clinically with the fact that the symptoms of motor irritation and paralysis are, in the later stages of the disease, almost invariably complicated with sensory and trophic disturbances, such as anesthesia, paralysis of the bladder, bed-sores, muscular atrophy, etc.

REMARKS

ON A

CASE OF PHLEGMONOUS GASTRITIS.¹

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MARTHA M., aged 49, was admitted into St. George's Hospital under my care on July 4th, 1883. It appeared from the history that she had been for several years subject to bilious attacks, in which severe vomiting with occasional diarrhoea were prominent symptoms. She had lost all her teeth, and was accustomed to drink large quantities of warm liquids. The diarrhoea had been very troublesome since the beginning of 1883; and on April 24th of that year she was an in-patient under Dr. Wadham, in a state of great prostration, after prolonged diarrhoea. There was on this occasion no vomiting. With rest and light diet (fish, etc.), the symptoms improved, and she was discharged relieved in a fortnight.

About a month after she had left the hospital, the diarrhoea returned; and on July 1st an old femoral hernia came down, causing her much abdominal pain, more especially in the epigastrium. When in the recumbent position, the hernia disappeared; but this was at first followed by profuse and continuous vomiting; at first of dark matter, then of thin bilious fluid. The bowels were freely open.

She was admitted into hospital at the request of Mr. Chippendale, of Upper Phillimore Gardens, who had been called in to attend her. At first, she was placed in a surgical ward, as it was supposed that the symptoms might depend upon the hernia. No rupture being discovered, she was at once transferred to me.

The patient was badly nourished and slightly jaundiced. She had lost all her teeth. She complained of abdominal pain, referred chiefly to the epigastrium. No tumour was detected. The abdominal walls were flaccid. The vomiting was almost continuous; the vomited matter was small in quantity, fluid, opaque, green, and strongly acid.

Small doses of acetate of morphia with sulphate of atropia were given to relieve the pain; while at the same time, with a view of maintaining the patient's strength, nutrient enemata containing brandy were administered. The injections were retained fairly well; but the vomiting did not entirely cease, nor did the character of the vomited matter alter.

On July 6th, the patient sank into a comatose state; the temperature gradually rose from normal to 104° Fahr., with the symptoms of peritonitis; and she died soon after midnight.

Necropsy, twelve hours after death.—*General Appearances.* Height, 5 feet; black hair; no teeth: jaw atrophied. *Lungs.* Posterior portions of both congested. *Heart.* Weight, 12 oz. The mitral valve was thickened and contracted; only one finger could be passed between its flaps. The remaining valves and orifices were natural. *Abdomen.* The peritoneal cavity contained a small quantity of purulent fluid. *Stomach.* The contents were thin greenish yellow fluid. The mucous surface at the cardiac third of the organ was very red, felt thick, and infiltrated with fluid. On transverse section, it was a quarter of an inch thick. The remaining two-thirds of the stomach, so far as the inner surface was concerned, presented no abnormal appearance; but, on transverse section, the pyloric end was found infiltrated with a purulent effusion between the muscular and mucous coats. On the posterior surface of the organ, below the lesser curvature, there was an area about one inch wide by three inches long, which was of a distinctly yellow colour; and, at this place, section showed an abundance of purulent fluid between the muscular and serous coats. The remainder of the alimentary canal was natural. *Liver.* Weighted

3 lbs. 12 oz.; its surface was smooth. The tissue was pale and fatty. *Spleen.* Healthy; weight, 7 oz. *Kidneys.* Weighted 8 oz. The capsules were adherent; the surfaces were granular; the cortical parts diminished, and mottled slightly with dark coloured spots. Microscopic examination confirmed the naked-eye appearances of the walls of the stomach. There was an extensive infiltration of the submucous tissue, chiefly by pus-cells. The pus was most abundant around the secreting glands, the cells of which were somewhat swollen, and their contents opaque and granular. At that part of the posterior surface of the organ where the yellow discoloration of the external surface was found, as described above, all the coats of the stomach were infiltrated with pus.

After the patient's death, the friends brought out a box labelled "podophyllin pills." They stated that she had taken one or two of them on the night before admission; but they were unable to say whether or not she was in the habit of taking them.

REMARKS.—Cases of acute inflammation of the walls of the stomach have received but little attention in the ordinary English text-books. In foreign works, however, more attention has been paid to this affection. Raynaud, in the *Bulletins de la Société Anatomique*, 1860; and Leube, in Ziemssen's *Cyclopadia*, vol. vii, devotes a considerable space to the disease—gastritis phlegmonosa. According to the latter, it occurs in two forms: 1. circumscribed suppuration; 2. diffuse purulent infiltration of the parietes, extending around and between the gastric glands. In the circumscribed form, the mucous as well as the serous layers are usually intact, or at most only erythematous. "The abscesses of the stomach are sometimes single, at other times multiple." "Cases of diffuse purulent infiltration of the parietes have been observed less frequently, and less exactly." In this latter, the condition of the mucous layer varies greatly in the different cases examined; it is sometimes unchanged, at other times infiltrated with pus or serum. Occasionally the muscular layer may be destroyed by the purulent infiltration.

The serous layer may be also unaffected, according to the seat and extent of the phlegmonous inflammation. In other cases, it may take on inflammation, accompanied by the accumulation of dirty grey thick masses of exudation upon the surface of the stomach. In these cases, the subserous layer may be infiltrated with serum or pus.

Of thirty-one cases collected by Leube, twenty-six occurred in males and five in females (Ziemssen's *Cyclopad.*, vol. vii, p. 157.) Bamberger (*Handbuch der Speciel. Pathol. und Therapie: Krankheiten des Chylpoetisch. Syst.*) devotes but a single short paragraph to this affection of the stomach; his description of the morbid appearances agrees with that of Leube.

Etiology.—It appears, from the records of the cases which have been observed, that the cause is unknown. According to Leube: "In a number of cases, the origin of the affection has been attributed to excessive indulgence in alcohol; in a few, to some wound occurring in the region of the stomach; in others, again, to some error of diet." An obvious objection occurs at once to such explanations of the cause of phlegmonous gastritis—viz., that in countries such as England, to say nothing of other nations, where drinking is carried on to so great excess, and in which errors in diet are an equally prevalent fault, instances of this disease are of rare occurrence. I purposely omit those cases in which inflammation of the walls of the stomach has followed acute diseases, such as typhoid fever, puerperal fever, etc. It is within the knowledge of all medical practitioners that, in many persons, certain articles of diet exercise a deleterious effect—e.g., in some, lobster or other shell-fish invariably produce an attack of urticaria. In my own experience, a few strawberries always have this effect in a boy about ten years of age. Podophyllin is differently tolerated by different individuals. In some, its action is most beneficial; in others, it has the reverse effect. In a patient of my own, a quarter of a grain is sufficient to render her so irritable that, as she expressed it on one occasion on which I prescribed it, she was "unable to sit still all day;" while the idiosyncrasy which renders many persons intolerant even of a few grains of potassium iodide, is too well recognised to require comment.

It may be that, in certain persons, an attack of phlegmonous gastritis may result from the ingestion of articles of diet usually considered wholesome, or from the administration of drugs in ordinary use; hitherto, however, its occurrence under any circumstances is a rare event.

We have no evidence that the patient whose case forms the subject of the present communication had ever taken podophyllin before; and with such deficient information no conclusion can be drawn in this case, one way or the other.

Leube states that, "as regards age, those who died of gastritis phlegmonosa were, almost without exception, in middle life." The age (49) of the patient in the present instance is a case in point.

¹ Read before the Medical Society of London.

Symptomatology.—To quote Leube again, the cases have been too seldom observed to enable an accurate description of the disease to be given, and in this he is in accordance with Raynaud.

Bamberger (*loc. cit.*) states that a diagnosis of the disease is, from want of observation, impossible, the main difficulty in diagnosis lying in the fact that the symptoms of the original malady are always masked by the concomitant peritonitis.

It would appear that the duration of the disease may vary from three to eighteen days, the average being nine days (Leube). The leading symptoms in the cases recorded are: that, after symptoms of gastrointestinal derangement have existed for some days, the patient is attacked with severe abdominal pain, chiefly in the region of the stomach, high fever, diarrhoea, and finally irregularity of the pulse, delirium, great prostration, and death. The rapid course of the inflammation of the stomach and to the peritonitis to which it gives rise is such as to render all treatment futile.

Of the recorded cases, I have selected one or two in which the notes are given in some detail; subjoined is an epitomé of them:

1. Dittrich's case (abridged from Ziemssen's *Cyclopaedia*, vol. vii, p. 160). A tanner, entirely convalescent for a fortnight from pleurisy, felt quite well on October 20th, 1866, dined with good appetite, and spent the day at work as usual. On returning home on the same evening, he experienced severe chills, alternating with fever, and accompanied by pain in the region of the stomach, heart, and left thorax, so that he could not sleep that night. Next morning, there was repeated vomiting of bilious matter, while the former symptoms continued. In the evening, the patient had the aspect of a person seriously ill, with increased temperature, rapid pulse (112). The abdomen was neither distended nor painful; he complained of nausea and eructation; there was occasional vomiting. One-twelfth of a grain of morphia was ordered to be taken four times during the night. The vomiting ceased next day; but, on October 24th, the patient was much worse in every way, *i.e.*, higher temperature, flushed face, occasional delirium, rapid and soft pulse, short and laborious respiration. Early on the following morning, collapse supervened, and, in spite of stimulants, the case terminated fatally on the morning of the 25th. At the necropsy, sero-purulent inflammation of the submucous layer of the stomach was found, also peritonitis and pleurisy; the two last being probably due to extension of the inflammation of the stomach.

2. Glaix (*Berliner Klinische Wochenschrift*, 1879, No. 16, 565-8, epitomé in *London Medical Record*, 1879, p. 303) records a case of a workman, aged 52, with rigors and violent sickness, which lasted twenty-four hours. The man was of intemperate habits. When first seen by Glaix, the patient's abdomen was flat and hard, and very tender to pressure in the ventricular and ileo-caecal regions; the spleen and liver were much enlarged; he had lost much flesh. The temperature was 104.5°; pulse, 136; respirations, 36. The treatment consisted of quinine internally, ice to the head, and galvanism applied to the neck once in two hours. On the following morning, the man was very restless; his extremities were cold, and cyanotic. Pulse soft, 130; temperature, 102°; respirations, 24; urine very scanty. The bowels acted several times spontaneously. A hypodermic injection of Fowler's solution and pure alcohol was made, after which the pulse rose; but he became again collapsed, and died next morning.

At the *post mortem* examination, the peritoneal cavity contained muddy stringy fluid mixed with shreds of mucous membrane. The blood-vessels of the peritoneum were much injected; the liver was enlarged and fatty; the spleen was of three or four times its natural size. The stomach was enlarged, and contained a few ounces of yellowish stringy fluid; its walls were abnormally thick and soft, and fluctuating to the touch. On section of them, a whitish stringy liquid mixed with pus trickled from the submucous cellular tissue. The walls of the stomach were infiltrated with this fluid; the mucous membrane was pale, save in some places, where it was hyperæmic and ecchymosed. The case was considered to be one of idiopathic phlegmonous gastritis and general peritonitis. Glaix states that he has observed a second case of this disease, which, however, was not verified by *post mortem* examination, as the patient recovered. The *etiology* of this case was clear, the patient having been imprudent in eating. This was followed at once by symptoms of inflammation of the stomach. Pus was vomited, whereupon the temperature sank, facts which lead to the supposition that a submucous abscess had burst.

3. In the *Bulletins de la Société Anatomique de Paris*, 1861, p. 63, M. Raynaud has an article on this subject, and gives the following cases, the first recorded by Cornil.

A man, 40 years of age, of strong constitution, but addicted to drink, was admitted into the Charité Hospital on May 26th, 1860. He had suffered no external injury; he had been ailing for three months,

but had only given up work for fifteen days. On admission, he was delirious; convulsive movements of the body existed; his face was pale, without expression; his lips were red, his tongue dry; his pupils dilated and inactive. Vomiting, which had been a prominent symptom for two days previously, had given place to profuse diarrhoea. The pulse was very small and irregular; respiration slow; the belly distended and hard, did not appear tender. The patient died on the day of his admission, without treatment.

The *post mortem* examination revealed extensive peritonitis. The stomach was adherent to the omentum, its walls were softened and thickened with an infiltration of plastic exudation and pus.

In this case, the mucous membrane was slightly injected, but not softened; the submucous tissue was much increased in thickness and infiltrated with colourless fluid and pus-cells. In short, the submucous tissue was in a condition similar to that of the subcutaneous areolar tissue in diffuse phlegmonous erysipelas.

This case during life appears to have been mistaken for meningitis.

The second case was that of a carter, aged 44. The patient was of a bilious temperament, strong constitution, but enfeebled by hard work and insufficient food. He had spat blood several times, ten years ago, and again a month or six weeks previously to his admission into hospital. He was short of breath, had cough, and sweated occasionally at night, but presented few physical signs of pulmonary disease. The pulse was very slow. Fifteen days later he was attacked with diarrhoea, accompanied by tenesmus and bloody stools. The left iliac fossa was tender to pressure; there was nausea, but no vomiting; the tongue was red and glazed. The diarrhoea had ceased on admission, and for several days the bowels did not act. He was kept on light diet, was free from abdominal pain, and was allowed to walk in the garden. He was, in fact, considered to have recovered, and, on the seventh day after his admission, it was proposed to discharge him. However, he then complained of a feeling of uneasiness, loss of appetite, and nausea. Vomiting then set in with abdominal pain, and, on the ninth day after admission, the belly was tender and somewhat distended, his pulse small, 120. Leeches and fomentations were applied to the belly. These symptoms having become more urgent, leeches were again applied to the abdomen, and, on the twelfth day following his admission, diarrhoea set in, the stools being bloody. Ice was applied externally, and opium was administered by the mouth. Next day the patient became delirious, with a dry tongue and weak pulse. The patient gradually became weaker, the abdomen more distended, and he died on May 29th, having been admitted on May 15th, 1860.

At the necropsy, extensive peritonitis was found. The walls of the stomach were thickened; the mucous membrane was thickened but not softened. Towards the larger *cul-de-sac* was a bright red patch, apparently the result of inflammation. Pus was found between the mucous and muscular coats, or between the latter and the peritoneal covering.

Raynaud's article, from which I have extracted the preceding cases, is the most complete of the earlier accounts of phlegmonous gastritis. In it he refers to records of such cases as far back as the year 1620; by Varandæus (*Tractatus de Morbis Ventriculi*), and again to a Dutch physician, named Henon (*De Morbis Ventriculi*, 1658), to Petri Borrelli in 1656, and to the *Sepulchratum* of Bonetus. Most of the descriptions, however, furnished by these authors; and by writers of subsequent periods, are those of circumscribed abscess of the stomach, rather than of diffuse phlegmonous inflammation of its walls.

Dr. Habershon, in his *Pathological and Practical Observations on Diseases of the Alimentary Canal*, relates the case of a woman, aged 40, who was admitted into Guy's Hospital on May 2nd, 1847, who had for a fortnight suffered from pain in the loins, and subsequently in the stomach and chest. She suffered also from anorexia, incessant vomiting of black bitter fluid, and intense thirst. Death was preceded by coma.

At the necropsy, peritonitis was found. At the pyloric end of the great curvature was a hard mass measuring four inches and a half by three inches and a half. On the inner surface corresponding to this mass was an irregularly shaped patch on the mucous membrane of a dark brown colour of the size of a shilling. On section pus was found infiltrating the submucous tissue. The pus was not fluid.

Dr. Budd, in his work on the *Organic Diseases and Functional Disorders of the Stomach*, 1855, mentions a similar case.

In some of the recorded cases, ulceration of the mucous membrane existed; but this ulceration is considered by Raynaud to be the result rather than the cause of the disease. One may add to this that, if ulceration were the cause of such a virulent form of inflammation, we should, considering the frequency of gastric ulcer, expect to meet with phlegmonous gastritis as a disease of every day occurrence.

In order to confine this paper within due bounds, I have limited my

remarks to simple purulent infiltration (if one may use such an expression) of the walls of the stomach, of which my case appears to be an instance, and have purposely omitted more than a casual reference to circumscribed abscess of that organ. As the two are, however, so intimately connected, I may, perhaps, conclude with Raynaud's summary at the end of his article referred to above.

1. The term gastritis should be restricted to the sense which custom has assigned to it, that is to say, inflammation of the mucous membrane, without any tendency to extension to the cellular tissue.

2. There are three distinct varieties of abscess of the walls of the stomach—intramucous abscess, circumscribed abscess of the submucous tissue, purulent infiltration more or less general.

3. The circumscribed abscess appears, in the majority of cases, as a chronic condition, and, perhaps, one of the pathogenic forms of gastric ulcer.

4. The purulent infiltration is an anatomical condition, liable to arise in many general diseases, characterised by a disposition of different organs to the production of pus.

NOTE ON ESBACH'S METHOD FOR ESTIMATING THE QUANTITY OF ALBUMEN IN URINE.

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ALTHOUGH much has recently been written in this country regarding the use of picric acid, it is rather surprising that no attention has hitherto been given to M. Esbach's method of employing it for the quantitative as well as for the qualitative determination of albumen in the urine.

This method, which dates from 1874, consists in measuring, in a graduated tube, the height of the deposit given by a measured quantity of albuminous urine, when treated with a solution containing picric acid and citric acid in the proportions respectively of 1 and 2 per cent.

The Test-Tube.—This is somewhat stouter than an ordinary test-tube. It is six inches long, and about nine-sixteenths of an inch in diameter. On its outer surface, it bears a scale of seven degrees, diminishing in size from below upwards, whereby the quantity of albumen represented by the deposit is intended to be read off in grammes per litre, *i.e.*, in parts per 1,000. Somewhat above this scale is a dotted line marked U. (urine), and at a certain distance above it another line marked R. (reagent).

The Test-Solution.—This is made by dissolving one part of picric acid and two parts of pure air-dried citric acid by the aid of heat, in about 80 or 90 parts of distilled water, adding subsequently enough water to make exactly 100 parts. If only the apothecaries' weights and measures be available, a solution of similar strength may be made by dissolving 35 grains of picric acid and 70 grains of citric acid in 8 fluid-ounces of distilled water.

Mode of Proceeding.—1. The urine should be filtered.

2. If not distinctly acid, it should be rendered so by the cautious addition of acetic acid, drop by drop, until it changes litmus-paper to a brick-red.

3. If the specific gravity exceed 1.010, the urine should be diluted by one or more volumes of water, so as to bring it below that figure.

4. If, after the first trial, the urine be found to contain more than 4 per 1,000 of albumen, it should be diluted so as to bring the deposit below the line 5 of the tube; and in either of these two cases, the indication given by the deposit must be doubled or trebled according to the degree of dilution.

5. With the urine thus prepared, fill the tube up to the line U. Add the reagent up to the line R. Close the tube with an India-rubber plug, and gently turn it up and down a dozen times, so as thoroughly to mix without frothing the contents.

6. Let the tube remain at rest in the upright position for twenty-four hours, and, at the end of that time, read on the scale the amount of the deposit.

Example.—A patient passes in 24 hours 1,000 cubic centimetres of urine, = 35½ fluid ounces—of specific gravity 1024. A filtered portion of this is diluted with twice its bulk of water, and the deposit, after 24 hours, reaches to the line 3 of the tube. Then, this number multiplied by 3, on account of the dilution, indicates 9 grammes per litre (*i.e.*, 9 parts per 1,000), as the quantity of dry albumen contained in the specimen.

Of course, the same number divided by 10 will give the parts per

100, and, if this be multiplied by the factor 4.4 (strictly, 4.375), it will give, very nearly, the number of grains per fluid-ounce; thus, $9 \times 4.4 = 3.96$.

If it be desired to use the test-solution merely to detect the presence of albumen, the following method will be found advantageous. Pour a little of the solution into a test-tube, add the urine, previously filtered, drop by drop, and if, at the point of contact of the two fluids, a haziness show itself immediately, the urine is certainly albuminous; but if the haziness be very slight, and not instantaneous, it may be due to a precipitation of uric acid. In this case, repeat the experiment, and warm the solution before adding the urine.

When I first became acquainted with M. Esbach's method in 1880, it seemed to me so handy and useful for clinical purposes, that I did not hesitate to introduce it into the clinical medical wards at Netley; but it was not until last autumn that I found leisure to compare it with the common method of precipitating the albumen of urine by heat and nitric acid, and to determine the value of the results in each case by comparing them with those given by the ponderal method. For the common method, I used a test-tube, having an internal diameter of 15 millimetres, = $\frac{1}{4}$ of an inch, with a cubic centimetre scale marked on its surface from below upwards. The quantity of urine used was always the same, *viz.*, 12 cubic centimetres. The boiling and acidulation were conducted in the usual manner, and the coagulum was allowed to settle for twenty-four hours. Of course, the urine was filtered before it was tested.

The following table gives the results of the last ten consecutive analyses then made.

No.	Specific Gravity of the Urine.	Quantity of Albumen in parts per 1,000.		Height of the Deposit given by Heat and Nitric Acid.
		By Esbach's Method.	By the Ponderal Method.	
1	1023	.. 3.0 ..	.. 3.8 ..	3.00 c. c.
2	1022	.. 4.2 ..	.. 3.9 ..	3.25 "
3	1022	.. 3.8 ..	.. 4.0 ..	3.75 "
4	1022	.. 3.9 ..	.. 4.0 ..	3.25 "
5	1025	.. 5.4 ..	.. 5.4 ..	6.25 "
6	1026	.. 4.8 ..	.. 4.8 ..	2.751 "
7	1025	.. 3.9 ..	.. 4.0 ..	3.75 "
8	1022	.. 4.2 ..	.. 4.1 ..	3.50 "
9	1020	.. 3.9 ..	.. 3.6 ..	3.75 "
10	1020	.. 3.5 ..	.. 3.0 ..	3.50 "
Means		4.06	4.06	3.675 c. c.

¹ Coagulum not all settled to the bottom.

From this it appears that Esbach's method affords results, which, if not always absolutely correct, are nevertheless accurate enough for all clinical purposes; and as such it is, I think, deserving of the attention of the medical profession in this country. It has the additional advantage of being so simple that it might be performed as a daily duty by any intelligent nurse, or even by the patient himself if in a state to make observations. It is certainly better adapted for cases in which the loss of albumen is large, as in nephritis and certain cardiac diseases, than for those in which the albuminuria is slight and transitory; but, in the latter, a daily quantitative estimation of the albumen discharged with the urine would very seldom be considered requisite.

It is also seen that, even with an ordinary test-tube, graduated in cubic centimetres, and with heat and nitric acid, the deposit from twelve cubic centimetres of urine enables one to form a fair approximation, directly, to the quantity of albumen contained in 100 parts, and thence, by an easy calculation, to the number of grains per fluid-ounce. In this case, the urine requires no preliminary acidulation, although, when its specific gravity exceeds 1020, dilution with one volume of water will generally give a better deposit, and therefore more accurate results. Moreover, the tests are those with which every one is familiar, and on which many medical practitioners continue to place the greatest amount of reliance.

I have only to add that albuminometers of the kind which I have described can be obtained at a very cheap rate from Messrs. Townson and Mercer, of 89, Bishopsgate Street Within, E.C.

POISONOUS PAPERS.—It would appear that the coloured papers commonly used in kindergartens are not all of them free from poison, for an investigation by Dr. Henry Leffmann shows that, in some cases at least, the blue papers contain ultramarine; the yellow, lead-chromate; but the red a large amount of arsenic. When we remember that children, even children whose parents believe in the kindergarten system, are fond of sucking everything they come across, it would seem that there is a real danger in the use of these papers.