that, like the subject of instruction in sex hygiene, the teaching of proportional values of mental stimulation and excitement for growing children has been avoided, except in rare individual cases, and (being rather complex in its nature) has been generally left to parents and circumstances. While free to admit that the subject is complex, it would appear to be intimately related, so far as preventive measures are concerned, to greater cultivation of self-discipline and a contented attitude towards greater simplicity in life. It sometimes seems as if the civilization of our present day had overreached itself. We must today, therefore, defend children against the alarming tendency (in this country at least) of hypertension and its eventual deleterious organic results, by urging a cultivation of a less strenuous, more simplified, more natural mental and physical existence for our children. Can not much be accomplished in urging the profession, special and general, to take up the causes in a systematic way and endeavor to show at the same time the methods and possibilities of elimination on the ground that such excess is proving a real menace to present and future health in childhood and youth?

THE SIGNIFICANCE OF MENINGEAL SYMPTOMS.

BY A. C. EASTMAN, M.D., SPRINGFIELD, MASS.

THE unstable condition of a child's nervous system is the cause of many mistakes in diagnosis, and should make us very careful lest we place too much or too little emphasis upon the symptoms arising from a disturbed nervous system. Careful consideration of all data obtained by physical and laboratory examinations is necessary before we can safely pass judgment upon the significance of such symptoms. It is by no means always easy, even with such data at hand, to definitely distinguish between a meningism and meningitis or between the various forms of meningitis.

Most of the symptoms supposed to be characteristic of meningitis have also been found present in a number of other diseases. Convulsions, headache, stupor, vomiting, and high temperature may be present at the onset of many acute infections. The more definite signs of meningitis, such as rigidity of the neck, change in reflexes, Kernig's sign, Babinski's reflex, Brudzinski's neck sign, ankle clonus, and opisthotonos, have each and all, at one time or another, been found present in other acute infections besides meningitis. Especially in pneumonia, typhoid, influenza, and the exanthems,

are we liable to find some of these signs present. Pneumonia, influenza and typhoid are not only peculiarly subject to meningismus in their early stages, but are also liable to develop a secondary meningitis later in the course of the disease. The poorly nourished, marasmic infant has frequently been diagnosed as a case of meningitis because of the pseudo-opisthotonos which is oftentimes present. The more we recognize the possibility of such signs occurring in various infectious diseases, the more difficult becomes the problem of a proper interpretation of their symptoms.

Whenever meningeal symptoms are present at the onset or during the early stages of a disease which we can positively diagnose as some infection other than a meningitis, we may be justified in declaring such symptoms due to a meningism, but whenever there is doubt as to the diagnosis, or the symptoms develop later in the disease, we must seriously consider meningitis.

From our present knowledge of immunity we know that most of the immunity of adult life is acquired during childhood, which means that the infant and young child is more susceptible to many of the acute infections, as well as being more frequently exposed to them. The various portals of entry are not yet well guarded, and the nasopharyngeal system and the blood stream give bacteria and their toxins easy access to the various parts of the body, including the central nervous system. Thus we find meningeal symptoms much more common in early childhood than in any later period of life.

It is beginning to be considered probable that the convulsions at the onset of an infection, as well as the early meningeal symptoms, are caused in many cases by actual changes in the quantity and quality of the cerebrospinal fluid. It is possible that these changes are brought about by the sudden increase in blood supply to the chorioid plexus at the onset of fever, causing a rapid increase in quantity of cerebrospinal fluid with its resulting increased pressure. The symptoms developing from this increased pressure will begin to subside as soon as the circulation has a chance to become regulated to the presence of high temperature, provided no bacterial invasion of the meningeal membranes has taken place.

In a series of cases recently published by Herick and Dannenburg,¹ it was found that of a large number of infections, not including the

various forms of meningitis, but having meningeal symptoms, 30% showed increased pressure of the cerebrospinal fluid, together with increased cell count and globulin content. To further quote their conclusions,—“These diseases are lobar and bronchial pneumonia, influenza, the exanthems, scarlet fever, measles, variola; herpes zoster, parotitis, typhoid fever, septic arthritis, pleurisy, migraine, reaction to typhoid inoculation, and others. The cerebrospinal fluid shows variation from normal in about one-third of the cases studied. Most, but by no means all, of the patients with a subarachnoid reaction have clinical meningismus. On the other hand, many examples of meningismus are without pronounced changes in the cerebrospinal fluid. The greatest caution should be used in making a diagnosis of meningitis or poliomyelitis from fever, meningismus, and the changes in the cerebrospinal fluid mentioned. Cases with less than one hundred cells should be viewed with skepticism, unless clinical, epidemiological, or other laboratory evidence is decisive.”

Levinson* found that in one hundred cases of meningismus all showed an increase of fluid with increased pressure, but only fifteen showed an increase of cells and globulin content. None of these cases showed any physico-chemical changes. Such conclusions as these, although they serve to explain many of the phenomena grouped under meningismus but associated with acute infections, at the same time make it all the more difficult for us to make a diagnosis from the cytological findings.

Although the meninges during childhood are more susceptible to infection than in adult life, yet even in childhood the protective power of the meningeal membranes against bacterial invasion is greater than that of other membranes of the body, and hence most infections of the meninges are secondary to infections somewhere else. Certain organisms have a special tendency to attack the meninges early. These organisms are the meningococcus, the poliomyelitis virus, and oftentimes the pneumococcus. In the first two especially, the invasion of other tissues is temporary and chiefly a portal of entry for the organisms which are then rapidly carried to the meninges. The streptococci and staphylococci practically always invade the meninges by direct extension from some local focus as the middle ear, mastoid, or cerebral abscess. The tubercle bacillus

invariably is carried to the meninges from some primary focus in another part of the body, although the first symptom of tubercular infection may be the onset of a meningitis, appearing clinically as a primary disease.

Whenever we are in doubt as to our diagnosis, lumbar puncture is the only means of solving the problem. Since lumbar puncture was first introduced, we have been gradually led to believe that the cytological examination easily decided the diagnosis, that a meningism might, and often did, show an increased pressure, but the cell count was always low and there was no globulin reaction. The more the fluid has been studied, however, the more we find discrepancies in these findings. The fluid may show a comparatively high cell count with globulin reaction and still the child improve clinically, the meningeal symptoms disappear, and the original infection run its normal course. Or the fluid may show no reaction and have a low cell count, but the symptoms persist, and later a typical meningitis develop with its characteristic cerebrospinal fluid.

Also, in cases of true meningitis, we find wide variations from our former knowledge of the findings. Epidemic cerebrospinal meningitis may show a comparatively clear fluid and make us think we have a tubercular form of meningitis until we find the organisms present in the cells. Also the tubercular fluid may not differ at all from that of poliomyelitis in its fundamental properties, the clinical course of the disease being the only way to distinguish the two unless the tubercle bacillus has been discovered in the fluid. Fortunately, the types of meningitis which are apt to develop early and confuse our diagnosis with some other common infection are the types which usually show positive bacteriological findings in the spinal fluid, and a lumbar puncture becomes of definite assistance in these cases, even when it proves to be a meningism with high cell count and globulin reaction but negative bacteriological findings.

Because of the difficulty in finding the tubercle bacillus, our chief trouble centers around the diagnosis of tubercular meningitis as opposed to poliomyelitis, encephalitis, or a meningism secondary to some acute infection with a mild or moderate onset. In these cases, repeated punctures with a careful searching for the tubercle bacillus, together with the clinical progress of the infection, will sooner or later

clear up the diagnosis. Such repeated lumbar punctures can safely be done once a day until we have secured sufficient evidence to make the diagnosis. It is surprising, oftentimes, to see the rapidity with which the findings in the cerebrospinal fluid will change and clear up what was apparently an obscure condition. As long as there is any doubt as to the diagnosis, we must never be satisfied with the results of a single lumbar puncture. A typical case to illustrate this point is the following:

E. M., 4 years, admitted to Springfield Hospital April 15, 1920. F. H.—Paternal grandmother died from tuberculosis. History otherwise negative. P. H.—Always well except pertussis three years ago. P. I.—For past month complained a great deal of "pain in stomach" after eating. Pain relieved after bowel movement. No nausea or vomiting. During past week has lost weight; has been very peevish and wished to be held. A question of some discharge from left ear. P. E.—Well developed and nourished. Rather drowsy. Cries if handled or moved. Tender over left mastoid. Slight rigidity of neck. No definite change in reflexes. Throat, chest, and abdomen negative. Seen by aurist and left ear incised. Temperature, 102; pulse, 140. For two days her condition remained about the same and the symptoms pointed toward a meningism secondary to the ear. Lumbar puncture gave fluid with no increased pressure; cell count, 40; globulin, negative; no sediment, and fluid sterile. Leucocyte count upon admission, 10,000. Two days later, 17,000. A second lumbar puncture the next day showed no change in fluid except a differential count gave 85% polymorphonuclears. Five days after entrance her condition became worse and she then showed a typical clinical picture of tubercular meningitis with characteristic reflexes. Two incisions of left ear drum had produced no results although there appeared to be evidence of some trouble there. A third lumbar puncture gave increased quantity with increased pressure. Fluid clear. Cell count, 130; globulin, positive. Differential count showed lymphocytosis. Typical sediment of tubercular meningitis. During the next two days the child grew rapidly worse and died. Autopsy showed many small millary tubercles on surface of brain and at base, a slight exudate at base. Left internal ear showed chronic necrosis.

The cases of tubercular meningitis with sud-

den, severe onset usually develop much more rapidly and give other signs as paralyses, ocular symptoms, neck sign, stupor, etc., which altogether help to differentiate the condition from a meningism, but which might still be confused with poliomyelitis or encephalitis. The further clinical progress of the case is the only means of differentiation between these diseases, as the cerebrospinal fluid in such cases of tubercular meningitis may very often show no characteristic properties before death takes place.

Gingold³ has described a reflex strabismus as an early diagnostic sign of basilar meningitis. His description of the sign is as follows: "By flexing the head on the chest, either a bilateral or unilateral internal strabismus develops, which will last as long as the head is kept in flexed position, and disappears as soon as the head is relaxed. In many cases the strabismus is accompanied by a retraction of the upper eyelids. In some cases I have also noticed a contraction of the pupils. This reflex strabismus was present in the early stage of almost every case that came under my observation in the past seven years. In the late or paralytic stage, flexion of the head failed to produce a strabismus. The reflex strabismus, above described, proved, in my experience, to be such a reliable symptom that I have often made a diagnosis of basilar meningitis days before any other meningeal symptoms appeared."

The various chemical and physicochemical changes in the cerebrospinal fluid serve further to differentiate the different types of meningitis, but are chiefly possible to obtain only in the large laboratories. For practical purposes we must rely upon the presence of increased fluid, increased cell count, differential cell count, globulin reaction, presence of sediment, and bacteriological examination to make a diagnosis.

CONCLUSIONS.

Meningeal symptoms are frequently present in many of the infections of childhood and may represent either a meningism or a meningitis.

Unless definitely positive of some diagnosis besides meningitis, the only means of determining their significance is by lumbar puncture, which, in many cases, requires several repetitions before we can make a definite diagnosis.

Like other laboratory examinations, the findings in the cerebrospinal fluid must be consid-

ered in conjunction with the clinical progress of the disease.

In the final analysis we must depend upon the bacteriological examination to furnish a positive diagnosis.

REFERENCES.

- ¹ Herrick and Dannenburg: Observations on the Cerebrospinal Fluid of Acute Disease. Jour. A. M. A., Vol. lxxiii, p. 1321, November, 1919.
- ² Levinson: Qualitative and Quantitative Changes in the Cerebrospinal Fluid of Various Diseases and their Significance. Am. Jour. Dis. of Children, Vol. xviii, p. 568, December, 1919.
- ³ Gingold: An Early Diagnostic Sign in Basilar Meningitis. Arch. Ped., Vol. xxxvii, p. 19, January, 1920.

DISCUSSION.

DR. J. J. THOMAS, Boston: Dr. Eastman's paper is one which covers, I think, the points very fully and very completely. There is not much that I can add to it, I am sorry to say. Perhaps there may be one or two things that may help to bring out the idea a little more definitely. At least I may be able to add something in that respect to what he has said. We probably have to distinguish in these cases which show clinical signs of meningitis between different groups of cases: first, those of meningitis where we have an actual infection of the meninges by some form of bacterium. These are familiar to most of you, and the one which is most difficult to diagnose, as you all know, is the tuberculous form, because the tuberculous is the one in which it is most difficult to find the germ when examination is made of the cerebrospinal fluid. In addition in regard to the infectious forms, that is, the true meningitis, I may add just a word, and that is that very frequently on doing lumbar puncture you may miss the organisms on examination of the fluid. On the first time that the fluid is withdrawn, I have frequently seen the fluid perfectly clear and on the second or third drawing the organism has been found, and that is true not only of tuberculous meningitis, for which you may have to examine the fluid many times before the germ is found, but it is also true of the epidemic form due to the meningococcus. Probably this difficulty would be less frequent if we did ventricular puncture more often or punctures of the cisterna magna at the base of the skull. That strikes us all as a dangerous procedure, yet if carried out with precautions, is a practical one, where the needle is introduced between the base of the skull and the first cervical vertebra and the fluid withdrawn. In tuberculous meningitis the meninges at the base of the brain are the parts first involved, and it may be some time before the germs show in the fluid obtained from the lumbar part of the spinal cord. So that, as was rightly said, repeated lumbar puncture is very important, or I may say in addition to that, the puncture of the ventricles may lead to a diagnosis earlier, or show that we have to deal with a germ.

You are familiar with the fact that the character of the cells obtained in the spinal fluid varies. In the epidemic form we get an excess of polymorphonuclear leucocytes, and in the tuberculous form we find the lymphocytes predominating; but those are not absolute, and we find variations. Next we ought to speak of the cases that give meningeal signs which are due to infection of the brain. Among these are the early encephalitis cases which Dr. Eastman has spoken of, and I should add to that the cases of poliomyelitis in which there is a meningismus. In these cases we have some virus which is in the nervous tissues of the spinal cord and occasionally of the medulla, and in the epidemic forms in the brain itself. And then we have the ordinary encephalitis where the virus which causes the disease is found in the cerebrum, and the reaction is not due to the actual infection but is more in the nature of a meningismus. Do not overlook the cases of encephalitis of the epidemic form, which is rare, but more common than we thought heretofore. Here the cells may be increased just as they are in other forms of encephalitis, but we find no germs.

Now, as for true meningismus, or the serous meningitis of Quinke and other writers, I think we should limit the term to the reactions where the sign is due to toxic substances. I have seen it in various acute infections, and in malaria, where we found the plasmodium in the blood, and the child had a convulsion every second day. There is a reaction of the meninges so that various poisons will give you these irritative phenomena, and while we have some reaction shown in the spinal fluid, it is not so intense as in true inflammation, and the germs are absent. I think among all signs the clinical symptoms must not be neglected and among these the most valuable is the variability of the symptoms.

DR. A. C. EASTMAN: I have very little more to say, except perhaps to emphasize the importance of repeated punctures, especially in connection with the early meningeal symptoms, that is the symptoms coming early in the disease in the line of meningismus. The tendency is always to wait until you feel sure that you have a meningitis and then do a lumbar puncture; but I think if we are at all uncertain we ought to go ahead and do a lumbar puncture early, and if we don't get assistance in the first puncture we can repeat. Also in these cases of meningismus we do good by relieving symptoms. In cases of pneumonia, typhoid and similar diseases, frequently a lumbar puncture by withdrawing a certain amount of fluid, will reduce the symptoms and make the child more comfortable.