Encephalitis

Morris Schaeffer
MEDICAL BACTERIOLOGIST*

Pathologists have recognized for a long time that the tissues can respond only in a limited manner to certain invading irritative substances. This is especially true of the brain. Thus, a number of different agents can produce an encephalitis (a nonspecific term meaning, merely, inflammation of the brain) which may be difficult or impossible to differentiate on the basis of the clinical picture alone, or even the appearance of gross and microscopic pathologic changes.

Encephalitis may be caused by a large variety of offending substances including physical and chemical agents, poisons, toxins, bacteria, protozoa, spirochetes, rickettsiae, and viruses. The last-mentioned agents are of special interest because certain viruses produce types of encephalitis which have important public health aspects in that they are readily transmissible through the agencies of animal reservoirs and arthropod vectors and are present in certain localities in endemic and epidemic form.

The virus encephalitides may be divided into two main groups, primary and secondary.

Secondary encephalitides are caused by, or associated with, viruses not ordinarily considered encephalitogenic, which produce familiar diseases such as herpes simplex, measles, mumps, chickenpox, influenza, lymphogranuloma venereum, vaccinia, and infectious hepatitis. A similar form of encephalitis may follow vaccination against smallpox and rabies. These types also have been termed postinfection or postvaccination encephalitis.

The primary virus encephalitides are caused by viruses which have a primary affinity or tropism for the central nervous system and include such diseases as the St. Louis type of encephalitis, Eastern equine encephalomyelitis, Western equine encephalomyelitis, Japanese B encephalitis, Venezuelan equine encephalomyelitis, Russian Far East encephalitis (tick-borne, spring-summer encephalitis), lymphocytic choriomeningitis, louping ill, rabies, and B-virus. In addition, there is a group of virus encephalitis infections less well defined such as pseudolymphocytic choriomeningitis, swineherd disease, and a form of leukencephalitis. There are others still more obscure and exotic like West Nile, Bwamba Fever, Semlike Forest, and Bunyamwera, for which viruses have been isolated from mosquitoes; and in a few instances either the virus or antibodies have been found in the blood of man, but their role or significance has not as yet been clearly indicated. Von Economo's disease, an acute and chronic disseminated encephalomyelitis, is another one of the primary encephalitides which appeared in epidemic form in this country about 30 years ago but rarely has been seen since. A virus etiology for this disease never has been established definitely. The first three diseases mentioned above are the principal epidemic encephalitis problems in this country at present.

It appears therefore that there is a large number of specific agents already known and categorized to some extent which are capable of producing encephalitis; but it is obvious also that there remain other encephalitogenic viruses still to be uncovered and classified. Thus we are dealing with a group of diseases which present many facets of considerable complexity.

Out of the somewhat scrambled and spotty picture which we have of the natural history of the epidemic forms of encephalitis, the information presently available would indicate that the viruses find harborage in a wide variety of birds and fowl and are transmitted among them, and incidentally from them, to man and other mammals (horses in the equine types of encephalitis) by different species of arthropod vectors: mites, lice, mosquitoes, and ticks. There is the suggestion (awaiting adequate confirmation) that the St. Louis encephalitis virus may be propagated by transovarian passage in chicken mites. This would add another missing link and explain one way in which the disease, primarily of summer incidence, may be maintained through the winter. Epidemics may be expected to occur during the summer in potential

* Virus and Rickettsia Section, Montgomery, Ala.

localities where the situation, with respect to climatic conditions for the breeding of arthropod vectors, permits the cyclic chain of events to be completed.

Similarities of the clinical and epidemiologic aspects of some of these diseases and the multiplicity of types and strains of their etiologic agents make it necessary to study elaborately the characteristics of the causal agents, all their possible reservoirs and vectors, and their laboratory diagnosis. The physician and health officer must depend almost entirely on the laboratory for accurate diagnosis, and upon the collaborative efforts of the virologist, epidemiologist, entomologist, and clinician for a complete understanding of these diseases in order to formulate means for their control and prevention.

At present an absolute diagnosis can be made only in one of the following ways:

1. Isolation of the virus from the central nervous system of fatal human cases or horses (in equine encephalomyelitis) involved in the locality. Occasionally virus may be isolated from the blood or spinal fluid of patients if obtained very early in the disease, but this is not dependable. In order to preserve the viability of the virus, material for virus isolation must be frozen (using dry ice) immediately and kept that way until it reaches the laboratory for animal inoculation.
2. Demonstration of the formation of specific antibodies against one of the viruses in the blood of the suspected patient during the course of illness. For this, two blood specimens must be obtained. The first (acute phase specimen) should be obtained as early as possible after onset, preferably within the first 4 days of the disease; the second (convalescent phase specimen), 3 or 4 weeks after onset. At least 20 cubic centimeters of blood should be drawn asceptically; the serum should be separated and saved in the refrigerator in a sterile, sealed container until the second specimen is obtained and the paired specimens, together with appropriate clinical and epidemiologic data, forwarded to the laboratory. No refrigeration is necessary in transit for blood or serum specimens to be tested for antibodies, provided there is no unusual delay. The tests for antibodies are made by complement fixation or neutralization tests. A significant rise in titer between the first and second specimens is accepted as presumptive evidence of infection.

There is no specific therapeutic agent as yet available for any of these diseases. Preventive measures have not been fully formulated but, on the basis of our present knowledge, would consist of vector control and vaccination. Effective vaccines have been prepared for some of these diseases, but their general use is not advocated except in special situations. Studies are in progress with the hope of filling in the many remaining gaps in our understanding of the epidemiology of the encephalitides and ultimately of their complete eradication.

Eastern Equine Encephalomyelitis

R. E. Kissling
VETERINARY PATHOLOGIST*
Harry Rubin
ASSISTANT VETERINARIAN**

Eastern equine encephalomyelitis is an acute and highly fatal infectious disease caused by a virus which exhibits a wide host range, including horses and mules, pigeons, pheasants, monkeys, and man. It is closely related to, but immunologically distinct from, the viruses of St. Louis encephalitis and Western and Venezuelan equine encephalomyelitis. The virus has a widespread distribution in the tissues of the infected host, but the symptoms of the disease are referable to inva-

* Virus and Rickettsia Section, Montgomery, Ala.
** Rabies Control Section, Montgomery, Ala.