than from the mastoid process (positive experiment of Rinne); also that low tones are better perceived by air conduction than high ones; and, lastly, if there is a history of symptoms pointing to the implication of the labyrinth, such as giddiness or total inability to hear the ticking of a watch through the head bones, then the existence of an affection of the labyrinth is very probable, and it is in such a case that the subcutaneous injection of pilocarpine should be employed. I use but rarely subcutaneous injections of pilocarpine in certain forms of acute inflammations of the middle ear.

In the first of my works mentioned below I have cited several cases of acute inflammation of the middle ear in which perforation had not taken place. Where a protracted local treatment failed to produce reabsorption of the hardened exudation products lying in the cavity, and where after three or four subcutaneous injections of pilocarpine a constant improvement could be observed, the effect must be referred to the speedy solution and absorption of such exudation products. In like manner the subcutaneous injection of pilocarpine is to be recommended in those cases of acute suppuration of the middle ear in which perforation of the tympanic membrane has occurred, and in the course of which deafness supervenes, dependent upon some complication of the labyrinth.

Within recent years I have used muriated pilocarpine locally in affections of the labyrinth and the middle ear, injecting from six to eight drops of a 2 per cent. warmed solution through a catheter into the Eustachian tube and cavum tympani. When pilocarpine is thus employed in the above-mentioned concentration and quantity no unpleasant results supervene; it is only in rare cases that salivation and abundant diaphoresis are observed, and these soon after injection. In chronic catarrhs of the middle ear I inject pilocarpine into the tympanic cavity, particularly in those cases where the power of hearing is distinctly improved after inflation of the middle ear, and where a slight swelling in the Eustachian tube can still be discovered upon auscultation. The object of the local application in these cases is to bring about a slight reaction in the mucous membrane of the middle ear, and in this way to cause the absorption of inflammatory products. And, as a matter of fact, in some particular instances the improvement of hearing following these injections has been more pronounced than that obtained by the use of 10 per cent. solutions of soda, which until now have been most frequently employed. On the whole, the cases in which the local injections of pilocarpine produce a notable result are but few; in most cases the faculty of hearing does not improve at all, or the improvement is only indifferent and of short duration. Here, too, the treatment must not be extended beyond two or three weeks. It is quite otherwise with the so-called cases of chronic catarrhs in the middle ear, where the adoption of this method of treatment must be deprecated. I have already shown that, in the year 1885 (*Wiener Med. Zeit.* 4, 5, 6), and again in my Text-book of Ear Diseases (1887), the treatment of dry catarrhs of the middle ear with pilocarpine is worthless. The position to-day has not changed. It must also be insisted upon that in every case of extreme deafness where the tuning-fork can be heard longer from the mastoid process than opposite the ear (air conduction, negative experiment of Rinne), and, in addition, where low tones are not at all or only faintly perceived through the medium of air, while high ones are at the same time very distinctly heard, the subcutaneous treatment with pilocarpine is strongly contraindicated.

The above remarks have been found necessary from the circumstance that for some time past I (I am not singular in this experience) have been consulted by many patients suffering from extreme deafness who had been previously treated by other aurists for several weeks, or even for several months, with subcutaneous injections of pilocarpine without the slightest benefit, in the cases of which an examination showed the presence of a marked sclerosis of the mucous membrane of the middle ear.

While it is true that the subcutaneous injections of pilocarpine usually can be tolerated by the patient for several weeks without any unpleasant consequences, still there are cases in which its protracted use has produced loss of appetite, faintness, and considerable emaciation. When I consider the abundant salivation and diaphoresis, so disagreeable to the patient, which are associated with the daily use of this remedy, I feel it my duty to say that those practitioners who in all cases of extreme deafness indiscriminately—that is, without a previous careful examination by means of the tuning fork and consequently without any differential diagnosis between affections of the middle ear and those of the labyrinth—subject their patients to a long and wearisome course of treatment with pilocarpine are not too conscientious in the discharge of their calling.

The following is a summary of the above: 1. The subcutaneous injections of pilocarpine are particularly indicated in recent affections of the labyrinth, be they of a syphilitic nature or not. In protracted diseases of the labyrinth these injections, if tried, must be abandoned if no improvement results after from ten to fifteen injections. 2. The subcutaneous injections of pilocarpine are but rarely employed in otitis media acuta, where the cavum tympani contains hardened exudative products, which resist reabsorption; moreover, in panotitis gemina diphtheritica, or in other diseases produced by infection. 3. The subcutaneous injections of pilocarpine are decidedly contraindicated in cases of dry sclerotic catarrhs of the middle ear. 4. Injections of several drops of a 2 per cent. solution of muriated pilocarpine through the catheter into the tympanic cavity are beneficial in some cases of catarrhs connected with swelling and a slight secretion of the mucous membrane of the middle ear, continued from one to three weeks alternately, with inflations of air by Politzer's procedure. The purpose of the present communication is to reduce to a just measure the therapeutic value of the subcutaneous injection of muriated pilocarpine in diseases of the ear, and to draw attention to the frequent abuse which has been made of this remedy for some time past.

DIVERTICULUM OF ŒSOPHAGUS CAUSING OBSTRUCTION; GASTROSTOMY; DEATH SIX YEARS AFTERWARDS.

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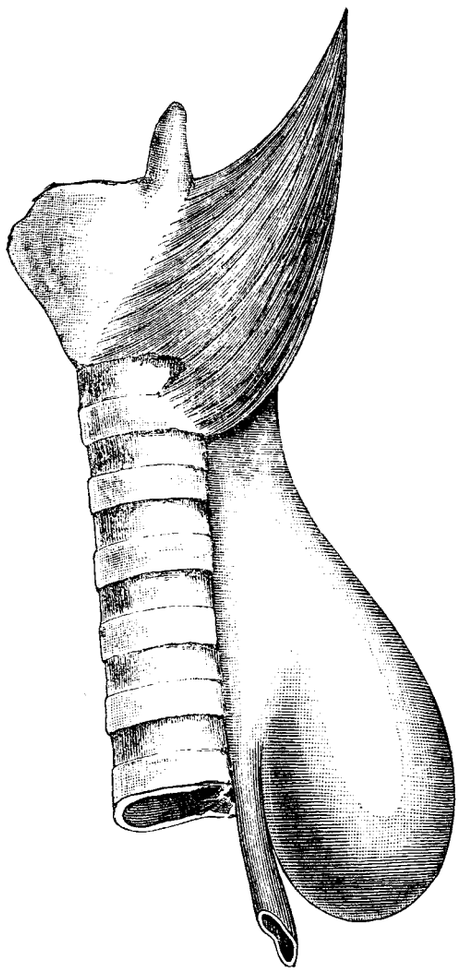
THE following is a good example of a rare lesion of the œsophagus; it is also of interest as affording an opportunity for the examination of a gastrostomy many years after operation. The patient, a married woman having had previously good health, began in her forty-ninth year to have difficulty in swallowing, which gradually increased in severity. On Nov. 13th, 1882, eight years later, when deglutition had almost become impossible, the patient came under my care. She was then unable to swallow any solid food, and even liquids invariably regurgitated. Attempts to pass bougies of various sizes uniformly failed. There was no history of syphilis, or of any kind of injury to the œsophagus, and the duration of the symptoms rendered the probability of cancer to be unlikely. The patient's condition was so critical that gastrostomy appeared the only method of averting imminent starvation, and this was in consequence performed four days later. Convalescence was rapid and complete, and the patient returned home on Dec. 6th. For two years she systematically used the gastric fistula for all alimentation, and her health continued excellent without suffering any inconvenience from the operation, or being conscious of any unusual condition of the gullet. She gained in weight, and was to all appearances in robust health—in fact, was so convinced that her old trouble had disappeared, and naturally objecting to the inconveniences of her singular mode of feeding, she commenced, by degrees, to take food by the mouth. For several months she was unconscious of any return of her old trouble, but gradually, and at first almost insensibly, she became aware that her power of swallowing was becoming less natural and her old symptoms steadily reviving. About this time (December, 1886) she consulted Dr. Watson of Burnley for sciatica, without, however, at first drawing his attention to the difficulty she experienced in swallowing. During his attendance he accidentally noticed that she swallowed in a peculiar manner, which he subsequently described as follows:—"When taking liquids the patient tossed her head backwards, gulping down the fluid at one mouthful. This was immediately followed by some

thickening and congestion of the neck and slight congestion of the face. On applying a stethoscope over the cricoid cartilage the usual gurgling sound was heard, immediately followed by eructations and a crackling noise, the latter being, perhaps, due to the meeting of the eructated gases with the descending fluid. When taking solids the same phenomena occurred, with this difference, that the eructations were accompanied by a whistling noise. Vomiting took place sometimes in a few minutes afterwards; at other times it was deferred for half an hour." Dr. Watson further informed me that the patient rapidly became anæmic, attacks of acute neuralgia became frequent, and eventually she died, apparently from sheer exhaustion, in January, 1888, without the obstruction of the œsophagus ever again becoming complete. He was fortunate in obtaining a post-mortem examination, and although not successful in securing the important parts removed, he was happily able to make a sketch of the œsophagus, which is here reproduced.



Dr. Watson also furnished me with a copy of his post-mortem notes, which are too valuable to be omitted. "I found," he states, "the different organs of the body smaller than normal, especially the stomach. The aorta, however, was enlarged. At the commencement of the œsophagus, just at the inferior constrictor, I found a large pouch, fully three inches in length and two inches in breadth. It apparently consisted of all three coats of the œsophagus, and projected from its posterior wall so as to lie between the œsophagus and the vertebral column. It was half full of partly digested food. There was a cicatricial scar and a small fistulous opening on the abdomen, indicating the point where gastrostomy had been performed. The attachment of the stomach to the skin consisted of the merest filament, and the opening into the stomach was almost closed."

Remarks.—Pressure or acquired diverticula—and this appears to be a typical example of that condition—are, according to authority, rare in women, for out of twenty-two reported cases collected by Zenker and von Ziemssen in which the sex was stated not one was recorded as occurring in a female.[1] The reasons which appear to justify this case being considered in the category of a pressure diverticula, in preference to regarding it as one of congenital origin, are: the late period of life when the symptoms commenced, their slowness of development, and the position of the pouch—viz., at the back of the pharynx—at its junction with the œsophagus. Zenker and von Ziemssen,[2] in an article based upon a series of thirty-four cases, twenty-seven of which were verified by necropsies, found that in pressure diverticula the commonest situation was, as in this case, the posterior wall of the pharynx at its junction with the œsophagus, where the tube becomes suddenly narrower and more liable to injury. They further point out that the pouch in these cases is formed by a protrusion of the mucous membrane, through the separated fibres of the inferior constrictor, and is, in fact, a simple hernia of the mucous membrane—a pharyngocele—and that these occasionally attain dimensions of five inches and a half long by two inches broad. The only other possible alternative is to assume that the malformation may have originally been congenital, for when we remember that the gullet is developed by the union of the ingrowing pouch of the pharynx with the alimentary canal it is conceivable that if the upper end of the œsophagus were to open only a little in front into its anterior wall instead of exactly into the apex of the pharyngeal pouch, a small cul-de-sac would be left behind the œsophagus at its junction with the pharynx, and such a cul-de-sac might eventually form the commencement of a large diverticulum. It may be stated that Hamburger,[3] Rokitansky,[4] and Ziegler[5] consider the acquired forms to be due to the lodgment of foreign bodies in the œsophagus leading to weakness of the muscular fibres, and subsequent protrusion of mucous membrane as the result of pressure. The reasons for excluding the possible congenital origin of this case are based upon the fact that the congenital varieties are chiefly found upon the anterior wall of the œsophagus and low down, whereas in this instance the pouch was situated high up and on the posterior wall. An excellent example of the congenital class is reported by Dr. Norman Moore in THE LANCET, vol. i., 1882, p. 482; and Mr. Howard Marsh and Dr. Ilott describe two cases in the Transactions of the Pathological Society, vol. xxvii. Hamburger, Rokitansky, and Ziegler also mention traction diverticula, which, although they regard of no practical importance, are found, they state, in the front of the œsophagus low down, and generally opposite the bifurcation of the trachea. These also may arise as the result of the traction of cicatricial tissue on the œsophagus—e.g., a bronchial gland becomes inflamed and adherent to the œsophagus, the inflammatory material cicatrises and contracts; by the contraction the œsophageal wall is drawn outwards into a small pouch. If we hazard a conjecture as to the development of an acquired diverticulum, we may assume some injury inflicted upon this particular portion of the œsophagus, which from its anatomical formation is most exposed to accident, atrophy of some muscular fibres occurs, and a weak spot is formed in the wall of the gullet which eventually leads to the formation of a small pouch, and this, by repeated distension, ultimately increases to the dimensions of a considerable sac. Once having increased beyond a certain size, other factors would naturally come into play, food would collect in the sac, and by sheer weight drag it further downwards behind the œsophagus, tending to make its axis directly continuous with that of the pharynx. The distended sac meeting with the resistance of the vertebral column would push forwards and compress the œsophagus and close the route to the stomach below the opening into the diverticulum, and at the same time keep open the mouth of the sac, and thus facilitate increased influx of food. There are a few other features of the case which appear to possess too much practical importance to be entirely neglected. There can be no doubt that gastrostomy saved and prolonged the patient's life. For two years the patient was nourished and her health maintained solely by alimentation conveyed by the gastric fistula. During this period the œsophagus had the advantage of absolute rest, and it would be difficult to find a better example of the power of rest as a therapeutic agent. The condition of the adhesion between the stomach and the abdominal wall six years after gastrostomy had been performed and four years after the gastric fistula had been made use of affords material for reflection. In six years the originally broad, firm adhesion between the stomach and abdominal wall had been converted into a long filamentous band

[1] Von Ziemssen's Cyclopædia, vol. viii., p. 51.

[2] Ibid. [3] Klinik der Œsophaguskrankheiten, p. 179.
[4] Lehrbuch der Pathologischen Anatomie, vol. iii., p. 126.
[5] Ziegler's Pathology, vol. ii., p. 258.

traversed by a narrow channel, illustrating how readily broad adhesions become in time attenuated into narrow bands. How far the disuse of the sinus had a tendency to induce the atrophy is difficult to estimate, but it would appear that in all cases of gastrostomy there are agencies at work which have naturally a tendency to weaken the adhesion between the stomach and the abdominal wall—the contraction of the abdominal muscles, the movements of the diaphragm depressing the stomach, and the distension of the colon raising it &c. There is another and final lesson to be learned from this case. In the events of its ever being necessary to dilate or enlarge a gastric opening of long standing, judging by this case, there would be considerable risk of opening the peritoneal cavity, and it almost imperatively suggests that under such circumstances it would be much safer to perform gastrostomy *de novo*, rather than attempt to utilise an old channel of such doubtful stability.

Manchester.

ON TESTS FOR LATENT GOUT.

BY DR. EMIL PFEIFFER,
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SIR WILLIAM ROBERTS, in a communication which appeared in THE LANCET of Jan. 4th, 1890, writes upon "Pfeiffer's Test for Latent Gout." I am extremely thankful that the eminent writer has occupied himself so thoroughly with the very interesting question of "free uric acid" and its bearing on the theory and practice of gout and urolithiasis, and especially that he has communicated my investigations as well as his own opinions regarding the subject to the English members of the profession so eminently interested in everything concerning gout and uric acid diathesis. As the result of my investigations leads me to the belief that the question of "free uric acid" in the urine is one of very great importance in the diagnosis and treatment of gout and urolithiasis, I deem it advisable to bring to the notice of the readers of THE LANCET a translation of the remarks which I have published in the "Verhandlungen des Congresses für innere Medicin (VIII. Congress, Wiesbaden, Bergmann, 1889)" on the same subject in June, 1889. My experiments and deductions therefrom are given as follows:—

"When the whole twenty-four hours' urine of a person is collected and two equal portions are separated from the whole quantity, to one of which is immediately added (the tenth part of its quantity) strong hydrochloric acid (of Heintz, sp. gr. 1·12), while to the other, previously passed through a filter on which some chemically pure uric acid has been placed, is also added the hydrochloric acid of Heintz, we find that after forty-eight hours, when we collect on separate filters and weigh the uric acid which has been deposited, the two portions have thrown down in many cases very different quantities of uric acid. In children, women, and old men you find as a rule equal quantities of uric acid thrown down, while in healthy male adults the portion passed through the uric acid is found to yield in most cases a considerably smaller quantity of uric acid than the portion that has not been in previous contact with it. As I have shown in former papers, this diminution of precipitated uric acid is caused by its being retained or abstracted by the uric acid placed on the filter. This abstraction of uric acid from the urine by the acid filter (*Harnsäurefilter*) takes place to the greatest extent in cases of patients suffering from uric acid diathesis—i.e., uric acid urolithiasis and gout.

"In cases of uric acid urolithiasis, when the patients are not yet in a cachectic state, we find as a rule that the whole of the uric acid is abstracted from the urine even by small quantities of uric acid—at least, as far as it can be thrown down by hydrochloric acid. When we pass in such cases 100 ccm. of the whole twenty-four hours' urine through from 0·2—0·5 grm. of uric acid, the filtrate mixed with the hydrochloric acid of Heintz yields no traces of uric acid. This examination is of great diagnostic importance, and one can in doubtful cases entirely exclude the presence of uric acid urolithiasis by the absence of this symptom; while, on the other hand, its presence proves the existence of this disorder. The same symptom is of almost equal importance in the diagnosis of gout; and this comes out very clearly in the case of gouty subjects, from whose urine the *Harnsäurefilter* abstracts the whole of the uric acid, when at the time of the examination they are not ailing, but present the appearance of perfect health. In clinical examinations of the abstraction of uric acid from the urine the following rules must, however, be observed:—Above all, the urine must be always a part of the whole passed in twenty-four hours. Single portions of urine collected at different times during the day may behave alike in gouty and in healthy cases. Of the whole twenty-four hours' urine three portions of 100 ccm. are separated, and the first and second portions passed through two filters, on one of which 0·5 grm. of chemically pure uric acid has been placed, on the other, 0·2 grm. When the filtrations are finished, the three portions are mixed with the hydrochloric acid of Heintz and put aside for forty-eight hours.

"A further rule is to make the examination only in the interval between the attacks, at a time when the gouty person feels quite well. Then it will be found that in gouty subjects—still liable to acute typical attacks—all, or almost all, uric acid is retained by the *Harnsäurefilter*, and that therefore the portions of the urine passed through it yield with hydrochloric acid no uric acid, or only traces of it. Numerous examinations of urine have shown that this symptom—viz., that on several consecutive days all the uric acid precipitable by hydrochloric acid is abstracted from 100 ccm. of the twenty-four hours' urine by 0·5 grm., or even by 0·2 grm., of uric acid—is only found in cases of uric acid diathesis—i.e., urolithiasis and gout; also that it is never found in cases of healthy persons or in patients suffering from other diseases. I think, therefore, that the following deduction may safely be made: When in the filtrates from the two *Harnsäurefilter* no trace, or at least no appreciable quantity, of uric acid is thrown down by hydrochloric acid, we may conclude in doubtful cases that we have to deal with gout, provided the patient has not been taking one of the drugs, such as salicylate of sodium (hydrochloric acid), which are found to produce a similar condition of urine. The rule to take two *Harnsäurefilter*, one with 0 5 grm. and one with 0·2 grm. of uric acid, is founded on the following fact. When we pass several portions of 100 ccm. of the twenty-four hours' urine of a healthy adult through increasing quantities of uric acid—viz., one portion through 0·2 grm., a second through 0·5 grm., a third through 1·0 grm., a fourth through 1·5 grm., and the remaining through 2·0 and 2·5 grm. respectively, we find in most cases, even in the urine of healthy persons, an upper limit, where the *Harnsäurefilter* abstracts from the 100 ccm. of urine all the uric acid capable of being precipitated by hydrochloric acid. It follows from this that the amount of uric acid abstracted from the urine varies, being in proportion to the quantity of uric acid previously placed on the filter; and it is only the great proneness of the urine of persons subject to gout to part with its uric acid which is characteristic of that disorder, and not the mere abstraction itself. While from 100 ccm. of the urine of healthy persons the uric acid is only abstracted by 2·0 or 3·0 grm. of uric acid, from the urine of gouty persons it is withdrawn by 0 2 or 0·5 grm. In order to estimate this proneness to deposition it is advisable to pass two portions of the urine through two different filters, containing 0·2 and 0·5 grm. respectively, for a urine that loses all its uric acid by passing through the 0·2 grm. filter, certainly contains it in a state more prone to precipitation than one which retains a certain portion and only parts with the whole of it to the 0 5 grm. filter. More than 0·5 grm. of uric acid should not be used, as larger quantities can abstract all the uric acid from the urine, even of healthy persons. But I have never found a case of a healthy person, or one suffering from a disease other than gout (with the exception above mentioned), in which 100 ccm. of the twenty-four hours' urine lost all its uric acid for several days in succession by passing it through the 0 5 grm. filter. I therefore reaffirm my contention, that the entire abstraction of the uric acid precipitable by strong hydrochloric acid from 100 ccm. of urine by 0·5 grm. of uric acid proves gout in doubtful cases; and this proof is still more certain when the same result is obtained by 0·2 grm. of uric acid.

The abstraction of all the uric acid from the urine of healthy persons by large quantities of uric acid has been proved by means of exact experiments made by Dr. W.