THE SERUM TREATMENT OF TETANUS AFTER SYMPTOMS HAVE DEVELOPED

FRANK VAN DER BOGERT, M.D., SCHENECTADY, N. Y.

That there still remains some doubt as to the value of antitetanic serum after tetanus has manifested itself is evidenced by the paper of Newberger[1] as late as last July, in which the statement is made that "when tetanus has set in, the value of antitoxin may be questioned."

From time to time reports of cases in which unusually large doses of the serum have been used with apparent good results, have been published in the journals. Strock[2] of Camden, N. J., in 1907, after relating an experience extending over six or eight years with small doses and a mortality of 100 per cent., reported five cases treated with very large doses and a mortality of only 20 per cent. Strock expressed the opinion that this was not a coincidence and attributed his latest success to the much larger doses used. Livermore[3] of Memphis, Tenn., in 1907 spoke of a patient who died probably because not sufficient serum was available, and advised giving large doses. In 1910 Caffrey[4] of Milwaukee reported one case in which 112,500 units were given in eight days with recovery. He believed that this patient would have otherwise died and recommended great quantities of antitoxin. In February, 1912, Young[5] of Valparaiso, Ind., reported three apparently hopeless cases in which large doses were used, and followed by recovery, one patient, a boy, received 150,000 units and another, a man, 220,000.

During the present year a patient under my care at the Ellis Hospital received, between April 26 and May 15, 587,500 units of tetanus antitoxin obtained from the state laboratory. The diagnosis was based on a history of the child, a girl, 14 years old, having stepped on a nail about one month before admission (confirmed by finding the scar of a punctured wound at the base of the left great toe), and on the physical examination which showed trismus, neck rigidity, rigidity of extremities and pain on palpation of flanks and abdomen. Spasms were present.

The dose given is apparently a record one and shows, if not the value of large doses, at least tolerance to such dosage since the patient recovered.

Two symptoms probably attributable to the serum were noted. May 15, the day on which the last injection was made, the left hand became swollen and there appeared a mild urticaria. This rash and the temperature, which rose, shortly before the eruption developed, to a point not reached since the second day after admission and assumed an almost septic type, were attributed to the serum, and treatment was discontinued. Three days later the temperature had reached normal and convalescence rapidly followed.

In reviewing the clinical notes of the case I find records of a papular rash, May 2, which was still present May 3. The last note of spasm was under date of May 6, but there is a further note, May 10, reporting the patient as comfortable with no twitchings or spasms on that day.

1. Newberger, Charles: Tetanus as a Complication of Burns, Am. Jour. Dis. Child., July, 1912, p. 35; abstr., THE JOURNAL A. M. A., Aug. 3, 1912, p. 401.
2. Strock, Daniel: Tetanus and Antitetanic Treatment, Jour. Med. Soc. New Jersey, October, 1907; abstr., THE JOURNAL A. M. A., Nov. 30, 1907, p. 1876.
3. Livermore, G. R.: Antitetanic Serum Treatment of Tetanus, abstr., THE JOURNAL A. M. A., Dec. 14, 1907, p. 2031.
4. Caffrey, A. J.: Tetanus Successfully Treated with Large Quantities of Antitoxin, THE JOURNAL, Nov. 5, 1910, p. 1643.
5. Young, Simon J.: A Case of Tetanus with Recovery, THE JOURNAL A. M. A., Feb. 24, 1912, p. 549.
6. Herxheimer: Technik der pathologisch-histologischen Untersuchung, 1912.

A RAPID METHOD FOR CELLOIDIN SECTIONS

MAURICE BUCHSBAUM, B.S., M.D., CHICAGO

Herxheimer's[6] quick celloidin method takes twenty-four hours. The tissues are fixed in formaldehyd solution or alcohol, and then placed in pure acetone in the incubator at 37 C. (98.6 F.) for from thirty minutes to one hour. This is followed by thin celloidin for four or five hours, and thick celloidin for two or three hours. By Cullen's[7] method permanent specimens from frozen sections may be obtained by previous formaldehyd solution fixation in from fifteen minutes to three hours. This would be the method of choice if it could be employed advantageously in the examination of very soft and finely divided material, as uterine scrapings or epitheliomas from the meshes of which microscopic particles and cell nests are apt to drop out "as there is nothing to hold them *in situ* as when celloidin is used." Frozen sections do not stain so readily, and often become distorted in the handling. Nevertheless the freezing microtome has its field of usefulness when quick diagnosis is paramount over nicety of appearance. I do not offer my method as a substitute for this, but rather as an aid to the pathologist and surgeon to facilitate a histologic-pathologic diagnosis when pressed for time.

The principle of my method consists in shaking the specimen in contact with the fluid reagents. The agitation thus brought about within the fluid dislodges the air-bubbles confined within the meshes of the tissue and prevents the formation of a coagulation membrane, thus allowing the reagent to act simultaneously both centrally and peripherally. With the usual method the fixing and hardening is delayed because of the formation of a coating of albumin on the surface of the specimen; this coat of albumin then acts like an osmotic membrane, and the reagents penetrate slowly through it by osmosis.

The following are the steps:

1. Place small pieces of tissue, from 0.5 to 1.5 c.c., in 50 c.c. of a freshly prepared formaldehyd solution (liquor formaldehydi, 1 part, physiologic salt solution, 3 parts) and shake vigorously from one to three minutes.
2. Remove specimen into 30 per cent. alcohol and shake for thirty seconds. (In this the formaldehyd is removed and the hardening process begins.)
3. Transfer into 80 per cent. alcohol for one minute and shake for thirty seconds.
4. Transfer to absolute alcohol for five minutes, and shake for one minute.
5. Place in alcohol and ether for four minutes.
6. Transfer to Celloidin No. 1 for five minutes; shake one minute.
7. Place in Celloidin No. 2 for five minutes; preferably in incubator at 37 C.; shake for one minute.
8. Transfer to Celloidin No. 3 for ten minutes; shake for one minute then place bottle in incubator.
9. Blocking. Expose to the air for one minute on the block, then place in chloroform from three to five minutes.
10. Place in alcohol 80 per cent. until ready to cut. Stain as usual. The sections thus prepared stain beautifully with hematoxylin and eosin and keep permanently.

The whole process takes from forty-five minutes to one hour.

GANGLIONIC GLIONEUROMA OF THE OPTIC NERVE

G. C. RUHLAND, M.D., MILWAUKEE, WIS.

Pathologist, Milwaukee Health Department Laboratory; Consultant in Pathology at the Milwaukee Hospital and Johnston's Emergency Hospital

The ganglionic glioneuroma belongs to the rarer forms of nerve-tissue tumors and is found most commonly in the central nervous system and cord. The following case, however, is of special interest largely because of the unusual location and evolution of the tumor, which beautifully explain the clinical manifestations.

The history of the case, as given by Dr. C. S. Beebe, in whose practice it occurred and by whom the removal of the eye and tumor was performed, is as follows:

The patient, a girl aged 8, first began to show evidences of eye trouble at the age of 6. No examination was made by a physician at that time and the diagnosis of "some eye trouble" was the parents' own observation. At the age of 8, the child

7. Bull. Johns Hopkins Hosp., April, 1895, and May, 1897

contracted scarlet fever with rapidly developing exophthalmos and complete blindness of the "eye that was wrong." On enucleation of the eye, to which the parents now consented, an oblong tumor was found to occupy the position of the optic nerve.

Macroscopically, the tumor showed a well encapsulated semi-fluctuating growth, measuring 3 cm. in length and 1.5 cm. in width (Fig. 1). Microscopic examination revealed neurogliar tissue forced apart by hemorrhages and edema which had occurred into it. It also showed typical ganglionic cells and nerve fibers. Examination of the eyeball itself showed absolutely no involvement (Fig. 2).

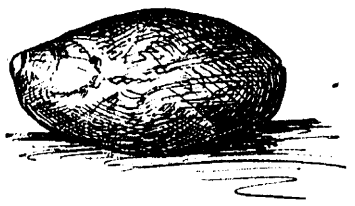

Fig. 1.—Gross appearance of tumor.

This tumor undoubtedly was congenital in origin and represents misplaced nerve-tissue. Its growth was characteristically slow until the febrile condition of the scarlet fever with its accompanying hyperemia stimulated the tumor into an active growth, which, together with the hemorrhages and edema, caused the rapidly increasing size of the tumor.

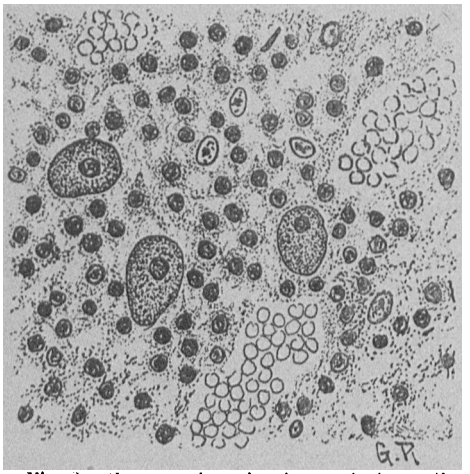

Fig. 2.—Cross-section, showing typical ganglionic cells.

While the location of the tumor naturally caused great damage to the patient, it was histologically a benign growth. Recurrence or involvement of the remaining eye has so far (now one and one-half years) not occurred. There is likewise no history of other members of the family having had similar tumors.

238 Twenty-Seventh Street.

Therapeutics

DIVERTICULITIS OF THE COLON

It is the object of this article to remind the physician of the possibility and not infrequent occurrence of an inflammation in the abdomen that is often overlooked, and consequently treated too tentatively for the safety of the patient. Though the physician generally recognizes appendicitis when it is present, still he is often puzzled by encountering on the left side of the body symptoms resembling those of appendicitis. It has been believed in certain cases that the appendix was located on the left side, and that abscesses due to inflammation of the appendix were sometimes found in the left side of the abdomen. More recent observations have demonstrated that at least a certain proportion of these cases of supposed left-side appendicitis were due, not to inflammation of the appendix, but to various pathologic processes occurring in the so-called diverticula of the lower portion of the colon.

A diverticulum may be encountered in various parts of the alimentary canal, as in the esophagus, the small intestine or the large intestine, but they are found more frequently in the lower part of the colon, especially in the sigmoid flexure, and sometimes in the appendix itself.

A diverticulum is a pouching or protrusion, more or less circumscribed, of the coats of the intestine. Little has been definitely determined about the etiology of this condition. The most that can be said is that for some reason a weakening of the coats of the intestine occurs, and that under the influence of some unusual pressure this protrusion or pouching takes place. It may include all the coats of the intestine, in which case the term "true" is applied to it; in others one or two of the coats of the intestine may be wanting, and then it is distinguished as "false." The latter variety is the more common. Some have believed that these diverticula occur more frequently along the mesenteric attachment of the intestine, but this is not an invariable rule, for not infrequently they are observed in the convex or free border of the intestine. In some cases they are found at the point where a blood-vessel enters the wall of the intestine.

Diverticula may be found on autopsy in persons who have never experienced any symptoms referable to their presence. Various pathologic processes may be set up in them, usually by the presence of food in a diverticulum of the esophagus, of intestinal contents in a diverticulum of the small intestine, or of fecal matter in a diverticulum of the large intestine. The pathologic processes vary greatly in character. There may be simply a catarrhal inflammation of the mucous lining, or there may be destructive changes, including ulceration and gangrene.

The inflammation may go on to perforation resulting in acute general peritonitis or the formation of an acute abscess or of an extensive indurated mass which may undergo resolution, or it may result in the formation of a chronic abscess. In the more chronic cases there may be inflammation with infiltration and thickening of the tissues which may go on to such an extent as to cause obstruction of the bowel. In some cases it is believed that a carcinomatous process may be developed on this infiltration. In some cases the extension of the inflammation may result in the formation of adhesions to other structures or of bands which interfere with the function of the intestine.

The symptoms resemble closely those of appendicitis, except that they are most frequently found on the left side of the abdomen instead of on the right side. There may be pain of greater or less severity, accompanied by a localized tenderness, and later an indurated mass may be felt on palpation. In the meantime there is usually considerable fever and acceleration of the pulse. There may be tenesmus or constipation.

In the chronic cases there may be more or less pain, soreness or tenderness and interference with the action of the bowels. If an abscess forms, it may discharge spontaneously through the rectum or bladder.

It is necessary, if possible, to distinguish this condition from malignant disease of the sigmoid flexure, and from inflammation of the left fallopian tube—salpingitis. McGrath[1] of Rochester, Minn., has recently emphasized the importance of not mistaking a simple inflammatory induration for cancerous degeneration. If the latter is believed to be present an unfavorable prognosis is given and operative interference is advised against, whereas if the simple inflammatory condition is recognized an operation may be curative.

1. McGrath, B. F.: Surg. Gynec. and Obst., October 1912, p. 420.