3. Variations in the protective potency of such organs as the liver, kidneys, etc., must cause proportionate variations in the symptoms.

4. The amount and character of the toxins present must vary according to the diet and to the amount and character of putrefactive changes taking place in the intestine.

The symptomatology of colonic intoxication may be greatly confused by the presence of other lesions which may be either the cause or sequel of the colonic trouble; such are appendicitis, gastric erosions or ulcers, cholecystitis, gall-stones, etc.

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DIFFUSE DILATATION OF THE ESOPHAGUS WITHOUT ANATOMIC STENOSIS (CARDIOSPASM)*

A REPORT OF NINETY-ONE CASES

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In 1908[1] I reported forty cases of "cardiospasm." In this series, thirty-eight patients had diffuse dilatation of the esophagus. Since that time fifty-six cases of diffuse dilatation of the esophagus without anatomic stenosis have come under my observation. The purpose of this paper is to report the result of treating these cases by dilating the cardia with a hydrostatic dilator.

The following have been advanced as factors in the etiology: first, primary cardiospasm (Meltzer[2] and Mikulicz[3]); second, primary atony of the musculature of the esophagus (Rosenheim[4]); third, the simultaneous development of cardiospasm and paralysis of the circular musculature of the esophagus brought about by degenerative changes in the vagi (Krause[5]); fourth, congenital disposition (Fleiner,[6] Zenker[7] and Sievers[8]); fifth, primary esophagitis (Martin[9]); sixth, kinking at the hiatus esophagi; seventh, gross lesions of the esophagus or stomach, such as ulcer, carcinoma, etc.; eighth, congenital or acquired asthenia. That some disturbance of the nerve muscle mechanism of the esophagus and cardia is responsible for the diffuse dilatation of the esophagus without anatomic stenosis seems to be the consensus of opinion.

Most American authors, following Mikulicz, have reported these cases under the heading "cardiospasm." The adoption of this term is premature and confusing, as the part that cardiospasm plays in the production of diffuse dilatation is by no means established and there are many cases, probably not directly related, that have spasm at the cardiac orifice without dilatation of the esophagus.

It is beyond the scope of this paper to discuss in detail the theories regarding the etiology of diffuse dilatation of the esophagus without anatomic stenosis or the etiology of cardiospasm. I wish, however, to call attention to a few points that have proved of practical interest in classifying these cases.

One hundred and thirty cases that have come under my observation have been grouped as follows:

1. Diffuse dilatation of the esophagus without anatomic stenosis, ninety-one cases. No gross gastric lesions were found in this group and only five of the patients were of a neurotic type.

2. Severe cardiospasm without diffuse dilatation of the esophagus, two cases. Both patients had periodic attacks continuing from three to fourteen days, during which they were not able to swallow either liquid or solid food. I think that in these cases diffuse dilatation of the esophagus would have ultimately developed.

3. Cardiospasm without diffuse dilatation but with gross lesions in the stomach, twelve cases. Of these, two patients had ulcer, two syphilis, five carcinoma and three suspected, but not absolutely demonstrated, ulcer.

4. Mild cardiospasm without diffuse dilatation of the esophagus or gastric lesions, twenty-four cases. Almost without exception these patients were of a neurotic type and many were distinctly hysterical.

That relatively few individuals of a neurotic type and none with gross gastric lesions were noted in the group having diffuse dilatation of the esophagus, that diffuse dilatation had not followed the cardiospasm in any of the patients with gastric lesions, and that almost constant evidence of an acquired or congenital asthenia was present in the patients with mild spasm without dilatation or gastric lesions seem to indicate that these groups are of different origin and not very definitely related.

Ten cases which will be reported in another paper should be mentioned here. Five had some anatomic deformity at the cardia or hiatus esophagi, and five a condition that is perhaps analogous to that found in congenital pyloric stenosis.

The diagnosis of diffuse dilatation of the esophagus without anatomic stenosis should rest on the demonstration of the dilatation and the absolute elimination of organic strictures. I presented the general principles of the technical methods of diagnosis[10] before this Section in 1909.

Russel, in 1898,[11] was the first to report a sufficient series of cases to demonstrate the efficacy of dilating the cardia with a silk-covered rubber balloon. Russel's work did not attract attention until Sippy[12] in 1906 reported a series of cures attained by this method. The description of the instrument and the technic which I have used since 1906 may be found in THE JOURNAL.[1]

After eliminating the questionable cases previously referred to and the cases of cardiospasm without diffuse dilatation of the esophagus, there are ninety-one cases of diffuse dilatation of the esophagus without anatomic stenosis in my series. Of these, four patients have died, three cannot be traced, seventy-three have been completely relieved of the dysphagia, and eleven are not completely cured. Among the deaths are two from pneumonia, one from tuberculosis, one, cause of death unknown, and one (Case 62) from rupture of the esophagus. Of the patients who died from tuberculosis or pneumonia all had been free from dysphagia for a period of two or more years. The cured patients have been free

* Read in the Section on Surgery of the American Medical Association, at the Sixty-Third Annual Session, held at Atlantic City, June, 1912.

1. Plummer: THE JOURNAL A. M. A., Aug. 15, 1908, p. 549.
2. Meltzer: Berl. klin. Wchnschr., 1888, No. 8.
3. Mikulicz: Deutsch. med. Wchnschr., 1904, xxx, 17, 50.
4. Rosenheim: Berl. klin. Wchnschr., 1902, No. 45.
5. Lossen: Deutsch. med. Wchnschr., xxix. Beil. Beil., p. 94.
6. Fleiner: München. med. Wchnschr., 1900, Nos. 16 and 17.
7. Zenker and v. Ziemssen: Cyclopedia of the Practice of Medicine, 1878.
8. Sievers: Hospitaltid., 1902, No. 30, Kjobenhavn.
9. Martin: Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1901, viii.
10. Plummer: THE JOURNAL A. M. A., Feb. 25, 1911, p. 560; also Tr. Sect. on Surg., A. M. A., 1910, p. 345.
11. Russel: Brit. Med. Jour., June 4, 1898.
12. Sippy: Paper read at the meeting of the American Medical Association in 1906. Not published.

from dysphagia as follows: three between six and seven years, twelve between five and six years, nine between four and five years, fourteen between three and four years, thirteen between two and three years, twelve for one year and ten for less than one year. Included among the cured patients are a few who at long intervals are conscious of the food passing the cardia.

Of the patients not completely cured two came under observation over four years ago, four over three years ago, one over two years ago, two over one year ago, and two within the last year. In five the treatment has not been sufficiently well followed up, two are of comparatively recent date and will require further treatment, three have had recurrences of dysphagia and regurgitation of food from the esophagus. They go, however, from periods of two to ten months without serious trouble. Two patients who were badly emaciated and regurgitated the greater part of their food still occasionally regurgitate and require water to wash down the food.

In my earlier work I determined the degree of pressure largely by the tolerance of the patient as indicated by the evidence of pain at the moment of greatest distention. The pressure was gradually raised at successive treatments until satisfactory results were obtained. In the majority of the first thirty cases the esophagus was dilated with a pressure of 500-575 mm. of mercury; in the next thirty-one, with a pressure of 675 mm. of mercury. In Case 62 the esophagus ruptured and the patient died of general peritonitis. The manometer indicated a pressure of 720 mm. at the time the rupture occurred. In the succeeding forty-five cases the esophagus was dilated with a pressure of 575-600 mm. The number of cases in which dilatation was obtained with a pressure of 575 mm. is sufficiently large to demonstrate the relative safety of using this degree of force. While I have always stopped the first treatment on the appearance of marked suffering and felt my way at succeeding treatments, I do not believe that the pain is a reliable index for judging the pressure to be used. The patient whose esophagus I ruptured did not complain of pain until the dilator was withdrawn.

In five patients in whom a pressure of 575 mm. failed to relieve the dysphagia and in three in whom the pain prevented me from using this pressure, I resorted to dilators of gradually increasing size, sufficient pressure being used to insure each size being distended to its maximum diameter at the cardia. This method of forcible dilatation is based on the assumption that there is some latitude between the points at which relief and rupture of the esophagus will occur. This method is tedious as the silk bag must be strongly made and of uniform diameter throughout its length and sufficient time must elapse between treatments to allow results to be noted.

A pressure of 675 mm. of mercury has invariably given marked relief and in a majority of cases a complete cure. It has been the routine practice to give the patients two or three treatments and allow them to go home with instructions to return if there be any recurrence of dysphagia. In no case have I followed up the treatment at frequent intervals over a long period of time, though a number of patients have returned at intervals of two to twelve months. It is remarkable that most of the patients with extreme inanition have needed but one treatment to effect a complete cure. In three patients I have had opportunity to demonstrate that the dilated esophagus has returned to normal size.

ABSTRACT OF DISCUSSION

DR. JESSE MYER, St. Louis: I rather object to the name diffuse dilatation of the esophagus without anatomic stenosis. Without an autopsy it is not impossible always to demonstrate that there is not an anatomic stenosis. With the aid of the esophagoscope and the Roentgen ray we can say definitely that there is a dilatation of the esophagus, but not that there is an anatomic stenosis. A case of mine of which I shall speak directly proves that point rather conclusively.

I believe that cardiospasm in most cases is a primary disturbance, and that the dilatation of the esophagus is a secondary condition, and the fact that these patients are relieved through the complete relief of the spasm seems to me to speak for the truth of this theory. The symptoms of dilatation manifested by the patient are the last and not the first to occur. The first symptoms are those of obstruction.

What constitutes a cure? It is possible in most cases, as Dr. Plummer has told us, to bring about a symptomatic cure, but I question very much if in these marked cases, cardiospasm with marked sacculation of the esophagus is ever completely cured from an anatomic standpoint.

I have had only fourteen such cases; I considered that a rather large series until I heard of Dr. Plummer's work. I have had eight patients under more or less constant observation for periods ranging from one to four years; three I have had under continuous observation for four years. In all the cases the sacculation remained practically of the same size after the patients had been completely relieved of their obstructive symptoms four years later. This I demonstrated by the Roentgen ray and an intragastric thin rubber bag attached to a stomach-tube which was introduced into the esophagus and filled under pressure with a suspension of bismuth in sour milk. I was able to introduce into the bag from 300 to 400 c.c. of the bismuth suspension, which was practically the same amount which I was able to introduce prior to the commencement of the treatment. The same thing was revealed in all the other cases (five) which I had under observation.

These sacculations persist, and give evidence of their existence throughout. The patients are greatly relieved and are apt to say that they are cured. If questioned closely, however, they will say that they are most comfortable when they eat slowly and chew their food thoroughly. Several patients have told me that cold drink or food causes more or less disturbance.

The method I have used of stretching or paralyzing the cardia is practically the same as that used by Dr. Plummer. I use, however, a dilator of the size of the cardia, and take the feelings and sensations of the patient as a guide in the treatment.

DR. MAX EINHORN, New York: One of my first cases of dilatation of the esophagus without obstruction I reported in 1888, and that was the second or third case that had been observed clinically up to that time. I believe that Meltzer was the first to describe this condition. Since 1888 I have seen each year a greater number of these cases. The condition is really rare, but anyone who has to deal with these troubles will see quite a number of cases and will have no trouble in recognizing them. I judge that I have seen over one hundred cases of diffuse dilatation of the esophagus.

Years ago I came to the conclusion that we could manage to keep our patients in a comfortable condition by arranging their diet and teaching them how to handle their food. Still there was never a cure established.

I was one of those who tried to stretch the cardia many years before Plummer did it, but I used bougies (54 French the largest) because I found that we could easily push them through the cardia. This upset the theory in my mind that we had to do always with cardiospasm, and I also saw that we did not bring on any change and so I gave it up. Later, when Plummer and others reported their very excellent results, I again started to dilate the cardia, using larger instruments, and I even attempted to construct a steel instrument, which could be unfolded when in the cardia or near it. About eight or nine years ago I found that the instrument was too stiff

to be handled easily, and so I discarded it again. Five or six years ago I devised another instrument, which could be introduced and opened up easily. I gauge the treatment by the way the patient feels, but I always try not to stretch too much at one time. I stretch it about twice a week, continuing the treatment until the patient is relieved of his symptoms.

I have treated about twelve patients in this way and the results are so striking that there is hardly any condition in which we can secure a greater improvement than I have obtained in these cases. After the stretching they eat just as well as anybody.

I have found that the esophagus does change in size, but this is really a matter of little consequence. What difference does it make if the esophagus does not change in size, provided the patient can eat well, and enjoy eating? There is, however, a change in the configuration of the esophagus. A skiagram made of such an esophagus a year after treatment showed that it was almost half again as large as before treatment. In another case there was no residue in the esophagus at all.

DR. ANTHONY BASSLER, New York: I wish to emphasize the point in Dr. Plummer's paper with reference to the 5 per cent. of cases that had disease at the lower end of the gullet. We all know that a large proportion of these patients are neurotic; that their trouble is of nervous origin, and of course they are immensely relieved by dilatation. Some patients can after dilatation eat anything and enjoy their meals for several weeks. Then they come back for another course of dilatation, have another period of relief and another dose of treatment; and so on. Finally, after a few months, one realizes the futility of the treatment, and sometimes we learn, much to our surprise and chagrin, that we are dealing with a case of malignant disease of the cardia. The cardiospasm which was temporarily relieved may have been an expression of organic disease of the esophagus or of the stomach, just as pylorospasm may be the expression of gall-stone disease or appendicitis. One should be sure that there is no local disease present. The esophagoscope is essential in diagnosis, but the *x*-ray helps very little until a marked stenosis exists, which is late in the disease or when malignant disease is present.

After very general use of the various instruments employed in the treatment of this condition, the dilatation of the cardia, I regard Dr. Plummer's method as being the best. I believe in complete dilatation of the cardia. It is paralyzed in suitable cases. When a person has not eaten any solid food for several months and has had several attempts at dilatation made, which have been only moderate, and then has dilatation by Dr. Plummer's instrument he gets complete relief and takes his first large meal, which is a satisfaction to him as well as to the doctor.

DR. H. S. PLUMMER, Rochester, Minn.: I believe that the majority of cases of diffuse dilatation of the esophagus are due to a spasm of the cardia. The truth of that statement, however, has not been demonstrated, and there is much evidence to refute it. I eliminated all cases that I considered questionable, and did not include them in the list of cases reported. I mentioned only ten cases. I tried to make the point that absolute demonstration of the absence of mechanical ileus of the cardia is essential.

With regard to the methods of dilating, I think we must rely either on the pressure used or on the size of the dilator. We tried to use different sizes of dilators and take the pain expression as a guide.

After Dr. Bassler's remark about dilating a malignant stricture, I doubt if that is a safe procedure for anyone to carry out except a very skilled esophageal worker. I would not make a diagnosis of diffuse dilatation of the esophagus with cardiospasm in any patient over 35 years of age unless the history showed that the trouble was of at least two years' duration. That is a very important point to bear in mind, because then you will avoid dilating a malignant stricture, to which Dr. Bassler referred.

NERVOUS SYMPTOMS FOLLOWING SUNSTROKE

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During the summer of 1911, because of the excessive heat there were more than the usual number of cases of heat exhaustion. It seemed to me, therefore, that a discussion of some of the resulting nervous symptoms would not be inappropriate. Of the number of cases seen in the course of years two are here reported because of their unusual character. The first was that of a patient in whom a severe sunstroke was followed by multiple nervous lesions producing acute cerebellar ataxia, loss of speech and spastic symptoms, an unusual combination. The second was that of a cook in whom because of overheating there occurred muscular spasms. The interesting feature was that he had an old poliomyelitis of one leg. In spite of this the muscular spasms were just as marked in the palsied limb as elsewhere, this being an interesting contribution to the theory that the spasms are produced by a degenerative process in the muscles and not by a lesion of the nervous system.

Although the literature of nervous symptoms following heat intoxication or exhaustion is not very large, yet a great variety of symptoms have been described. Besides the usual rise of temperature, headache and sometimes coma, there have been quite a number of cases of motor lesions which have been either hemiplegic or paraplegic and rarely acute ataxia and disturbance of speech. Curiously enough I have not found a single instance of such lesions producing sensory disturbances, although there is no reason why this should not occur.

Not much is known of the pathology chiefly because of the rarity of necropsy studies. As Oppenheim[1] states, it has been thought that there occurs cerebral hyperemia; by others thrombosis and multiple capillary hemorrhages in the medulla, anemia of the brain, edema of the pia, meningitis, cell-changes, dehydration of the tissues, auto-intoxication, etc. Considering the fact that the symptoms come on suddenly after a rise of temperature, it is not improbable that there is produced an auto-intoxication which in some cases causes multiple hemorrhages. Most of the reported findings tend to support this view and it is the explanation which I have given in the first case here reported.

It is evident that there must be something in the physical condition of an individual which renders him liable to succumb to excessive temperature. This needs no argument. I served for several years as a surgeon in the United States Army, being on duty in the Philippine Islands, and had an excellent opportunity to study the influence of temperature on newly arrived soldiers, and yet in spite of the excessive tropical heat there were very few cases of sunstroke. Among the natives this hardly ever occurred, whereas among the soldiers it happened in those who either foolishly exposed themselves or who drank. The memory of one case is distinct. It was that of a soldier who had been drinking quite severely the native drink, *bino*, distilled from some of the native plants. As was customary at that time, drill was held early in the morning, that is, about 7 o'clock, and this man went through an hour's work without difficulty. He ate very little breakfast and later on

1. Text-Book of Nervous Diseases, ii, 790.