The 1956 Lancet Editorial First Proposing the Name "Benign Myalgic Encephalomyelitis" Reformatted

Although the name of the author is not given for the 1956 Lancet editorial article "A New Clinical Entity" first formally proposing the name "benign myalgic encephalomyelitis" for a likely new clinical entity identified from multiple similar outbreaks of disease, Dr. E.D. Acheson was almost certainly its author.

Acheson later wrote an expanded paper in 1959, "The Clinical Syndrome Variously Called Benign Myalgic Encephalomyelitis, Iceland Disease and Epidemic Neuromyasthenia," that examined in detail 14 outbreaks of disease and argued further that "benign myalgic encephalomyelitis" was the most appropriate name for a new clinical entity based on the common features of these outbreaks.

https://www.me-international.org/uploads/1/2/7/6/127602984/acheson_amjmed-1959.pdf

A PDF of the original Lancet article "A New Clinical Entity" can be found here:

https://mecfsj.files.wordpress.com/2019/05/lancet_e383a9e383b3e382bbe38383e383881956_me.pdf

I found in the original formatting this very important article in ME history was difficult to read. I made the following transcript adding paragraph breaks and other format changes that others also may find more user-friendly for reading than the original.

Jerrold Spinhirne, October 12, 2020

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A New Clinical Entity?

IN 1917 VON ECONOMO [1] reported a small outbreak of an illness in which the main features were fever, stupor, and ophthalmoplegia [paralysis or weakness of the eye muscle]: 2 of his 13 patients died and at necropsy there was evidence of inflammation of the brain substance.

During the next two years a great many similar outbreaks were recorded and by 1921 the disease had reached epidemic proportions in almost every country in Europe. [2] In spite

of perplexing variations in the clinical picture from case to case, locality to locality, and even from season to season. [2] it soon became clear that for practical purposes a new clinical entity had appeared. [3]

In 1924, 5039 cases of encephalitis lethargica [also called sleeping sickness] were notified in England and Wales alone, [4] but by the beginning of the next decade confirmed cases of this dangerous disease had become sporadic and by 1939 they were extremely rare. [5]

By the end of the late war, the centre of interest had shifted to poliomyelitis as by far the most prevalent and disabling infection of the nervous system. The work of RITCHIE RUSSELL and others [6] focused attention on the importance of diagnosis in the pre-paralytic stage; and from other sources [7, 8] there was evidence of a change in the epidemiology of poliomyelitis.

Against this background of intense interest in poliomyelitis and its problems came reports of outbreaks [9–11] and isolated cases [12, 13] which, for one reason or another, led to difficulties in diagnosis. Broadly, these can be divided into those in which the cerebrospinal fluid (C.S.F.) is abnormal and those in which it is normal.

Of the abnormal group (group I), only in the series described by LAURENT [10] was no convincing causative organism isolated. The conditions described by KELLEHER [12] and JENNINGS [13] proved to be aberrant poliomyelitis, and there was presumptive evidence of the same disease in the outbreak reported by BARRETT [9] from Cambridgeshire in 1949.

The Coxsackie group of viruses has also been implicated in this group: [11, 14] in such cases pleocytosis [increased lymphocyte cell count in C.S.F.] is the rule and signs of parenchymal [relating to or affecting the functional tissue of an organ] damage to the nervous system are very uncommon. Much more perplexing outbreaks are those in which no changes are found in the C.S.F. (group II).

Since we discussed these illnesses in 1954 under the noncommittal title, Not Poliomyelitis, [15] another epidemic with similar features has been reported from Durban [16, 17] and two further outbreaks are described in this issue by Dr. SUMNER and by Dr. RAMSAY and Dr. O'SULLIVAN.

Only a brief description [18] has so far been published of the alarming outbreak at the Royal Free Hospital last year, but there are arguments for including it in this group. In

none of these cases has it been possible to incriminate the poliomyelitis or Coxsackie virus, nor indeed has any other known infective agent been isolated.

There seem good reasons, in our present state of ignorance, for placing in a third and intermediate group the epidemic which took place in Akureyri, Iceland, in the winter of 1948-49, [19] and about which Dr. SIGURDSSON and Dr. GUDMUNDSSON write on p. 766.

In all 8 cases examined the C.S.F. was abnormal; on the other hand, the protracted course and mental symptoms described by Dr. SIGURDSSON are prominent symptoms in group II. The outbreak in the nurses training school in the University of Pennsylvania in 1945 [20] is also difficult to classify since it happened before the isolation of the Coxsackie viruses: there was pleocytosis in 2 out of 5 cases.

The unusual epidemic reported by WALLIS from Cumberland in 1955 [21] has many features of group II—notably vertigo, diplopia [double vision], myalgia, cervical lymphadenopathy, and protracted convalescence with mental symptoms. Unfortunately there is no information about the C.S.F. The recorded atypical outbreaks can thus be grouped as follows :

Group I: C.S.F.: Usually abnormal

Laurent [10] (1947) Virus: Unknown
Kelleher et al. [12] (1949) Virus: Poliomyelitis
Curnen et al. [11] (1949) Virus: Coxsackie
Jennings et al. [12] (1949) Virus: Poliomyelitis
Barrett et al. [9] (1952) Virus: Poliomyelitis
Galpine and Macrae [14] (1953) and others Virus: Coxsackie

Group II: C.S.F.: Normal in nearly all cases

Adelaide [22] (1949) Virus: Unknown
New York State [22] (1950) Virus: Unknown
Middlesex Hospital [24] (1952) Virus: Uncertain
Coventry [25] (1953) Virus: Unknown
Berlin (1954) (Summer) Virus: Unknown
Durban [17] (1955) Virus: Unknown
Royal Free Hospital [18] (1955) Virus: Unknown
Hampstead (1955) (Ramsay and O'Sullivan) Virus: Unknown

Group III: C.S.F.: [Mixed findings]

Pennsylvania [20] (1945) Virus: Unknown; C.S.F.: Abnormal 2/5
Alkureyri, Iceland [19] (1948) Virus: Unknown; C.S.F.: Abnormal 8/8
Cumberland [21] (1955) Virus: Unknown; C.S.F.: Unknown

Of the 8 outbreaks in group II, all except that at the Royal Free were initially confused with poliomyelitis, and all occurred during or shortly after the seasonal period of prevalence of poliomyelitis. The three British outbreaks [18, 24, 25] were in late summer, in contrast to the former peak incidence of encephalitis lethargica in the first three months of the year. [2]

Five outbreaks took the form of dramatic localised epidemics, four of which were in nurses' homes. Dr. RAMSAY and Dr. O'SULLIVAN describe cases in the neighbourhood of one of these outbreaks, and HILL [17] had a similar experience in Durban.

The attack-rate in closed communities is high. The onset in this group is usually acute with systemic prodromata such as are common in poliomyelitis. In contrast, fever is usually low and may be absent. [14, 18, 25] The course is generally two to eight weeks but occasionally symptoms may last for months. Relapses are frequent. Usually the immediate outcome is favourable but in a few cases paresis or mental sequelae may be incapacitating for many months. [22, 23, 26]

Depression, emotional lability, or irritability in convalescence have been a constant feature in all group-II outbreaks. Although previous experience has shown that a long period of observation will be necessary before the harmlessness of the disorder is assured, it can at least be said that the immediate mortality-rate of nil is in striking contrast to the epidemic infections of the nervous system previously described; [4] and this in itself is very encouraging.

Among the more characteristic features of group II are the severe muscular pains, often accompanied by exquisite tenderness, which often dominate the clinical picture. [22–25] As WHITE and BURTCH 23 have pointed out, these pains differ from those of poliomyelitis in that they are not simply a short-lived precursor of paresis but may last for weeks. Most commonly they affect the neck, back, or limbs but there may also be Bornholm-like chest and abdominal pains. [17, 18, 23, 25]

Continuous or intermittent painful muscular spasms were noted in the outbreaks at the Middlesex and Royal Free Hospitals, and they are also reported by Dr. RAMSAY and Dr. O'SULLIVAN. In nearly every patient there are symptoms or signs of disease of the central nervous system, but the weight and site of the damage vary considerably from outbreak to outbreak.

The Hampstead and Berlin epidemics illustrate this variation. The innervation of the eye muscles (diplopia and nystagmus) and the seventh and eighth cranial nerves (deafness, hyperacusis, vertigo, and facial weakness) suffer most commonly. Sensory symptoms and signs are common and pyramidal signs have also been observed.

Some patients in Adelaide and Durban and at the Middlesex Hospital had retention of urine. The paresis, usually short-lived but occasionally persisting for weeks or months, is in itself an interesting problem, for in many cases it is not accompanied by the classical disturbances of tone and reflexes which would point to damage in the anterior horn or pyramidal tract. [23, 25, 26]

Pain, muscular spasm, and involuntary movements often make the degree of palsy difficult to assess. In this connection the striking electromyographic records obtained by Dr. RAMSAY and Dr. O'SULLIVAN are of great interest. Although they do not as yet point to the exact nature of the lesions, they may provide evidence of organic paresis in patients who might otherwise be suspected of hysteria, and in a disease at present so bereft of positive laboratory findings they may be a help in diagnosis in the future.

The outbreaks mainly differ in the severity and site of the damage to the nervous system; but the lymph glands are another point of difference. Enlarged lymph glands, particularly in the posterior cervical triangle, were prominent in the Hampstead and Royal Free cases, and they were also noted in 4 cases by WHITE and BURTCH [23] and in the more doubtful Cumberland
outbreak. [21]

In retrospect lymphadenopathy was found to have been present in 2 of the 14 cases reported from the Middlesex. [27] Hepatitis and splenomegaly may also turn out to be part of the picture. It is doubtful whether the absence of these features in the other reports can be attributed entirely to observer error [27] and it must be accepted as a real discrepancy.

A study of the available material in group II shows sufficient common ground to suggest that this is a new clinical entity which may be expected to appear again here or elsewhere in the late summer and autumn.

From the purely practical standpoint it would be useful to have a name for this syndrome. As the most helpful single feature in the recognition of this syndrome in the past has been the predominately normal cerebrospinal fluid, the names which have already been suggested, "Iceland disease" and "Akureyri disease," are not really appropriate.

The objections to any but a purely descriptive name for a disorder without a known cause or established pathology are obvious. For this reason, the term "benign myalgic encephalomyelitis" may be acceptable.

It in no way prejudices the argument for or against a single or related group of causal agents; and it does describe some of the striking features of a syndrome characterized by (1) symptoms and signs of damage to the brain and spinal chord, in a greater or lesser degree; (2) protracted muscle pain with paresis [partial paralysis, muscle weakness] and cramp; (3) emotional disturbances in convalescence; (4) normal C.S.F.; (5) involvement, in some variants, of the reticuloendothelial system [part of the immune response system]; (6) a protracted course with relapses in severe cases; and (7) a relatively benign [no immediate mortality] outcome.

It remains to identify this syndrome more precisely; but we believe its characteristics are now sufficiently clear to differentiate it from poliomyelitis, epidemic myalgia, glandular fever, the forms of epidemic encephalitis already described, and, need it be said, hysteria.

1. von Economo. C. Wien. klin. Wschr. 1917, 30, 581.
2. Hall, A. J. Epidemic Encephalitis. Bristol, 1924.
3. Kinnier Wilson, S. A. Lancet, 1918, ii, 7.
4. MacNalty, A. S. Epidemic Diseases of the Central Nervous System. London, 1927.
5. Epidemic Encephalitis. 3rd Report by Matheson Committee. New York, 1939.
6. Russell, W. R. Poliomyelitis. London, 1956.
7. Dauer, C. C. Amer. J. Hyg. 1948, 48, 133.
8. MacLean, F. S. New Zealand Department of Health. Annual Report of Director General, 1945, p. 90.
9. Barrett, A. M., Gairdner. D., McFarlan, A.M. Brit. med. J. 1952, i, 1317.
10. Laurent, L. J. M. Proc. R. Soc. Med. 1947, 40, 927.
11. Curnen, E. C., Shaw, E. W., Melnick, J. L. J. Amer. med. Ass. 1949, 141, 894.
12. Kelleher, W. H., Bratton, A. B., MacCallnm, F. O. Brit. med. J. 1949, ii, 213.
13. Jennings, G. H., Hamilton-Paterson, J. L., MacCallum, F. O. Ibid, p. 210.
14. Galpine, J. F., Macrae, A. D. Lancet, 1953, i, 372.

15. See leading article, Ibid, 1954, ii, 1060.
16. Editorial, S. Afr. med. J. 1955, i, 331.
17. Hill, R. W. Ibid, p. 314.
18. See Lancet, 1955, ii, 351.
19. Sigurdsson, B., Sigurjonsson. J., Sigurdsson, J., Thorkelsson. J. Gudmundsson, K. R. Amer. J. Hyg. 1950, 52, 222.
20. McConnell, J. Amer. J. med. Sci. 1945, 209, 41.
21. Wallis, A. L. Lancet, 1955, ii. 290 ; Ibid, p. 1091.
22. Pellew, R. A. A. Med. J. Aust. 1951, i, 944.
23. White. D. N., Burtch, R. B. Neurology, 1954. 4. 506.
24. Acheson, E. D. Lancet, 1954, ii, 1044.
25. Macrae, A. D., Galpine, J. F. Ibid, p. 350.
26. Adnams J. N. Personal communication.
27. Acheson, E. D. Lancet, 1955, ii, 394.