patient. Either the surgeon has not gone far enough beyond the seat of the disease or has traumatized the intestine, or has made the gross blunder of infecting the patient himself, which is practically a thing of the past among those who do intestinal surgery.

CARDIOSPASM,

WITH A REPORT OF FORTY CASES.

H. S. PLUMMER, M.D.

Physician to St. Mary's Hospital.
ROCHESTER, MINN.

The first series of cases of so-called idiopathic dilatation of the esophagus was collected by von Ziemssen and Zenker in 1878. The report was based mostly on postmortem findings, little being known of the history of the cases. For our knowledge of the symptomatology of the disease we are indebted to the writings of Lichenstein, von Strumpell, Meltzer, Rumpel, Kraus, Rosenheim, Fleiner, Ewald, Netter, Kelling, Einhorn, Martin, Oppler, Gottstein, Dauber, Zausch, Strauss, Losen, Sippey, and others.

In 1904 Mikulicz estimated that 100 cases could be collected from literature. Since that time a number of cases have been reported, notably in German literature, and by Sippey, Lerche and Erdmann in this country. Gottstein, in Keene's "Surgery," states that 140 cases have so far been reported, and himself adds 25 more. This paper is based on 40 cases which have come under my observation. Eleven cases of this series were reported in a paper¹ read before the Minnesota State Medical Society in June, 1906.

The disease has been attributed to the following causes: first, primary cardiospasm (Meltzer); second, primary atony of the musculature of the esophagus (Rosenheim); third, simultaneous presence of cardiospasm and paralysis of the circular fibers of the esophagus, brought about by paralysis of the vagus (Kraus); fourth, congenital disposition (Fleiner, Zenker, Luschka and Sievers); fifth, primary esophagitis (Martin), and, sixth, kinking at the hiatus esophagi.

The unsatisfactory condition of our knowledge of the etiology of this disease is indicated by the variety of the titles under which the cases have been reported—idiopathic, fusiform, diffuse dilatation; dilatation without anatomical stenosis, etc.

Gottstein classifies motor disturbances of the esophagus as follows:

1. Hyperkinetic, in which spastic contractions of the muscle exceed the physiological normal.
2. Hypokinetic, in which there is an impairment of the normal muscular tone.

While it is probable that primary atony of the esophageal muscle rarely occurs, I doubt very much its being a frequent factor in the cases which have been reported under the headings, idiopathic dilatation of the esophagus, cardiospasm, etc. In the study of a considerable number of cases the almost invariable history of spasm at the onset, followed at a later period by the evidence of dilatation—that is, retention of food in the esophagus—is most convincing evidence that the spasm precedes the dilatation and that primary atony is relatively a rare condition. This conclusion is further borne out by the evidence of early muscular hypertrophy in nearly all cases which have come to postmortem; by the frequent observation of severe cardiospasm unaccom-

panied by dilatation of the esophagus, and by the good results which have followed forcible dilatation. The occasional case in which spasm can not be demonstrated does not exclude its being the primary factor. The spasm is in many cases periodic, and after dilatation takes place little if any more than the normal tone of the cardiac muscle is required to produce evidence of stenosis. Rosenheim, in support of the theory of primary atony, reports cases in which the food passed slowly through the esophagus, causing discomfort or pain, but he fails to show that these cases ever develop spasm at the cardia or dilatation of the esophagus.

The history, as given in Cases 11 and 24, suggests the possibility of dilatation almost from the onset. These histories date back eighteen and twenty-six years, and it is very probable that the more striking symptoms that came on with the dilatation overshadowed the early symptoms of spasm in the patient's memory. Severe spasm was present at the time these cases came under observation. Atony of the musculature of the entire gastrointestinal tract, without obstruction, is frequently observed, but it is almost invariably associated with other neurotic manifestations. There is no reason why spasm should exist in primary atony unless we accept Kraus' theory of a simultaneous atony and spasm from degenerative changes in the vagus. His theory rests on the finding of degenerated fibers in the vagus in one case which came to postmortem. His observations have not been confirmed.

The theory of congenital disposition is interesting but not susceptible of demonstration. That anlage may be a factor in some instances is suggested by the few rare cases reported as occurring congenitally or in early childhood (Luschka, Fleiner, Zenker, Gottstein). In six cases of this series sufficient angulation at the cardia was present to prevent the passage of a sound by the usual method. Whether this exaggeration of the normal obliquity of the insertion of the esophagus through the diaphragm is the cause of the spasm, or is a result, I am unable to state.

The cause of the spasm is largely speculative. A few cases have been reported associated with gross lesions of the esophagus, such as ulcers, fissures and small carcinoma in the cardia, carcinoma of the stomach, etc. We have seen three cases of carcinoma complicated by cardiospasm, and one case of hour-glass stomach due to syphilis with secondary cardiospasm. Esophagitis and ulcer of the esophageal wall not in close proximity to the cardia are to be looked on as secondary to cardiospasm and dilatation, not as primary factors. In the majority of cases, however, no such possible etiologic factors are to be found. Cardiospasm is not often present in inflammatory conditions of the esophagus which come under observation. Evidence of esophagitis previous to the onset of the cardiospasm could not be elicited from any of the cases. With three exceptions, none of the forty cases reported had neurasthenic symptoms. The age and sex of the patients may be seen by referring to the accompanying table. The average age at the onset is 29 years; twenty-two patients are females, eighteen males.

Deglutition is a complicated, reflex act in which many muscles cooperate. Without entering into a discussion of the exact part played by each, it may be stated that food is carried from the mouth into the esophagus by the omohyoids, hyoglossi, pharyngeal and associated muscles. In the upper portion of the tube the bolus is rapidly carried down by the cross striated muscles of its

1. Northwestern Lancet, September, 1906.

walls; in the lower portion it is moved more slowly by the peristalsis of the smooth muscle fibers. The cardia is supplied through the vagus with both dilating and contracting fibers, presided over and controlled by the medulla (von Operchowski). The normal action of this mechanism is but imperfectly understood, and even less is known of its disturbances in cardiospasm. The cardia is normally closed by contraction of its sphincter muscle, and is opened by its dilator nerves, and the bolus of food is pushed into the stomach by the peristalsis of the esophagus. Authors differ as to the way in which liquid food is carried along the esophagus. According to Cannon, Meltzer and Moser, liquids are carried deep into the esophagus or to the cardia by the forcible contraction of the omohyoids and associated muscles. Schrieber says that liquids are moved down the esophagus by peristalsis. Be this as it may, the esophagus is unable to force liquids and solids past an obstructed cardia with equal facility. After dilatation takes place the bolus of solid food is carried forward in a normal manner as far as the upper end of the dilatation. At this point the peristaltic contraction ring ceases to exert any direct force on the bolus, but sweeps around it.

Food is propelled from the dilated esophagus into the stomach by two forces, gravity and increased esophageal pressure. The force exerted by gravity equals the height of the column of liquid food in the esophagus. The intraesophageal pressure is raised above the normal by those forces which raise intrathoracic pressure, and by crowding more food into the partially filled dilatation. At each act of deglutition sufficient food is ejected into the stomach to lower the intraesophageal pressure to a point where it is no longer sufficient to overcome the resistance offered by the spastic cardia. Little, if any, more than the normal cardiac tone is required to cause the retention of food in the esophagus. These points are well illustrated by Cases 35 and 37. Each patient had learned to take sufficient food to maintain a fair state of nutrition, by first swallowing solid food to the point where he was conscious that more would be regurgitated; then at the end of inspiration several swallows of fluid were rapidly and forcibly thrown into the esophagus, thus using the pharynx and upper portion of the esophagus much like the pump of a hydraulic press. The patient in Case 37 has acquired to a nicety the ability to practice this act. She stated that very rarely, if ever, during the last few years of her trouble had any food been regurgitated. During the period in which she was under examination, previous to dilating the cardia, from four to twelve ounces of food could be withdrawn from the dilated esophagus five hours after eating.

In the development of cardiospasm three stages, more or less clearly defined, may be recognized in the clinical histories.

1. In this stage the peristaltic contraction of the esophagus is sufficient to force the food through the spastic cardia. This is characterized clinically by the complaint of discomfort, pain, choking sensation, etc.

2. The peristaltic force of the esophageal muscle is no longer able to overcome the resistance of the contracted cardia, and the food is immediately regurgitated. This may result from an actual or relative increase in the obstruction at the cardia; that is, the spasm may become accentuated or the esophageal muscle may tire under the unusual load. In some cases the spasm is sufficiently severe from the onset to cause the immediate regurgitation of food. As a rule the spasm is, during

the early part of the history, periodic; but as the condition develops mild spasm becomes more continuous or constant. Marked exacerbations in the spasm characterize the entire course of the disease. The increased work of forcing the food past the contracted cardia results in an early hypertrophy of the esophageal musculature. As the spasm increases in severity the esophagus becomes less and less able to overcome the obstruction, and atony and dilatation result.

3. Once the esophagus begins to give way the dilatation is rapid. This stage is characterized clinically by the retention of food in the esophagus and its regurgitation at irregular intervals after ingestion. If the spasm is mild for a sufficient length of time from the onset, the hypertrophy may become extreme and dilatation not take place or be delayed for years. The hypertrophy, after becoming well developed, may overcome a most marked resistance at the cardia. Relatively frequent severe spasm at the onset may lead to early dilatation before hypertrophy of the esophagus has time to develop. An impaired tone in the esophageal muscle at the onset of the trouble may also have something to do with the rapid early dilatation that takes place in some cases.

The symptom-complex in cardiospasm is as a rule almost pathognomonic. It may be divided into the three stages noted in considering its development: first, cardiospasm without food regurgitation; second, cardiospasm with immediate food regurgitation; third, cardiospasm with dilated esophagus, the retention of food in the dilated portion and its regurgitation at irregular intervals after taking. In the majority of cases the first attack of spasm occurs suddenly and unexpectedly when the patient is at the table. A spasmodic, choking sensation is felt at some point in the course of the esophagus, generally located at the cardia and radiating to the back or neck. It is rarely described as a pain. Some patients locate the discomfort entirely in the epigastrium, left hypochondrium or in the upper portion of the esophagus. It may occur for months independent of the taking of food. In Case 40 many physicians had, during the course of twenty years, agreed with the patient in attributing the obstruction to an adenoma of the thyroid. The spasm is often described as a slight delay in the passage of food or the food "seems to stick beneath the lower sternum."

In the second stage the food is, after swallowing, immediately regurgitated. During the early portion of the history the attacks occur periodically, or mild spasm is continuous, with periods of marked exacerbation which are characteristic of the entire course of the disease. The patient in Case 9, in which the esophagus was not dilated, had periods of three to seven days in which all solid food was immediately regurgitated, alternating with periods of days or weeks of absolute freedom from dysphagia. In Cases 6, 27 and 28 the first attack of spasm was severe enough to cause immediate food regurgitation.

After dilatation of the esophagus takes place the spasmodic, choking sensation may be absent. The first portion of each meal is retained. After filling the sac, further food is regurgitated or forces the preceding portion into the stomach. Of the contents of the esophagus at the completion of a meal the more fluid portion may slowly seep through the cardia; the solid food with much mucus is either regurgitated at irregular intervals or remains in the sac until the next meal. Solid food, like meat, may be delayed at the cardia for several days,

softer food in the meantime passing into the stomach. The sac is never completely emptied, several ounces of solid food often being present in the dilated esophagus when the patient states that it is going through all right. On many occasions I have withdrawn from two to sixteen ounces of food after twenty-four to thirty-six hours of fasting. That there is a sense of weight or discomfort in the chest, and that the food is not sour, are statements common to all cases. The vomiting is painless and but rarely accompanied by nausea. Some insist that the food enters the stomach but will not stay. The majority state that it lodges beneath the lower sternum. Ten cases of this series complained of waking at night to find the nasal passages filled with food.

Of this series, seven cases came under observation during the second stage and thirty-three during the third stage. Four cases of secondary cardiospasm are not included. Loss of weight was notable in the majority of the cases. A few maintained a fair degree of nutrition in spite of the dysphagia. Two of the cases in the second stage and ten of the cases in the third stage were markedly emaciated. The patients in Cases 10 and 41 could not walk without assistance, and the patients in Cases 22 and 42 were brought in on cots.

Fig. 1. Whalebone staff with olive drilled for silk thread.

Fig. 2. Whalebone staff, spiral tip and olive.

Fig. 3. Dilator.

In the diagnosis and study of a case of cardiospasm the following points should be demonstrated:

1. The food is regurgitated from the esophagus and not from the stomach.
2. The existence and character of obstruction at the cardia.
3. The presence or absence of esophageal dilatation and its shape, size, etc.
4. The presence or absence of gross lesions in the esophagus or neighboring organs which might excite the cardiospasm.

In the demonstration of these points the following measures have been resorted to:

1. The various stomach-tube tests, including the methods proposed by Rumpel, Kelling and Einhorn.
2. Passage of a bulbous sound on a whalebone staff.
3. The passage of the sound which I have devised.
4. Radiographing a bismuth mixture in the dilated esophagus.
5. Determination of the size of the dilatation, by means of a rubber balloon distended within the esophagus.
6. Esophagoscopy examination.

Each case is given a test meal. If sufficient obstruction exists to prevent the food from entering the stomach, it is given with a tube; a second portion of the meal is given in the usual way. At the end of an hour the tube is again introduced and the stomach contents obtained. The tube is now withdrawn into the esophagus, the contents obtained, and the two portions are analyzed. The esophageal contents in all cases were alkaline or neutral in reaction. In the analysis of the stomach contents no marked variations from the normal were noted. The average acidity was below normal; a high acid content was present in a few cases. In all except Cases 7, 13, 17, 18, 30, 39 and 43 (second stage) food was present in the esophagus. On many occasions several ounces of food were withdrawn from the sac after

Fig. 4. Dilator, showing arrangement of tubing.

twenty-four hours of fasting. In nearly every instance when a tube was passed during a remission in the complaint, and the patient stated that the food was going through all right, small or even large amounts of food were found in the esophagus. The simple introduction of a tube usually causes the regurgitation of non-acid food, which comes up in a manner that leaves little doubt of its source. In eight cases it was possible to pass a tube in the ordinary manner; in sixteen cases, with a wire stylet or a staff and olive; in ten cases by means of a silk thread and a whalebone staff; in six cases by means of a silk thread, staff and spiral tip referred to later. The more complicated tests of Rumpel,

Einhorn and Kelling were frequently resorted to, but are unnecessary and often unsatisfactory.

The failure to pass a stomach tube when an olive readily passes the cardia is at once suggestive of cardiospasm. In thirty cases the passage of a 15 mm. olive was possible without encountering any marked obstruction. In ten cases it was impossible to pass the bulb, at least without using greater force than I considered safe. In these cases the sound was passed on a silk thread as a guide. The patient slowly swallows six yards of silk thread. This passes down through a sufficient number of coils of intestine to prevent its withdrawal on being pulled taut. It is advisable to have the patient swallow three yards in the afternoon and the remaining three yards on the following morning. In this manner the first portion forms a snarl in the esophagus or stomach, which passes out into the intestine during the night, the remaining portion passing without snarling. The olives are drilled for threading from the tip to one side of the base. With five exceptions, no difficulty was experienced in passing a 15 mm. olive when using the silk thread as a guide. As a rule a slight increase in the resistance was encountered at the cardia, but frequently the olive passed without its detection. Rarely was that slow giving way at the cardia, frequently described by authors, encountered when the sound was passed on the thread. It can at least be stated that marked resistance to the passage of a sound in cardiospasm does not often exist, provided it enters the cardia directly. Obstruction sufficient to prevent the passage of a sound is due to its being caught by contact with the bottom of the sac; and the sense of the slow giving way of the cardia under gentle pressure is due to the resistance encountered in sliding the olive along the flaring wall of the esophagus or the straightening out of some fold just as it is about to enter the cardia. The character of the resistance met at the cardia with the knowledge obtainable only by passing the sound on a thread, that the sound is directly entering the cardiac orifice is of the utmost importance in the differential diagnosis of organic and spasmodic stricture at the cardia. In six cases, using a whalebone staff 3 mm. in diameter, I found it impossible to pass the sound. The patient in the first case encountered could take sufficient food to make it seem improbable that sufficient narrowing at the cardia could exist, either from cicatricial or spasmodic stricture, to prevent the passage of a 3 mm. olive. Only one conclusion was tenable, namely, that there must be some angulation or tortuosity present. I had the flexible tip shown in Fig. 2. made to screw on to the end of the whalebone staff. It is very flexible, but will not double on itself when subjected to a force that can be safely used in sounding. With this tip the 15 mm. olive readily passed the cardia in the six cases referred to.

Radiographs were made in all except Cases 1, 2, 3 and 4. Exposures were generally made after giving the patient two ounces of bismuth mixed with kumiss, mucilage of acacia, or starchy food. The greatest contrast was obtained by radiographing a rubber dam balloon, distended with the bismuth mixture, within the dilated esophagus. The dilated esophagus is most clearly shown by placing the tube and plate so that the rays penetrate in an oblique direction from the right anterior to the left posterior, thus throwing the shadow to the left of the spine on the plate. However, this distorts the shadow more than a picture taken directly anteroposteriorly. The shadows in all except Cases 7, 13, 17,

18, 20, 30 and 43 indicated dilatations which would hold from six to twenty ounces, and corresponded in size with the measurements obtained with my sound and by filling with water. In all but three cases the dilated esophagus was clearly shown and the position of the shadows and their shapes and relations to the surrounding parts were such that it would be almost impossible to mistake them. Radiographs showed the dilatation to be spindle-shaped in 18 cases, cylindrical in 12 cases and pyriform in 3 cases.

The dilatations which were spindle-shaped had an S-like curve, the lower convexity being to the left and the upper convexity to the right. The dilatation almost uniformly extended as high as the third dorsal vertebra. In Case 5 the shadow extended from the cardia to the upper margin of the sternum and had an almost uniform diameter of two and three-fourths inches.

In Cases 7 to 40 the radiographic findings were confirmed by a method of sounding to which I first resorted in Case 7 to demonstrate the absence of dilatation. This sound is constructed the same as the Russell bag used for dilating the cardia. A rubber dam balloon is attached to the lower end of a stomach tube in such a manner that the tube is closed, and holes are punched in the tube so that its caliber communicates with the interior of the balloon. A spherical or oval silk bag 22 mm. in diameter is drawn over the balloon and fastened to the tube. This is introduced into the stomach with a whalebone staff and distended with water under sufficient pressure to make the stilet, tube and balloon form a solid sound. The sound is drawn up to locate the cardia, collapsed, drawn into the esophagus and distended. If, under distension, the sound can be moved freely up and down, it is withdrawn and the silk bag replaced by a larger one. In this way, by using a series of sounds of increasing size, the diameter of the esophagus at any point, and an approximate idea of the shape and size of an existing dilatation, may be obtained. The demonstration of a sac by this method is also proof that the sac is a dilatation and not a diverticulum, provided the sound is first introduced sufficiently far to give assurance that it has entered the stomach.

Strauss' method of measuring the capacity of the esophagus is more accurate and should be carried out in all cases for the purpose of determining the fate of the dilated esophagus after the cure of the cardiospasm. A rubber balloon is distended within the esophagus with air, and the amount of air measured as it is introduced.

To complete the diagnosis an esophagoscopic examination should be made. In addition to the catarrhal changes, evidence of muscular hypertrophy, ulcerations, scars, papillary excrescences, etc., may be noted. The cardia appears funnel-shaped or like a rosette. Fissures and ulcers at the cardia should be carefully watched for, as they may be looked upon as exciters of the spasm and require either preliminary treatment or additional care when dilating the cardia. The occlusion at the cardia is in many cases sufficient to prevent the passage of the instrument. This is due not so much to the strength of the spasm as to the impossibility of presenting the esophagoscope perpendicularly to the plane of the cardia. This difficulty may be overcome either by an esophagoscopic obturator, or, in some cases, by painting the cardia with a cocaine solution.

The obturator is made of steel and tapered so that the flexible tip gradually merges into the rigid tube of the esophagoscope. The olive and cylindrical portion of the obturator are drilled so as to permit of passing it

on a silk thread. This obturator in many cases very much facilitates the introduction of the instrument at the introitus.

Until recently the treatment of cardiospasm has consisted in such ineffectual measures as attention to the patient's general condition; fluid, non-irritating diet; effervescent drinks; bromids; the frequent passage of large sounds; and, as a last resort, gastrostomy. In the first four cases of our series gastrostomy was done. Three of the cases were operated on at St. Mary's Hospital for stenosis of the cardia. Its cause was unrecognized at the time, but in each instance the condition of the patient warranted a gastrostomy. After the operation, periods of feeding by the mouth alternated with periods of introducing food through the gastrostomy opening. In one case the patient completely recovered; two were finally lost sight of. Case 4 was afforded much relief for several years by the frequent passage of a large bougie. These cases are introduced into the series for future reference.

Mikulicz² reported that he had operated in four cases with apparent perfect success. A gastrostomy was done and under the control of two fingers a long, curved forceps, the blades covered with rubber, was worked into the cardia. The forceps was gradually opened until the maximum distance between the blades reached six or seven centimeters. Erdmann³ reports one case entirely cured by this method, after an interval of twenty months. Case 8 of this series was completely relieved for four months by this operation. This major operation only is warranted provided the condition is serious and that equally good results can not be obtained by simpler means. Russell⁴ was the first to report a sufficient series of cases to demonstrate the efficacy of dilating the cardia with a silk-covered rubber balloon. Four cases were completely cured, one was much improved, and one was not improved. About thirty cases have been reported as cured by this method.

The construction of the dilator as used in the cases here reported is readily seen from Figures 3 and 4. It is made by cementing a rubber dam balloon to one end of a piece of non-elastic rubber tubing in such a manner that the tube just passes through the balloon. A number of holes are so punched in the tubing that its lumen connects with the interior of the balloon. A sausage-shaped silk bag is drawn over the balloon to preserve its shape on distention. Several sizes, 10 cm. long and from 20 mm. to 40 mm. in diameter, are provided. A second rubber balloon is drawn over the instrument to facilitate its introduction. If the dilator is slightly constricted in its middle third, the tendency for it to slip into the stomach is lessened. Provision is made, for removing the staff to prevent its maceration when not in use. To avoid the annoyance incident to exchanging silk bags, it is well to have several dilators, only one staff being necessary. The metal tip of the staff is threaded for the set of olives used in sounding and for the spiral steel tip previously referred to. The dilator is connected by rubber tubing with a water-tap or pump. Into this tubing two hard-rubber T joints are inserted, one for an altitude gauge or manometer, and the other for a piece of tubing to permit draining the instrument without disconnecting at the tap, and for controlling the pressure. The position of the cardia having been previ-

ously determined, the dilator is introduced sufficiently far for the cardia to engage the balloon at its middle third. The instrument is firmly held with the right hand, the index finger resting against the teeth, to prevent the dilator being drawn into the stomach. The tap is now opened enough to fill the instrument and deliver a good stream through the tubing for drawing the instrument, but not to indicate any pressure. The pressure is now slowly raised by pinching the tube between the thumb and index finger of the left hand. Various pressure regulators have been used, but none give the absolute control which is possible with this simple device. The pressure is, up to a certain point, determined by the tolerance of the patient, as indicated by the pain caused at the moment of distention. It must be sufficient to paralyze the sphincter. If a pressure of 500 mm. will not accomplish this, the pain is discarded as a guide, and dilatation is carried out by the use of dilators of gradually increasing size, sufficient pressure being used to assure each size being distended to its maximum diameter at the cardia. The safety of this method of forcible dilatation is based on the assumption that there is a certain latitude in the size to which the cardia may be dilated between the points at which paralysis of the sphincter and rupture of the esophagus will occur.

Many of the cases are relieved by a pressure of from 50 mm. to 100 mm. The number of dilatations has ranged from one to five, with a few exceptions. In two cases, 7 and 13, in which dilatation of the esophagus was not present, the treatments were repeated respectively seven and eleven times. The cases in which angulation at the cardia was present required more forcible and a greater number of dilatations. Within four or five days after the first dilatation, some food begins to lodge in the esophagus. The first two or three treatments are repeated at intervals of three to four days. The case is not discharged until the esophagus is found free of food remnants at the end of ten days. In the early cases which recurred, this rule was not carried out.

If a fissure is known to exist at the cardia, or if the patient is greatly emaciated, or if severe pain is caused by dilating, an attempt to carry the dilatation hurriedly to the point of giving relief should be avoided, and a pressure of only five or six feet of water employed. This will give relief for a few days at a time, and allow the healing of a fissure or ulcer. Later, the dilatation can be carried to the point of producing sufficient paralysis to effect a permanent cure.

In those cases in which the olive can be passed through the cardia, the dilator enters with equal facility. In those cases in which difficulty was encountered, it was overcome by the same methods which were used in passing the bulbous sound. In the recent cases I have invariably used the silk thread, as it makes the passage of the instrument more rapid, safe and certain.

In two cases of long standing cardiospasm without dilatation, it was some time before I succeeded in dilating the esophagus or recognizing the cause of failure. In these cases the upper portion of the dilator can not fully expand, and hence the portion within the stomach acts as an expanding wedge, drawing the instrument down. If the staff is firmly held against the incisor teeth, the diaphragm is drawn up to its limit and the cardia then dilated.

The immediate results are most striking. The patients are almost invariably able to take any kind of food at the following meal. There is often a complaint

2. Deutsch. med. Wchnschr., January and February, 1904.

3. Ann. Surg., February, 1906.

4. Brit. Med. Jour., June 4, 1898.

of soreness for the first twenty-four hours. The gain in weight and strength is rapid; even in those cases which were apparently well nourished there has been a gain in weight from ten to forty pounds. Attention is called to the tabulated history in which the number of treatments and recurrences may be noted. In 29 cases there has been no recurrence. Marked return of the symptoms has not occurred in any case as all were cautioned to return on the slightest evidence of trouble. That the disturbance has recurred in so few cases has been a constant source of surprise. Among those more recently treated there will undoubtedly be some recurrence, but I think that recurrences will be rare in the cases which have remained free from trouble for two years or more. The cases which I have had opportunity to examine at some time after treatment show that there is a tendency for the dilated esophagus to return to its normal size. As far as possible these cases will be followed up and the ultimate results reported.

TABULATED REPORT OF FORTY CASES OF CARDIOSPASM.

Case Number.	Sex.	Age.	Stage.	Duration, Years.	Date First Treatment.	Number Treatments.	Date, Recur. and Treat.	Number Treatments.	Second Recurrence.
5	F.	58	3	5	2-6-06	3	0	..	..
6	M.	18	3	1/2	1-20-06	6	7-6-06	4	0
7	M.	29	3	3	1-9-06	6	7-8-06	3	0
8	F.	21	3	3	2-28-06	3	8-1-06	2	0
9	F.	30	3	3	5-12-06	2	0	..	..
10	M.	40	3	3	6-30-06	2	0	..	..
11	M.	30	3	18	6-30-06	10	10-27-06	3	0
12	M.	32	3	3	8-2-06	6	0	0	0
13	M.	33	3	3	8-17-06	10	0	0	0
14	F.	41	3	20	9-20-06	10	0	..	..
15	F.	37	3	3	11-1-06	3	0	..	..
16	F.	24	3	3	11-3-06	3	0	..	..
17	F.	40	3	3	12-20-07	6	3-06	2	..
18	F.	42	3	3	2-27-07	10	0	..	..
19	M.	27	3	1 1/2	3-4-07	10	0	..	..
20	F.	25	3	3	3-9-07	3	0	..	..
21	F.	57	3	3	3-18-07	3	0	..	..
22	F.	38	3	3	3-20-07	3	0	..	..
23	F.	34	3	3	3-29-07	4	0	..	..
24	F.	41	3	25	4-20-07	4	7-3-07	3	0
25	M.	38	3	4	4-25-07	6	0	..	..
26	M.	29	3	15	4-28-07	3	10-27-07	3	0
27	M.	29	3	2	5-15-07	7	8-3-07	3	0
28	M.	21	3	6	5-15-07	5	11-11-07	3	..
29	F.	31	3	4	6-5-07	10	0	..	..
30	M.	38	2	2	6-8-07	6	0	..	..
31	M.	33	3	1 1/2	7-19-07	5	0	..	..
32	F.	18	3	3	9-6-07	10	1-8-08	4	..
33	M.	46	3	12	9-10-07	6	0	..	..
34	F.	38	3	12	10-13-07	6	1-3-08	3	..
35	F.	32	3	3	10-13-07	3	0	..	..
36	F.	34	3	9	10-19-07	5	0	..	..
37	F.	21	3	3	10-26-07	2	0	..	..
38	M.	47	3	2	12-23-07	3	0	..	..
39	F.	17	2	1	1-3-08	4	0	..	..
40	F.	62	3	20	1-6-08	4	0	..	..
41	F.	38	3	6	2-2-08	5	0	..	..
42	M.	59	3	15	2-5-08	2	0	..	..
43	M.	19	2	4	2-10-08	3	0	..	..
44	F.	38	3	3	3-6-08	2	0	..	..

Negri Cell Inclusions in the Salivary Glands.—Hoen, in the *Bulletin of the University College of Medicine, Richmond, Va.*, July, calls attention to the demonstration of the Negri cell inclusions in the secreting cells of the salivary glands (parotid) by a special combination stain of hematoxylin, fuchsin, ammonium picrate and gram. While priority of discovery is always of interest, as indicating the investigative trend of any laboratory, it is of minor importance when compared to the significance of the finding of structures, in the organs furnishing the infectious fluid, which are identical, morphologically, with those found in the brains of rabid animals. This discovery practically validates the assumption of Dr. Negri that these bodies, named after him, are the etiologic factor of rabies.

A NEW TECHNIC FOR NEPHROPEXY.

ALEXANDER HUGH FERGUSON, M.D.

CHICAGO.

The purport of this short paper is to describe in as few words as possible the technic of a nephropexy which has satisfactorily suited my cases for more than three years. In the multiplicity of procedures practiced by surgeons and in the many relapses that occur some prominent internists find reasons to discontinue altogether operations for the relief of floating kidney. It is safe to assert that there is no drug or external support that cures a case of this order, while even the simplest fixation by operation relieves many of them. Since applying the technic here described there have been no recurrences.

Floating kidney is no longer a border line disease; it belongs to the field of surgery. A brief survey of its pathology is overwhelmingly convincing of the demands on surgery for its treatment.

Let me make it clear that a kidney should not be insulted by an operation simply because it is extensively floating. I have in mind a case of twenty-five years standing in which the kidneys can be easily made to cross the spinal column, but no operation has been advised for two reasons: 1, No distress is occasioned, and 2, no important urinary changes have arisen.

INDICATIONS FOR OPERATION.

A few of the pathologic indications for operations are:

1. Transitory hydronephrosis due to kinking or folding of a ureter and characterized by intermittent renal colic and other less prominent symptoms, such as an irregularity in the quantity and quality of the urine voided.

2. Chronic hydronephrosis with more or less atrophy of kidney tissue, following a more or less permanent obstruction to the urinary flow through the ureter. The kidney may now become fixed away from its normal site.

3. Unilateral interstitial nephritis not infrequently is caused by the kidney floating. The classic symptoms and signs are present, with constant aching and occasionally colic in the region of the wandering organ.

4. Acute torsion of the ureter, giving rise to great and constant pain with, of course, enlargement and tenderness of the floating kidney.

5. Acute infection of a hydronephrosis—inflammatory symptoms constitutionally, chill, rapid pulse, fever, colic, pyuria, etc.

6. Suppression of urine in a floating kidney with Dietl's crisis bespeaks a condition that should be relieved by operation.

The above mentioned conditions give rise to many local and remote disturbances. In addition to abnormal findings in the urine, e. g., albumin, bile, blood, casts and pus, we often find the patient suffering with painful and frequent micturition; or headache and symptoms of uremia; or gastrointestinal disturbances, dyspepsia, vomiting, abdominal distress, constipation, and even diarrhea.

AN ILLUSTRATIVE CASE.

The nervous condition known as neurasthenia is certainly not benefited by the presence of a floating kidney as a factor, if not in producing, certainly in perpetuating the nervousness when renal colic, etc., is present. Two years ago I fixed the right kidney in a neurasthenic young woman, spare, small and emaciated. In the region of the right kidney there was constant pain, magnified the moment her attention was drawn to it. The