point. We were further impressed with the potentialities of this attack by observing that during the "sulfa" treatment of gonorrhœa we did not see any chancroid, which has now again reappeared with the advent of penicillin therapy; hence we are attempting to treat and cure early incubating syphilis by treating every case of gonorrhœa with a dose greater than that which Alexander and Schoh have shown to be effective.

Our own use of this amount for the treatment of gonorrhœa has shown it to be possible as a single injection and comparatively painless. Even with the shortest treatment schedule on an ambulatory basis it is but human to expect lapses and disappearances, so it is felt that the first dose, even if it is the only one given the patient, will be sufficient to go a long way towards effecting a cure. And finally, maybe Ehrlich's dream of "therapia sterilisans magna" is close at hand.

REFERENCES

1. HYMAN, H. T., CHARGIN, L. AND LEIFER, W.: *Am. J. M. Sc.*, **197**: 480, 1939.
2. MAHONEY, J. F., ARNOLD, D. C. AND HARRIS, A.: *Ven. Dis. Inform.*, **24**: 355, 1943.
3. MOORE, J. E.: Penicillin in Syphilis, Charles C. Thomas, 1st ed., p. 117, 1946.
4. STOKES, J. H.: Modern Clinical Syphilology, W. B. Saunders Company, 3rd ed., p. 1256, 1944.
5. Eighth Annual Meeting, Chicago, December 3, 1949.
6. REIN, C. R., THOMAS, E. W. AND KITCHEN, D. K.: Paper read at American Academy of Dermotology and Syphilology, Chicago, December 7, 1949.
7. NELSON, R. A. JR.: Paper read at American Academy of Dermatology and Syphilology, Chicago, December 7, 1949.
8. ALEXANDER, L. J. AND SCHOH, A. G.: Paper read at American Academy of Dermatology and Syphilology, Chicago, December 7, 1949.
9. DATTNER, B.: Paper read at American Academy of Dermatology and Syphilology, Chicago, December 8, 1948. Discussion by Arthur C. Curtiss, Paul O'Leary, Evans Thomas and Herman Bierman.

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MYOCARDIAL INFARCTION FOLLOWING THE ADMINISTRATION OF TETANUS ANTITOXIN.—A case of serum sickness complicated by acute myocardial infarction is presented and the literature reviewed. The patient was a 32-year old male without evidence of any of the stigmata which might be associated with coronary artery disease. The serum sickness followed the prophylactic administration of tetanus antitoxin and the complicating infarction was suggested as being due to coronary arteritis the result of the allergic disease. It is suggested that many injected materials, including penicillin, streptomycin, insulin and liver extract, may be capable of producing myocardial infarction in sensitive patients. —McManus, J. F. and Lawlor, J. J.: *New England J. Med.*, **242**: 17, 1950.

AMAUROSIS IN WHOOPING COUGH*

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IT is a well known fact that the acute infectious diseases in the ordinary course of events are readily diagnosed. However, unusual complications such as those involving the central nervous system may obscure the common symptoms to a point where the disease is not readily recognizable. Such was the event in the cases of amaurosis in whooping cough that are here presented.

Blindness in whooping cough is an exceedingly rare complication. Walsh,[1] in discussing the subject, refers to the papers of Lazarus and Levine[2] who reviewed 20 cases from 1870 to 1930 and presented an additional case of their own in 1934. These authors concluded: (1) Amaurosis in pertussis is a rare manifestation and is seldom permanent. (2) The blindness occurs between the first week and the fourth month of the disease and it may last from a few minutes to nine months. (3) The incidence is widespread and the condition rarely occurs after adolescence. (4) There are no pathognomonic eye findings. (5) At least half of the cases of amaurosis show cerebral manifestations. (6) In the majority of the cases reviewed, it would appear that the ocular changes are due to cerebral œdema or to an acute toxic hæmorrhagic encephalitis.

A highly significant fact in these two cases is the similarity of events. The girls were sisters; they contracted the disease about the same time, developed convulsions the same day, and both became blind during the period of convulsions.

CASE 1

M.E., a white girl, aged 3 years, was admitted to the Port Arthur General Hospital on January 14, 1949. The history given by the parents revealed no past illnesses, nor any hereditary or constitutional diseases. A younger brother, a breast-fed infant was not afflicted.

One week prior to admission to hospital, the child manifested what the parents described as a head and chest cold. She became progressively worse and began to have paroxysmal coughs. On the seventh day she developed convulsions and was taken to hospital.

On admission the child was unconscious, frothing at the mouth, listless, and could not be roused. Her temperature was 105° F. The head appeared normal. External examination of the eyes revealed no significant findings. The pupils were dilated and did not react to light or other stimuli. It was difficult during this period to ascertain the nature of her sight. The media, disc,

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vessels, macula and peripheral fundus appeared normal. With exception of the frothing, examination of the nose, mouth and pharynx showed no abnormal signs. Examination of the lungs revealed numerous moist and crepitant râles. Cerebral irritability was marked and the reflexes were hyperactive. There was no evidence of paralysis. Several hours following admission to hospital, the child developed her second and last attack of convulsions which persisted for about one hour. Muscular twitchings at this time became numerous and general. Amaurosis was one of the most striking of complications. At intervals she suffered from severe coughing which was whooping in nature. On the following day her temperature rose to 106° F.; with treatment this gradually receded to normal. It was not until the third day that the child began to regain consciousness and to appear orientated. On the third day in hospital, she was noted to grope about with her hands. She had a fixed stare. At this time both of the pupils were normal in size. They reacted sluggishly to light stimulation. In a darkened room her attention could not be attracted by a strong beam of light but she would turn her head in the direction of noise. The paroxysms of cough, cyanosis and vomiting continued for a period of about one and a half months. The temperature became elevated on three occasions during the interval, reaching a height of 103° F. and lasting for 2 to 3 days. A left internal squint appeared during the second week. It was not until one month after admission that it was first noted the child had some light perception. The recovery of vision commenced in the temporal field of the left and the nasal field of the right eye. The fields increased gradually in a clockwise direction. By the end of the second month the child was able to see and pick up objects. As the vision improved so did the squint. By the third month, the child was considered sufficiently recovered physically to be discharged from hospital although it was relatively certain her vision had not returned to normal.

At the fifth month it was felt visual recovery was complete. Eye examination revealed no residual damage such as muscle paresis, optic atrophy, etc. Physically and mentally the recovery seemed to be satisfactory.

Laboratory data.—A chest x-ray revealed the presence of a generalized bronchopneumonia. The early blood studies showed: hæmoglobin 80%; white blood cells 16,750; polymorphonuclear leucocytes 32%; lymphocytes 68%. Within one and a half months the relative values changed and the blood count became normal. Other tests such as the blood Wassermann, heterophile antibody tests, spinal puncture tests, blood culture and sputum were negative. Early in the disease a trace of albumen was to be noted in the urine; this rapidly cleared.

Chloroform was administered to control convulsions. Sodium luminal, phenobarbital and morphine were used for sedation. Penicillin and glucose in saline were administered intravenously. There was no specific treatment for the eyes.

CASE 2

V.E., aged 9 years, was admitted to the Port Arthur General Hospital on the same day as her younger sister. One week previously she had developed what was termed a cold characterized by severe coughing spells.

On admission she was unconscious, breathing deeply and in a state of convulsions. This child was more irritable and irrational than her younger sister; she was very sensitive to handling, indicating marked cerebral irritability. Examination of the head and neck was not significant with exception of pupillary reactions. Both pupils were dilated and did not respond to ordinary stimulatory methods. The media, disc, vessels, macula and peripheral fundus appeared normal. Twitchings of the various muscles were numerous and general. Moist and crepitant râles were generally present in both lungs. The deep and superficial reflexes were hyperactive and the Babinski sign was positive.

She remained stuporous for about three days. At intervals she would emit weird screams and babbling sounds. Compared to her younger sister she appeared to be much more seriously afflicted with the disease. There seemed to be a reversion to what might be described as an "animal-like" state. With treatment the recovery was however much more rapid. For four days the temperature varied between 99 and 101° F. On the fifth day it came down to normal and did not rise again. In so far as the vision was concerned, there was complete amaurosis for one week. At the end of this period she seemed to manifest some light perception. The nature of field recovery was indefinite but it appeared as if her central vision was most affected.

After the first week in hospital her general health improved rapidly. By the end of one month's time her visual acuity had improved markedly in both eyes but recovery was not complete. Examination about three months later found the visual acuity to be normal. Five months later re-examination showed the child to be in a normal state of health and having no residual visual damage.

Laboratory data.—The chest x-ray showed the presence of a generalized bronchopneumonia. The early blood studies showed: hæmoglobin 100%; white blood cells 20,650; polymorphonuclears 56%; lymphocytes 44%. Blood cultures, heterophile antibody test, Widal and blood Wassermann were negative. Throat cultures proved to be negative. The urine at first showed a slight trace of albumen, and a few fine granular casts, occasional pus cell and the odd red blood cell.

On performing the spinal puncture the pressure was found to have risen to 325 mm. of water. There were present 10 red blood cells per c.mm. In one month's time the pressure became normal. During the early part of the disease the colloidal curve was of a meningitic nature. The globulin portion showed a slight increase on the first test.

Treatment.—This was followed as outlined in Case 1. Chloroform controlled the convulsions and morphine seemed to be the only effective sedative.

COMMENT

The case histories show that the various signs, symptoms and tests point toward brain involvement. The pupils were dilated and non-reactive during the early stage of the disease when cerebral irritation was most marked. Once the convulsions were controlled the pupils became normal in size and reaction. Except for amaurosis the subsequent eye findings were negative. It was concluded that the optic nerves, chiasm and tracts were not affected. The view is taken that the bilateral amaurosis was due to an acute toxic encephalitis with cerebral œdema, complication of whooping cough. In autopsies performed on pertussis patients, frequent pathological findings have been in the nature of œdema and petechial hæmorrhages.

SUMMARY

1. Two cases of amaurosis in whooping cough are reported.

2. Comment is made on the assumption that blindness was due to an acute toxic encephalitis.

REFERENCES

1. WALSH, F. B.: Clinical Neuro-ophthalmology, The Williams and Wilkins Co., Baltimore, 1st ed., p. 568, 1947.
2. LAZARUS, S. D. AND LEVINE, G.: *Am. J. Dis. Child.*, 47: 1310, 1934.

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