TYPHOIDAL HEMIPLEGIA WITH REPORT OF THREE CLINICAL CASES AND ONE WITH NECROPSY.

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HEMIPLEGIA following or occurring during typhoid fever is a comparatively rare complication, and any cases of this character are well worth reporting. It has been mentioned as early as 1860 by Gubler,¹ but since then, to my knowledge, there have been few cases published.

Among the more recent literature on this subject the paper of Smithies² takes up and classifies the symptoms, prevalence, age, time of occurrence, etc., in considerable detail.

In compiling the cases already published, with those described in this paper, several points are of interest.

The male sex was more often affected (70 per cent.) than the female (30 per cent.) in the 40 cases in which the sex was defined.

Where the side involved was tabulated, left hemiplegia occurred in 19 (44 per cent.) and right hemiplegia in 24 (56 per cent.).

In 28 cases of right side involvement aphasia was noticed in 23 (82 per cent.), while it is mentioned in only 3 (18 per cent.) of the 17 left hemiplegics.

The complication occurred before the age of ten years in 9 instances (24 per cent.), before twenty in 10 cases (26 per cent.), and in 19 (50 per cent.) before the age of thirty years.

Of those cases in which the time of onset was given, 10 (24 per cent.) occurred in the first and second week, 8 (20 per cent.) during the third week, 4 (10 per cent.) during the fourth, 15 (40 per cent.) during convalescence, and 2 (5 per cent.) so late after the attack as to render the dependence of the condition upon typhoid rather doubtful.

Athetosis was reported by Curschmann,³ Osler,⁴ and Barrett,⁵ and is also present in my first case.

Visual defects have not occurred, with the exception of a left-sided amaurosis and nerve atrophy in an interesting case described by Benedikt.⁶ Here there was also stuttering with the left-sided hemiplegia. Stuttering was also one of the symptoms of my first case. In Pigott's⁷ case the pupils were dilated, the right larger than the left and less reactive to light than the left,

¹ Arch. Gén. d. Méd., 1860.

² Jour. Amer. Med. Assoc., August 3, 1907, vol. xlix, No. 5. In this paper a very complete list of literature up to 1907 is given.

³ Nothnagel's Handbuch, Band iii, I Teil.

⁴ Johns Hopkins Hosp. Rep., 1899 to 1900.

⁵ Report of Michigan State Asylum for Insane.

⁶ Elektrotherapie, 1868.

⁷ Maryland Med. Jour., 1909, iii.

also deviation of eyes to left. This was a left hemiplegia. A case of left hemiplegia reported by Parisot,⁸ which ran a course of a few days, showed inequality of the pupils and strabismus. In Case IV there was deviation of the eyes toward the right with left-sided hemiplegia.

Sensory changes in most instances were absent or so slight as to be doubtful in value. Such changes were present in the case of Raschofsky's⁹ and Case I of this paper.

An interesting case, more of a meningitic type, is described by Laure¹⁰ in which, in addition to the typical signs of a meningitis, there occurred an aphasia with loss of ability to write, but no paralysis.

The hemiplegia occurring in typhoid does not differ materially from one following any other cause, and should therefore be readily diagnosed. In some cases the onset may be acute, with convulsions or delirium or a rapidly developing coma or general restlessness with increase in the temperature, while in others it may be very gradual, extending over a period of hours or days or even weeks (Nothnagel), and in some instances reported there was no positiveness concerning the exact time of the occurrence of the paralysis.

This variety of onset is dependent to some extent upon the reactive or resistant condition of the patients' nervous system, but much more upon the character of the pathological lesion causing the condition. Thus, in the early cases where there is perhaps more likely to be a hemorrhage or an embolus, the onset is acute and there may be convulsions and coma. When, however, the symptoms are the result of a toxemia, there would more probably be a delirious condition of some duration, with temperature elevation in cases of an encephalitic character. In the convalescent stage there would be, more likely, a gradual onset, such as might occur in thrombosis, this lesion being in all probability the one which most frequently occurs during this stage of the disease.

A higher percentage of cases has been reported as occurring during the stage of convalescence, and this is due in all probability to the generally weakened condition of the patient at this period, the sluggishness of the circulation, and the condition of the vessels and the blood, tending to aid in the formation of thrombi in the cerebral vessels just as they do in the better known thrombotic processes elsewhere in the body.

The prevalence of this complication in the male sex is dependent upon a greater percentage of typhoid occurring in males, and also upon the fact that the male, through excessive venery and abuse of alcohol and tobacco, is more subject to such vascular changes as predispose to a thrombotic process.

⁸ Rev. méd. de l'Est. Nancy, 1909, xli.

⁹ Wien. med. Woch. 1911, No. 39

¹⁰ Rev. Neur., Paris, 1908, xvi.

The question of atetosis and choreic movements present in Case I, also reported by Curschmann, Osler, and Barrett, is of considerable interest as it is caused by involvement of some vessel other than the one so frequently affected. It might be due to an affection of the meningeal vessels, causing irritation, or of the vessel or vessels supplying the lenticular and thalamic regions.

The pathological condition most frequently occurring in these hemiplegics is in all probability a thrombosis, this being due in great part to the causes mentioned earlier in my paper. Thrombi were found in the Sylvian fissure branch of the middle cerebral artery by Dankin,¹¹ Hawkins,¹² Osler,¹³ Barrett,¹⁴ and in my fourth case. Embolic softening would in many cases be difficult to distinguish from thrombosis—cases of this nature were published by Curschmann¹⁵ and Hruska.¹⁶

Parisot¹⁷ reported a case in which the internal carotid was thrombosed at its entrance to the skull, and as a result the central part of the right hemisphere, the external capsule, and basal ganglion were totally destroyed, leaving only a shell of the cortex intact. Curschmann's case had a diffuse hemorrhage in the lenticular area.

Some of the hemiplegias, particularly the more transient and rapidly healing ones, may be caused by a toxemia affecting the cerebral cells, and this interpretation would more readily explain the great percentage of cases having a complete or almost complete recovery. Barié and Lian¹⁸ referred to the probability of such an origin in their case.

Encephalitis may also be the basis of some of the cases, as in the one reported by Raschofsky.¹⁹ Here the increase of the temperature at the time of the onset of the symptoms with restlessness and the recedence of the fever with the return to normal, led the author to consider an encephalitis as the cause.

The prognosis is dependent to a great degree upon the character of the lesion present. In those cases of toxemia or encephalitic origin the recovery may be almost complete. Where a hemorrhage or thrombosis is the basis of the condition, the change would be much slower and less complete. As a rule, the leg regains its power and function much earlier than the arm and hand. Aphasia, if present, may improve, but very seldom clears up entirely (Smithies). Treatment is largely symptomatic with the necessity of avoiding the use of any drugs or measures that might lead to hemorrhage. The late stages require the usual massage with general tonic treatments.

¹¹ *Lancet*, April 23, 1892.

¹² *Loc. cit.*

¹³ *Loc. cit.*

¹⁴ *Loc. cit.*

¹⁵ *Loc. cit.*

¹⁶ *Clin. Soc. Trans.*, 1889.

¹⁷ *Loc. cit.*

¹⁸ *Präg. med. Woch.*, 1901, Nos. 41 and 42.

¹⁹ *Bull. et. Mem. de la Soc. d. Hôp. de Paris*, 1907.

Papers have been written on cerebral and meningeal complications of typhoid by Du Castel,²⁰ Cabanes,²¹ Barthelemy et Durbin,²² Starkiewicz,²³ Diller,²⁴ Antony and Dofter,²⁵ and Laignell-Levas-tine.²⁶ Cases have been reported by Lesieur,²⁷ and Tondi,²⁸ also an atypical case of post-typhoid thrombosis by Pasteur.²⁹ I regret that I have been unable to obtain the records of any of these.

I am greatly indebted to Dr. William G. Spiller for the privilege of reporting the following cases, 2 of which were examined by me at the University and Philadelphia Hospitals, and a microscopical examination made in the third.

CASE I.—E. K., aged twenty-two years. Admitted to the Philadelphia Hospital December 2, 1908.

Family history negative as regards possible affections of the nervous system.

Patient was well up to the age of six, when he developed typhoid fever. During the attack of typhoid fever he was confined to bed for three weeks. He remembers that he was wildly delirious during this illness, and wanted to jump out of the window. He also claims to have been unconscious during part of his illness, but does not know the duration of the unconsciousness. About two months after onset, and a week or so after beginning to improve, upon attempting to get up one morning he found that he could not use his left leg and arm, but does not know whether or not he could move his arm and leg while in bed. He was not able to walk for some time. His speech was affected so that people said he talked "tongue-tied," and he had trouble in swallowing. There were at no time any other symptoms of head involvement such as headache, vomiting, etc.

The athetoid movements of toes and fingers were present as long as he can remember, but he does not think they anteceded the attack. His left foot felt heavier and colder than the right, and he dragged it in walking.

Examination upon admission. Left arm was flexed and adducted, and was continuously and rhythmically flexed and extended. Fingers were flexed and athetoid movements were constant. The biceps was markedly contracted and prominent, and various muscles of the forearm could be seen contracting rhythmically. The left arm with a great deal of exertion could be raised to the level of the shoulder. The forearm could be flexed and extended slightly, and with very little strength. Pronation and supination were not possible. The handgrasp was very weak, as were the

²⁰ *Gaz. des Hôp. de Paris*, 1910, lxxiii.

²¹ *Gaz. med. de Nantes*, 1906, xxiv.

²² *Jour. Amer. Med. Assoc.*, May 27, 1905.

²³ *Arch. d. méd. et pharm. Paris*, 1905, xlv.

²⁴ *Bull. et mem. des Hôp. de Paris*, 1907, xiv, No. 30.

²⁵ *Bull. de la Soc. méd. des Hôp. de Lyons*, 1907, vi.

²⁶ *Arch. Middlesex Hospital, London*, 1909, xvi.

²⁷ *Bull. med. del. Algerici*, 1908, xix.

²⁸ *Gaz. lek. Warszawa*, 1906, xxiv.

²⁹ *Napoli*, 1909, xxiv.

individual finger movements. The entire upper extremity was very spastic and resistant to passive movements; no atrophy. The left lower extremity showed retained power of flexing the leg on the abdomen, but this was greatly weaker than on the right. The leg could be flexed and extended at the knee, but no movement was possible with the toes, nor could the foot be elevated. The left foot was held in a partially flexed and adducted position, and continuous athetoid movements of the toes were present. Reflexes were markedly increased with ankle clonus and a Babinski strongly positive. Spasticity with resistance to passive movement was excessive. The leg was dragged in a spastic manner, but not swung in a circle, as it so often is in hemiplegics. Hypalgesia and hypesthesia were found over the entire left side, but no change in the temperature and position senses. Some slight asteriognosis was present in the left hand. There was no involvement of rectum and bladder.

Examination March, 1910. There remains distinct foot drop and atrophy of tibialis anticus and the peroneal group. The calf muscles are contracted. The thigh muscles are still somewhat weaker than the right. At present the athetoid movements occur in the fingers and in the arm when the patient attempts to resist passive movements. There is some slight impairment of the lower facial movements.

CASE II.—W. P. (No. 4218). Aged sixteen years. Male. Admitted to the University of Pennsylvania Dispensary December 11, 1905.

The patient was perfectly well up to four years ago, at which time he had a severe attack of typhoid. During this illness he had a left-sided hemiplegia, this having occurred while he was still in bed and running a temperature. The leg and arm were both affected; he could not move his fingers properly, nor the left side of the face. The patient said he could not talk for two days, although this might have been due solely to the involvement of the tongue and face. The eyes and ears seemed to be all right.

Examination on admission showed the left arm somewhat contracted and atrophied, with the reflexes increased, and the hands cyanotic and cold. No sensory changes were present. The power in the shoulder muscles was fair, but impaired. The patient had a distinctly spastic hemiplegic gait. All reflexes were increased with positive Babinski and some atrophy of the entire leg. There were no sensory changes.

CASE III.—Mrs. L. G., aged thirty-two years, white. Admitted to the University Dispensary September, 1910.

The patient always had good health until four years ago (1906), when she had typhoid fever, and was in bed twenty-one weeks. During her illness her right arm and right leg would frequently drop over the side of the bed and would have to be lifted back.

They were manipulated by the nurse, and when handled were always stiff and sore. When the patient got out of bed she experienced no especial difficulty in walking or standing, although she had to use a cane. The arm and hand were used only with great difficulty, and their original strength has never returned.

The right upper limb has never recovered in strength or size. She uses it awkwardly, and is forced to make more fine movements with the left hand. When she walks the right foot is dragged along the ground.

Examination. At rest the right side of the face shows some drooping in the lower half. Actively this is less marked, but still present. She winks the right eye less than left; wrinkles brow well. The tongue deviates to the right on protrusion. Ocular movements are good; reflexes normal. The upper extremities show marked weakness in all movements of the right arm, with all reflexes considerably increased. The entire limb is smaller than the left. There are fibrillary twitches, but no sensory anomalies. The left arm is normal. There is no vesical or rectal disorder.

Lower extremities. Gait is unilaterally spastic. The patient drags the right foot. Individual movements are fairly well performed with right leg, very well with left. The whole right lower limb is distinctly smaller than the left. There are no fibrillary twitches or sensory changes.

Reflexes. On the right the patellar tendon and Achilles tendon reflexes are greatly exaggerated. Ankle clonus is not demonstrable, probably on account of spastic contracture of Achilles tendon. The Babinski sign is present. On the left the reflexes are somewhat increased but not abnormal, and no Babinski.

Other than a slight thickening of speech during the first period of the attack, there was no disturbance of speech.

CASE IV.—B. C., male, colored, aged twenty-four years. Admitted to Dr. B. F. Stahl's ward in the Philadelphia General Hospital (and examined by Dr. Spiller), January 6, 1906. Died January 10, 1906.

During the course of typhoid fever the patient sat up in bed and fell out toward the right, striking first upon his buttocks. He was put back to bed and Dr. Spiller, upon examining him, found a left-sided hemiplegia with deviation of the eyes away from the paralyzed side. The fall was regarded as due to cerebral hemorrhage or a thrombosis of the middle cerebral artery. The patient died five days later. Necropsy showed congestion of liver, spleen, and lungs, and typhoid ulceration of the ileum; apparently typhoid in the third week.

At necropsy a thrombus was found in the artery of the right Sylvian fissure and microscopically the areas supplied by this artery were found to have undergone softening with the presence of a great many fatty bodies or cells and cellular debris.