of the State, knows no geographical confines of political subdivisions, and most important, is above local influences.

In such a case the law seems to be settled in California that a medical practitioner may bring a private suit on his own behalf to prevent the unlicensed person from practicing medicine, because, by practicing, the unlicensed man will interfere with the exclusive privilege or property right to practice medicine granted the physician.

Peculiarly, the court reasons it has power to prevent a wrong to the public because it will deprive a physician of an income; yet as late as six weeks ago it denied the State an injunction to prevent a person from practicing medicine whose license to practice was a limited one, not medical in character.

So under this theory of the law the test seems to be loss of money as opposed to a possible injury to humanity.

Perhaps, as I stated before, the remedy for a situation such as this lies with the legislature.

State Building.

POLIOMYELITIS—THE LOS ANGELES EPIDEMIC OF 1934*†

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I

THE problem of poliomyelitis is still controversial, as shown by the increasingly voluminous literature; but each contribution adds information to the aggregate which will some day tell the story of this dreaded disease.

Following the 1930 epidemic in Los Angeles, we reported [1] unusual findings which were questioned by a few on the grounds of inaccurate diagnosis. The findings in the present epidemic of more than 1,800 cases were still more "atypical," and worthy of discussion. The same rigid diagnostic criteria, of either definite neurological findings or conclusive spinal fluid changes, were adhered to.

Representatives of the United States Public Health Department, the California State Department of Health, the Rockefeller Institute, and the Mayo Clinic were here during the epidemic, and their detailed reports on certain phases of the outbreak will probably include some mention of the peculiarities of the disease, as seen here this year. Our observations were largely clinical, and did not include a search for the causative agent; although monkey inoculations and cultural work were done in our laboratories and will be reported independently.

THE CAUSATIVE FACTORS IN THIS EPIDEMIC

We feel certain that the causative agent in this epidemic was a different strain of virus, or that some unusual factors were pertinent, because of (1) the very different clinical picture; (2) the incidence of the disease among adults, particularly doctors and nurses; (3) the appearance of mild symptoms in a few patients who had had poliomyelitis; and (4) the failure of convalescent serum to alter the course of those first few fatal cases seen early in the epidemic. This assumption is in keeping with the work of Burnet and Macnamara,[2] who showed conclusively, by neutralization tests, that different strains of virus exist in monkeys. The clinical pictures in our cases might have been altered by complicating antecedent or coincidental infections, for it is of interest to note that the high seasonal incidence of diphtheria at the time of the outbreak dropped sharply as poliomyelitis increased still more rapidly; that 24 per cent of the poliomyelitis cases admitted to the hospital showed positive diphtheria throat cultures; and that "sore throat" was a very common complaint in this epidemic. Diphtheria toxin notoriously contains a neuotrophic fraction.

Again, the fact that a large percentage of cases (approximately 16 per cent) suffered a preceding enterocolitis, often with mild encephalitic symptoms, suggests the possibility of a primary intestinal infection, with neurological complications; or a possible gastro-intestinal tract atrium of poliomyelitis virus. Neurotrophic toxins of gastro-intestinal origin are not uncommon. Botulism is an outstanding example. Several other organisms have been incriminated, including the typhoid-enteritidis group, and the dysentery bacillus. A mild epidemic of bacillary dysentery in Los Angeles in 1932 was complicated with a high percentage of encephalitis and six deaths, the latter showing inflammations of the brain and cord quite independent of the dehydration. Dysentery bacilli were found in the stools of several patients in this epidemic of "atypical" poliomyelitis.

Toomey and Wolfgang [3] demonstrated the presence of a toxin in the gastro-intestinal tract of monkeys ill with poliomyelitis which could be neutralized with convalescent poliomyelitis serum, and which produced ileus when introduced into the intestines of healthy animals. This probably represented involvement of the sympathetic plexuses, a step removed from the central nervous system. Infection of the latter by way of the digestive tract, and the lymphatics adjacent to the inferior mesenteric and lumbar spinal arteries, is not improbable. European investigators have infected monkeys by tube-feeding, and by injecting, through the intestinal wall, virus-containing media. Flexner [4] believes that this avenue is not an etiologic factor in human poliomyelitis. However, those of our cases with marked intestinal symptoms and involvement of the lumbar cord, and without symptoms referable to the respiratory tract or the area of the usual portal of entry along the olfactory pathway,[5,6] suggest this possible atrium of infection.

The adult incidence was another reason for suspecting a different strain of virus. The majority of cases (63 per cent) were in the age groups up to fifteen years, but the relative numbers of those in the late second, third, and fourth decades of life were increased. If, as we believe, a large majority of the adult population of urban communities are immune to poliomyelitis,[7] as a result

* From the Los Angeles County Hospital.
† See also pages 108 and 111.

of repeated contact, and mild, unrecognized, or subclinical infection, then these patients from a community of this size should have been adequately protected. Particularly is this true of doctors and nurses; yet the incidence among this class was far greater than among the population at large, and these patients were, on the average, far the sickest. In the case of those working in the Communicable Disease building, fatigue and breaks in technique were undoubtedly factors, for the epidemic precipitated so suddenly that help and space were at first inadequate, and nurses and doctors worked long hours in crowded rooms.

BORDER-LINE CASES

A few patients were seen who gave histories of having had poliomyelitis and who showed symptoms of mild systemic intoxications, with some peripheral findings. These were not proved cases, but the symptoms were of intractable origin, and suspiciously those of the prevailing epidemic disease.

During the spring of 1934 we lost eleven cases of the bulbar type, a mortality rate of 14.4 per cent of a total of seventy-six cases for that period. These patients were treated with large amounts of convalescent serum by all routes. They showed, on postmortem examination, medullary changes typical of poliomyelitis. The disproportion of bulbar to spinal types suggested a strain of virus with marked affinity for the higher portions of the central nervous system, a predilection fortunately not shown throughout the epidemic. The failure of convalescent serum to help these patients also suggested a different strain of virus, although we doubt the value of any therapy in cases with involvement of the cardio-inhibitory or respiratory centers in the medulla. The term "bulbar polio" is loosely used to include those cases showing respiratory failure from intercostal (thoracic) or phrenic (cervical) nerve paralysis, or those with transient cerebral changes incident to the initial toxemia.

ROSENOW'S FINDINGS

The work of Rosenow of the Mayo Clinic has been reported from Rochester.[8] We are not prepared to comment on his findings, except to say that he found in pharyngeal and nasal smears from patients streptococci which were morphologically and cataphoretically similar to those previously found in proved cases of poliomyelitis; that these streptococci produced symptoms in animals, and could be recovered at necropsy; that filtrates from emulsions of spinal cords of these animals produced typical symptoms of poliomyelitis when injected into other animals; that vaccines made from these organisms gave positive skin reactions for susceptibility in 80 per cent of those patients tested within the first two days of illness, and showed negative reactions on these same individuals after their recoveries; and that 76 per cent of those in intimate contact with the disease and not contracting it showed an immunity by this test. We are not convinced that this streptococcus is the answer to the question of etiology, but we do agree with Doctor Rosenow that it *may* represent a mature form of an ultramicroscopic and filterable organism, or *may* be a symbiotic relation. The incidence of the disease among casual contacts and its epidemic tendencies still classify it as "contagious," and we still feel that the frequency of "isolated" cases indicts the "healthy carrier."

PATHOLOGICAL STUDIES

Opportunity for pathological study was afforded only in cases of the fatal cerebral type. The microscopic changes were those characteristic of most central nervous system inflammations, namely, a perivascular infiltration of small, mononuclear, round cells forming a "collar" about the blood vessels to obstruct or narrow their lumen and effect a vascular congestion with adjacent cellular edema and degeneration. Poliomyelitis has a tendency to pick out isolated groups of nerve cells instead of affecting large areas, and in the cerebral type seems to have a predilection for the medulla and basal ganglia. Although the characteristic cord changes are those of degeneration of the motor cells of the anterior horns, occasional cases show changes in the lateral and posterior horns as well. One case came under observation which showed definite posterior horn-cell degeneration, an interesting observation in view of the clinical sensory findings, to be discussed later. Generalized lymphatic hyperplasia, as described by Burrows,[9] was noted.

IMMUNITY

Immunity has been considered in the discussion of adult morbidity. The tendency to relapse, after apparent recovery, was evidence of poor immunologic response on the part of the patient, or poor antigenic properties of the causative agent of the disease, as seen in this epidemic. An interesting observation was that reported by Howitt,[10] in which she noted that the convalescent serum of patients treated with immune serum, and thus passively immunized, showed less virus-neutralizing power than the convalescent serum of patients who were not treated specifically, and who developed their own antibodies. This is in accord with immunologic concepts of the superiority of active immunization, and would be expected of a disease such as poliomyelitis in which the immunity afforded by infection is ordinarily adequate and permanent. It is, however, the first time it has been demonstrated by a controlled series of cases. She also showed that the serum of recovered cases contained more virus-neutralizing power than the serum of severely paralyzed patients. Obviously, the immunologic response was greater in the recovered cases.

SYMPTOMS AND SIGNS

Symptoms.—The poliomyelitis seen here this year was so variable in its manifestations, that no syndrome can be described which accurately portrays the clinical picture. A majority of cases showed some degree of initial toxemia, but in many there was a conspicuous absence of the early neurological changes, or localizing findings, which ordinarily help to differentiate preparalytic poliomyelitis from the systemic phase of other acute infections. A few cases were afebrile during the

entire period of observation. A few were without the severe frontal, or postorbital, headache and painful occulomotion, that have heretofore been largely present in our experience. Many complained of sore throat, often without evidence of local inflammation or adenopathy. Many cases suffered an initial diarrhea, often with abdominal cramps, sometimes with vomiting, and occasionally with mental dullness or lack of power of concentration, suggestive of cerebral involvement. These appeared to be primary gastro-intestinal infections, some with, and some without, a generalized toxemia. Nearly every patient complained of some pain in the cervical or lumbar areas, but this was less severe, on an average, than formerly. Stiffness of the neck and spine was often absent, but in spite of this occasional flaccidity, spinal flexion, with stretch of the dura about the inflamed nerve roots, caused cervical or lumbar pain in most cases. This "spine sign" is usually the earliest neurological finding. Headache, or pain in the back or abdomen, induced or accentuated by slight trauma, was noticeable, even in those cases showing no increase in intrathecal pressure. The degree and duration of muscle pain, tenderness and severe cramping, were out of proportion to the motor phenomena. Some cases apparently suffered no premonitory symptoms, but developed neurological findings very suddenly at the onset.

Signs.—The objective findings were as atypical as the subjective symptoms. The frequency of a positive "spine sign" without spinal rigidity has been mentioned. Reflexes remained intact, although often diminished and frequently asymmetrical, in a large majority of cases. Muscle weakness was often very mild or transient, but pain on motion was sometimes pronounced. Frequently, muscle checks showed no weakness nor asymmetry, sometimes no pain, but a marked fatiguability of the affected extremity on repeated tests. In some cases there was a delayed fatiguability that was very persistent, and was aggravated by testing. Sensory phenomena seemed to be uniformly out of proportion to motor changes. Muscle tenderness was often acute. Hyperesthesia was pronounced, and in some cases was localized and contralateral to the paresis (Brown-Séquard phenomena, due to unilateral myelitis involving the lateral spinothalamic tract). An occasional case developed temporary surface anesthesia over areas corresponding to the nerve distribution from individual cord segments. In some cases the degree of abdominal pain, distention, and vomiting, was quite alarming, but absence of abdominal rigidity precluded the probability of peritoneal inflammation, while associated urinary retention and lumbar and lower extremity pains seemed to incriminate the sympathetic and central nervous systems. Also, the sudden relief after spinal puncture, even in cases showing no increased intrathecal pressure, indicated a paralytic ileus, a phenomenon not common in poliomyelitis, particularly of such low virulence. Further evidence of sympathetic involvement was the common finding of localized sweating over the surface of the affected extremity.

(To be continued)

THE ORTHOPEDIC TREATMENT OF CHRONIC ARTHRITIS*

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DISCUSSION *by LeRoy C. Abbott, M. D., San Francisco; Ralph Soto-Hall, M. D., San Francisco; Maynard C. Harding, San Diego.*

THE orthopedic treatment of chronic arthritis is directed in the earlier stages of the disease to the conservation of joint and muscle function and the prevention of deformity and in the later stages to the correction of deformity and reconstructive joint surgery. The conservation of joint and muscle function is accomplished by physical methods and demands both physiological rest and exercise. The principles of rest and exercise are more adequately applied when the constantly changing pathology occurring in both the major types of arthritis—atrophic arthritis, the so-called rheumatoid or proliferative type, and hypertrophic arthritis, the so-called degenerative or osteoarthritic type—is understood.

PATHOLOGY

Atrophic arthritis begins in the soft tissues and fibrous structures surrounding the joint with the gradual spreading of inflamed tissue into the joint capsule and synovia in the presence of early and extreme bone atrophy, which constitutes a valuable early diagnostic sign. Later, marginal granulation tissue forms and creeps centrally over the articular cartilage as a vascular ring of pannus, which may entirely replace the articular cartilage. This process occurring over both of the articular surfaces of the joint, the opposing layers of granulation tissue may adhere to each other, becoming vascularized in the form of firmly organized connective tissue. Also, the connective tissue of the marrow, proliferating, contributes to form a vascular granulation tissue which extends up to and may even ulcerate through the overlying articular cartilage. Therefore, the cartilage of an atrophic joint may be destroyed either by the extension of granulation tissue over its surface or by the extension of granulation tissue from the marrow.

LOCAL JOINT THERAPY AND SUPPORT

With this pathology clearly in mind, the importance of both physiological rest and exercise combined with the judicious employment of the physical methods in order to maintain joint motion and nutrition and muscular activity will be recognized. Rest to the atrophic joint is best obtained from the application of adequate braces and removable splints, having clearly in mind that the atrophic joint does not demand complete and continuous rest, but exercise as well. Absolute fixation of an atrophic joint by a plaster cast is contraindicated if we are endeavoring to preserve motion and prevent bony ankylosis. In general, braces are not applied sufficiently early in the management of atrophic arthritis. The initial onset of deformity is early and its progress rapid,

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