Busey (S. C.)

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OF THE

SINUSES OF THE DURA MATER

IN

FATAL CASES OF DYSENTERY IN YOUNG CHILDREN.

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BY

SAMUEL C. BUSEY, M.D.,
Professor of the Theory and Practice of Medicine, Medical Department of the University of Georgetown; one of the Physicians to the Children's Hospital, Washington, D. C.

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It has been my misfortune to lose a number of cases of dysentery in young children; and, in every case, so far as I can recall the clinical histories, the fatal issue has taken place under precisely similar circumstances. In several cases during the acuteness of the attack, but more frequently after the characteristic symptoms had subsided and when convalescence seemed almost established, the child would be seized with convulsions, which, in occasional cases, recurred a second and a third time, and were followed by coma and death. In no instance has consciousness returned after the first convulsion.

These observations have led me to suspect that in dysentery, as in fatal cases of exhaustive diarrhea in young children, the convulsions which so frequently precede death found their cause in thromboses[1] of the sinuses of the dura mater, but in the absence of post-mortem examination, I could not verify it. The more commonly accepted opinion ascribes the final convulsions of exhaustive diarrheas to cerebral anemia, and it is undoubtedly true that both conditions not infrequently co-exist.

Marshall Hall was the first to recognize and differentiate spurious hydrocephalus from other intercranial diseases with which it had been previously confounded; yet he probably included in the clinical description of hydrocephaloid disease its congener, with which it is so closely allied in cause, symptoms, course, and result. In fact, both are the proximate effects of exhaustion and waste. Nothnagel, Gerhardt, and others assert that thromboses of the cerebral sinuses occurring

[1] Gerhardt found thrombi in the sinuses of the dura mater in seven autopsies of children who had died of profuse diarrhea, attended with cyanosis, coma, and convulsions.

in young children, when the walls of the venous channels are free from disease, "originate in conditions of the nature of marasmus." J. Lewis Smith, Steiner, and Vogel enumerate chronic gastro-intestinal diseases among the causes, and more recently Bouchut has definitely determined the fact that such formations may and frequently do occur in a variety of the chronic affections of young children, and occasionally at the termination of acute diseases. In such cases death is preceded by convulsions.

I have also observed, in a limited number of fatal cases of protracted diarrhea in very young children, edema of the lower extremities; in two instances associated with discoloration of the integument of the feet and legs, supervening several days previous to death. Whilst I had learned to regard these phenomena as indications of a fatal issue, I had not until the recent researches of Bouchut recognized the formation of thrombi in the pelvic veins as the cause of the venous stasis and the serous transudation into the subcutaneous cellular tissue.

Dysentery in young children is comparatively a rare form of intestinal disease in this locality, but three cases having been treated in the Children's Hospital since its foundation in 1871, consequently opportunities to make post-mortem examinations are not often secured. But recently, and only in a single case, have I obtained permission to examine the brain of a child dead of this disease.

This child was a patient in the Children's Hospital, under the care of my colleague Dr. W. W. Johnston, who has permitted me to use the notes of the post-portem. From the date of admission I made daily inquiries concerning the course and progress of the case, and when informed by the house physician that the dysenteric symptoms had all subsided, I replied that the child was very far from being out of danger; in fact, that the case had reached the stage when the danger of convulsions was imminent, and if such should occur, coma and death would follow. The next day he informed me that the patient had had three convulsions and was comatose. It died a few hours later.

Post-mortem by Dr. A. C. Adams, twenty-four hours after death. Body emaciated, eyes sunken, abdominal walls retracted; rigidity slight.

Brain: weight 2 lbs. 5¼ oz., anemic, effusion (estimated) into the arachnoid cavity 1 pint, slight in ventricles. Black clots in

all the sinuses, and a large white fibrinous thrombus at the junction of the right lateral with the petrosal sinuses.

Heart: effusion into pericardium; white fibrinous clots in superior vena cava, extending into right auricle and firmly attached to base of tricuspid valve. No blood in either ventricle; valves intact; weight 1¼ ozs.

Lungs: float in water; weight 7½ oz. ; left normal; right contained in middle lobe a cheesy mass as large as a hen's egg; this lobe was firmly attached to the pleura. Lungs anemic; no tubercular deposits. A cheesy bronchial gland as large as a pigeon's egg.

Abdomen: abdominal walls thin, destitute of fat; omentum contained but little fat; mesenteric glands slightly enlarged and congested. Intestines contain small quantities of feces; nothing abnormal in the small, in the large intestines patches of inflammation were found all along the track from the cecum to the anus. Liver anemic, buff-colored. Gall-bladder distended.

Large depots of pus at lower extremity of either kidney. Weight 1¼ oz.

The evidences of exhaustion, waste, and cachexia are obvious, and sufficient, perhaps, to account for the fatal result. But the inquiry cannot rest here, as the object is not so much to determine the cause of death as to ascertain the relation of thrombosis to the clinical phenomena; to study the means of prevention, and thereby avert the occurrence of a lesion which is incompatible with the long continuance of life.

The intracranial effusion, fulness of the venous channels, and anemia and lessened weight of the brain-mass are the usual post-mortem conditions concurrent with thromboses of the sinuses, but are also found, especially, in fatal cases of spurious hydrocephalus, independent of the presence of thrombi. They are not necessarily the result either of partial or complete occlusion of the sinuses by thrombi, but may be and frequently are the morbid effe ts of diseases characterized by great exhaustion, inspissation of the blood, and weak heart-action. Cases of such diseases as are complicated with intracranial effusion, shrinkage of brain-mass, and thickening of the blood are not necessarily fatal; they may be successfully met with timely and appropriate treatment. But those in which thrombosis may be either a superadded or a primary lesion, if not certainly fatal, acquire such gravity as to preclude any reasonable hope of recovery. The inquiries relate, then, especially to the diagnosis and prophylaxis.

Intracranial lesions are among the most difficult of diagnosis, and often the most cultivated and experienced diagnostician will be baffled in his efforts to determine, in a given case, the anemic or hyperemic condition of the brain. But in such diseases as favor the development of spurious hydrocephalus or marantic thrombosis of the sinuses, the previous clinical history would establish the condition of cerebral anemia beyond a doubt.

A long-continued exhaustive disease, characterized by emaciation, great prostration, a weakened heart, probably a subnormal temperature, dilatation of the pupils, eyes sunken in their sockets, half-closed lids, in very young children a depressed fontanelle, followed by apathy, somnolence, coma, and death, together with the post mortem evidences of anemia and shrinkage of brain-mass; thickening and stasis of blood in the venous sinuses, and sub-arachnoid effusion would complete the clinical history and post-mortem appearances of a case of hydrocephaloid disease. But even such an array of symptoms would not warrant an absolute diagnosis during life, to the exclusion of marantic thrombosis of the cerebral sinuses. If however, motor disturbances either of a paralytic or convulsive character should be added, the presence of the latter complication would be freed, in a measure, from ambiguity.[1] It is nevertheless true that, occasionally, symptoms of brain-irritation co-exist with all the usual objective and subjective phenomena of marantic thrombosis, whilst the post-mortem discloses only the conditions usually found in the hydrocephaloid disease. The two affections so often co-exist, and both are so alike in their precursory symptomatology, that it is not possible always to make a positive diagnosis of either to the exclusion of the other. Nor is the differentiation a matter of undisputed practical utility, except so far as it enables the practitioner, in the case of marantic thrombosis, to express a hopeless, and in the case of hydrocephaloid disease a less hopeless prognosis.

Marantic thrombosis may be the primary or a superadded lesion. That is, primary or secondary so far as regards the

[1] In thirty-eight observations of final convulsions in cachectic children, Bouchut found thrombosis of the sinuses in thirty-five cases, and in three, overfilling with blood and encephalitis.

consecutive development of the intracranial lesions which are usually present in fatal cases of the hydrocephaloid diseases and of cerebral thrombosis. General anemia, waste, thickening of the blood, a weakened heart, and brain-anemia and shrinkage, are the constant premonitory and causative conditions. Whether venous stasis from mechanical obstruction to the return current of the blood is ever a cause is a disputed question.

Thrombosis may be single or multiple, and the obstructed channels may be partially or completely occluded. Stasis and clotting of blood in the sinuses, and effusion (when secondary to the formation of these fibrinous masses, as undoubtedly they are in a majority of cases of thrombosis), vary in extent according to the locality of the masses and consequent interruption to the venous circulation. The amount of fluid in the cavity of the arachnoid (Nothnagel) and ventricles is not always increased.

The cure of thrombosis of the sinuses of the dura mater is not probably within the resources of medical science. The few reported successful cases were most likely instances of mistaken diagnosis.

It is only during the premonitory stage, when conditions which favor the development of thrombi are present, that treatment is of any use. In such cases the indications are expressed in exhaustion, weakened heart, and thickening of the blood. Stimulants, tonics, and diet are the resources at command. These must be employed promptly and vigorously. Unfortunately, it too often happens that the obstacles to nutrition are insurmountable, and treatment proves unavailing.

With each recurring observation of final convulsions in dysentery, I have mentally reviewed and compared the management of the case, hoping to discover the error which might be avoided in subsequent cases. Sometimes it has seemed that stimulants were too long delayed or inefficiently employed; at others, that they were too lavishly used and too little attention was given to the diet. In very young children, the pulse, condition of the anterior fontanelle, and symptoms of collapse, are safe guides in regard to the use of stimulants. After the closure of the fontanelle, the pulse and state of exhaustion constitute the only criteria by which the administration of

stimulants can be regulated. I apprehend error more often consists in an inefficient rather than in the excessive administration of stimulants. "It is better," says Jacobi, "for children to take in the course of the day three or six ounces of brandy and ten or twelve grains of camphor . . . than it is for parents to bury them the next day." Delay in commencing their use is perhaps a more frequent mistake. Stimulants withheld until collapse threatens immediate death, accomplish, as a rule, little more than the prolongation of life for a few hours. But, after all, nourishment constitutes the main reliance. Blood impoverishment progresses with extraordinary rapidity in dysentery. To counteract and stay the development of complications incompatible with life, believed to be the successive results of devastation of the blood, nutrition must be maintained during the acute stage of the disease, and not neglected or deferred until exhaustion is so far advanced and the blood has become so impoverished that digestion and assimilation are physiological impossibilities.

The proper alimentation of sick children is one of the most complex problems of the present day. The routine dietary of the nursery and sick-chamber indiscriminately and uniformly supplied to every sick child and to every disease, without regard to the demands of the animal economy and to the dietary and digestive idiosyncrasies of the patient, is as reprehensible as routine medication without a knowledge of the nature and progressive stages of the morbid process. Death demonstrates failure, but recovery oftentimes falls far short of establishing the value of the treatment.