Eyesight impaired; sleepy by day; nervous.
1235	10	11.5.20	II + —	5.2.21	Permanent paralysis left side; tremors; mentally deficient.
1237	64	21.9.20	II + —	26.2.21	Slight paresis left leg.
1238	72	16.10.20	II + —	26.2.21	Mental condition good, following detention in lunatic asylum. Slight paresis right arm.
1242	18	9.10.20	I	18.1.21	Still dull mentally.
1245	33	3.10.20	II + —	24.1.21	Still under treatment; nervous.
1254	28	25.10.20	II + —	Jan. 1921.	Slight palsy of eye; left side paresis; clonic spasms right leg.
1261	53	30.10.20	I	27.1.21	Athetosis.
1265	23	4.10.20	II + —	28.1.21	Slight facial palsy; sluggish.
1266	45	5.11.20	II —	4.2.21	Weakness right arm.
1278	43	9.10.20	II —	7.12.20	Great emaciation and slight rigidity.
1296	10	18.11.20	I + —	8.2.21	Still under treatment; improving.
1297	26	29.11.20	II + —	4.2.21	Recovering except for mammary abscess.
1313	58	6.12.20	II + —	31.1.21	Still paresis left arm and some tremor.
1320	30	7.12.20	II + —	25.1.21	Still dull and lethargic.
1328	18	11.12.20	II + —	1.2.21	Slight weakness of palate and of right side of body.
1333	14	16.12.20	I —	4.2.21	Much improved, but still asthenic.
1335	31	17.12.20	II —	1.2.21	Still under treatment.
1344	18	13.12.20	II + —	2.2.21	Still in hospital; eyesight troublesome.
1353	29	14.12.20	II + —	19.2.21	Recovering; some general paresis and epiphora.
1370	22	13.12.20	II —	4.2.21	General asthenia, mentally dull; ptosis (L.).
1399	23	18.11.20	I —	14.2.21	Mentally dull.
1407	45	14.11.20	I + —	16.2.21	Difficulty in reading and writing.

APPENDIX II.—E. Table showing the various *sequelae* reported as occurring in the case of 271 patients attacked by encephalitis lethargica—*continued*.

Schedule number.	Age.	Date of onset of illness.	Type of disease. (See footnote on p. 55.)	Subsequent history.	
				Date of inquiry.	Condition of patient.
1421	21	17.12.20	II + —	26.2.21	Tremors and athetosis during sleep; very weak and nervous.
1422	26	16.12.20	I —	26.2.21	Pain in throat and chest.
1423	21	22.11.20	II + —	26.2.21	No grip right hand.
1435	29	9.12.20	II + —	19.2.21	Tremors; sluggish mentally; childish.
1440	47	1.12.20	II + —	23.2.21	Optic atrophy left side; accommodation weak both sides.
1454	40	3.12.20	II + —	16.2.21	Recovering, but prone to tiredness.
1503	7	20.12.20	II —	Feb. 21, 1921.	Still in hospital; improving slightly.
1532	18	27.10.20	II + —	8.2.21	Slight nystagmus; cannot spell as before.
1673	5	13.10.20	—	24.1.21	Insomnia.
1681	45	1.12.20	—	Feb. 1921.	Slight eye palsy.
1732	17	27.12.20	II + —	1.2.21	Tremor of hand.
1531	57	26.12.20	II + —	7.2.21	Very neurasthenic.
1822	15	17.12.20	II + —	4.4.21	Physically and mentally weak; paralysis persists in pharynx and face; can walk a little.
1852	5	13.12.20	II + —	17.2.21	Vomiting continues every few days, also squint; otherwise improving.
1972	41	9.12.20	II + —	6.3.21	Shaky at times, lethargic; print soon becomes blurred. Will not be fit for business for some months.
574	19	15.12.19	II + —	17.2.21	Some muscular weakness, mentally dull.
2018	52	15.11.20	II + —	14.3.21	Right eye cannot distinguish light from darkness; still trembles; tired after walking.
2027	36	6.11.20	II + —	Feb. 1921.	Still sleepy; no residual palsies.
1800	30	6.12.20	—	16.2.21	Mental blurring; permanent paralysis.
1813	45	28.11.20	—	7.2.21	Slight dizziness and some ptosis.
1831	45	3.12.20	—	Feb. 21, 1921.	Slight trembling; speech affected; insomnia.
1842	65	21.11.20	—	1921	Marked tremors; athetosis.
1858	14	15.12.20	—	15.2.21	Irritable.
1860	48	7.12.20	—	Feb. 1921.	Slight apathy.
1863	36	1.6.20	—	14.2.21	Ocular palsies still present.
1887	4	5.5.20	—	1921	Extremely wicked and uncontrollable; lethargic.
1888	37	1.6.20	—	Feb. 1921.	Athetosis; mentally clouded.

BIBLIOGRAPHIES.

The literature of encephalitis lethargica is represented in the following bibliographies by references to the writings of some 1,200 authors, which include over 1,500 separate articles contributed during the years 1917-1921, inclusive.

Bibliography No. II, unlike Bibliography No. I, does not deal exclusively with the current literature peculiar to encephalitis lethargica, but contains also references to writings of historical interest together with a few references to such relevant subjects as the cerebro-spinal fluid, epidemic hiccup, other varieties of encephalitis, &c.; to this list have also been added certain recent references to encephalitis lethargica which were too late for inclusion in Bibliography No. I. The bibliography numbers are carried on in sequence from the first to the second list and are those used in the text of this report by all three authors concerned.

Apart from their authoritative use in connection with this report it is hoped that the three bibliographies which follow are comprehensive enough to form a guide to the general world literature of the subject; they are certainly not intended to suggest a measure of our first-hand acquaintance with what has been written, both at home and abroad, concerning encephalitis lethargica. With most of the outstanding reviews and reports of this disease we have, of course, made ourselves familiar, but many of the references, we freely admit, have been collected by digging in other peoples mines of information; during these labours also we have tried to check the references to articles published either in those medical journals which cannot as a rule be consulted in this country, or in those continental publications which were not received by our libraries during the War.

Among the sources to which we have constantly turned for bibliographical information we gratefully mention the *Index Catalogue of the Library of the Surgeon-General's Office*, United States Army; the *Index Medicus* (Washington); the *Quarterly Cumulative Index to Current Medical Literature* (Chicago); the *Journal of the American Medical Association*; the *Revue Neurologique* (Paris); the *Centralblatt für Bakteriologie, Referate*; the *British Medical Journal* (Epitome Section), and *Medical Science* (Abstracts and Reviews).

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The table below affords some idea of the widespread experience of encephalitis lethargica and shows approximately the comparative literary output in the various countries concerned:—

Country.	Number of publications containing references to E.L.	Total number of references.	Publications chiefly concerned.
Argentina	6	31	<i>Semana médica</i> , Buenos Aires.
Australia	2	7	—
Austria	2	29	<i>Wiener klinische Wochenschrift</i> , Vienna.
Belgium	4	7	—
Brazil	5	13	—
Canada	2	5	—
China	1	3	—
Cuba	1	1	—
Denmark	1	12	—
Egypt	1	1	—
Finland	1	2	—
France	59	529	<i>Bulletins et mémoires de la Société médicale des hôpitaux de Paris</i> ; and <i>Bulletin de l'Académie de médecine</i> , Paris.
Germany	27	115	<i>Münchener medizinische Wochenschrift</i> , Munich; and <i>Berliner klinische Wochenschrift</i> , Berlin.
Germany and Austria	6	19	—
Great Britain	38	222	<i>British Medical Journal</i> , London; and <i>The Lancet</i> , London.
Holland	1	15	—
Honduras	1	2	—

Country.	Number of publications containing references to E.L.	Total number of references.	Publications chiefly concerned.
India	2	7	—
Italy	42	186	<i>Il Policlinico</i> , Rome; and <i>La Riforma Medica</i> , Naples.
Japan	1	2	—
New Zealand	1	2	—
Norway	2	3	—
Peru	2	5	—
Phillippines	2	2	—
Poland	2	5	—
Portugal	2	2	—
Roumania	2	3	—
South Africa	2	5	—
Sicily	1	1	—
Spain	8	17	—
Sweden	3	5	—
Switzerland	8	35	<i>Schweizerische medizinische Wochenschrift</i> , Basel; and <i>Revue médicale de la Suisse Romande</i> , Lausanne.
United States of America	78	255	<i>Journal of the American Medical Association</i> , Chicago; and <i>Archives of Neurology and Psychiatry</i> , Chicago.
Uruguay	2	10	—
Venezuela	1	2	—

NOTA BENE.

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BIBLIOGRAPHY III.

Subject Index.

In the following Index an attempt has been made to classify the subject matter of the various contributions to the general literature of Encephalitis Lethargica, and the figures printed in ordinary type refer to the authorship, title, date, etc. of such contributions as are shown in Bibliographies I. and II. In addition, the subject matter of the report itself is here indexed by means of figures in heavy type which refer to pages.

N.B.—To avoid constant repetition it may be stated that the Local Government Report on Encephalitis Lethargica (see 558a, Bibl. I.) contains allusions to the majority of subjects catalogued in this Index.

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