found a positive reaction after death. Ehrlich first noticed this *ante-mortem* fading of the reaction, but it has since been contradicted by some writers. That it really does fade is a fact. Those declaring the contrary have simply not met with the cases.

Petri considered a concentrated state of the urine to be necessary to the production of the characteristic positive tint. With a view to testing the accuracy of this observation, I have recorded the specific gravity of every case, and found that of the sum total of positive cases (including measles, typhoid, etc.) the average specific gravity was 1025. Only in 6 cases was it less than 1016; but in reference to this point we have to consider that the concentration of the urine is largely dependent upon the temperature of the patient previous to and at the time of voiding, and, in the case of phthisis, upon night sweats. Those with the highest temperatures and the most profuse sweats are generally the worst cases, and therefore the class in which we may expect the reaction. In the same way the reaction cannot be wholly dependent upon pyrexia, as some have affirmed, since it is often present in apyrexial cases.

I have further observed that those cases giving the reaction to the end have run a more rapid course, and have died in a less exhausted condition than those who had once given the reaction; and then had ceased to do so. This I believe to be the cause of the constant affirmation that a diazo patient had the worst outlook, and I believe that writers who have made this statement have not met with cases which ceased to yield the reaction a month or six weeks prior to death. There seems, in short, to be required a certain amount of vitality for the production of the characteristic aniline colour of Ehrlich's reaction. This is borne out in a remarkable way by the following case:

G. C., aged 20. Admitted to Walton Workhouse, December 16th, 1901. Temperature 97.6°, pulse 168, respirations 56. In extreme exhaustion. A large vomica at right apex. Diazo negative. Patient was so ill that no expectation of his surviving the night was felt. He rallied, however, and showed marked improvement the next and four following days. On December 21st, that is five days after admission, he gave a well-marked reaction. A day or two later he began to sink; his temperature, which had gone up to 100°, fell to subnormal again, the diazo reaction faded, and he died on the 29th of the same month.

We further have to record a very singular case in which the patient, although in the last stages of phthisis, failed to give the reaction. He was under observation for a month, during which time his urine was tested daily. On the last day of the month he died from hæmoptysis, and in idle curiosity I set the blood aside in a measure glass, decanted the serum and subjected it to the diazo test. To my astonishment it yielded a rich reaction. Encouraged by this event I repeated the experiment in 6 other cases of hæmoptysis, but did not again succeed in eliciting the reaction. I aspirated the heart immediately death occurred in a large number of cases, but never obtained a positive coloration. My attempts to get it in pleuritic effusions also failed.

CONCLUSIONS.

The conclusions I draw from this investigation are that the reaction is not of any value in the prognosis of chronic tuberculosis. It is certainly true that those patients who gave the reaction have nearly all died; it is certain that in most cases the course was rapid; it is also true that its appearance and disappearance corresponded to the exacerbations and to improvement; it is also a fact that those not giving the reaction are patients who have shown improvement in their clinical course. This, to a large extent, agrees with what has been before written. Were this all, such facts would indeed be significant, but they reveal only half the truth. The positive cases have died, but they have been precisely those cases for whom no hope could ever be entertained; they have run, many of them, a rapid course, but an equal number have run a slow course and even improved; its appearance coincides with the exacerbations of the tuberculous process, but it follows the onset of the graver symptoms, and though it disappears with improvement, such disappearance again follows the suspension of the alarming signs; while, again, it may disappear because the patient is growing weaker. A reaction blowing hot and cold in this manner cannot be of very great reliability. Lastly, patients who have not given the reaction have mostly been in the early stages when hope is justifiable, while if it is absent in the later stages it means nothing, since its appearance is intermittent: is present one week, absent the next.

It is probable that every fatal case of tuberculosis will yield the reaction at some period of its course. In view of the

frequency of the intermissions, we cannot say that any case is negative unless it has been tested constantly for long periods. Of the 17 cases which were negative and yet died, 6 succumbed to intercurrent diseases. Of the remainder, 2 were admitted *in extremis*, when the reaction is known to fade, so that it is not an unjustifiable conclusion to suppose that the other 9 were tested during an intermission.

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TETANY AS A COMPLICATION OF GASTRIC DILATATION,

WITH NOTES OF A CASE.*

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THERE are few subjects in the whole range of medical literature round which so much controversy has raged as that of tetany. Its first description, in association with gastric dilatation, is attributed by some to Newman¹ in 1861, by others to Steinheim.² Its etiology was the despair of theorists and experimental workers alike, Maestro³ regarding it as an "essential neurosis," Oddo⁴ as "a toxæmia produced in the presence of a hypersecretion of hydrochloric acid and gastric juice," while Berliozheimer⁵ cites a case in which no ptomaines could be found.

Its pathology was for years the will-o'-the-wisp of the men of the microscope and of the morbid specimen, but to their credit be it said they promise fair to be the guides out of the present morass. Its treatment has been the shuttlecock of the battledores of the physician and surgeon, the former of whom have tried everything, from calomel to the latest antiseptic, while the latter have performed operations as varied in intensity as gastric lavage, puncture of the spinal cord, and gastro-enterostomy.

I shall leave, however, the domain of controversy and speculation, and pass on to the clinical notes of a case, and subsequently make some observations on them and on tetany in general.

Personal History.

R. H. S., aged 45, married, by occupation a commercial traveller for a distillery, but, nevertheless, very moderate in his use of alcoholic liquors. Of an active, nervous temperament, slightly built, and rather short of stature. For years he had complained of attacks of indigestion, which were the more remarkable to him because of the enormous quantities of food and fluid he was capable of taking at one meal. Pain ensued during these attacks about an hour after eating, and was only relieved by vomiting, which he sometimes produced artificially, in quantities as large as a chamberful, often wondering "where it had all come from," and being astonished to see in it at times food he must have eaten several days previously—for example, barley groats.

History of the Present Illness.

First Day.—I was called to see the patient first about 8 p.m. on Thursday, September 8th, 1904, and on arrival was informed that he had "had a fit" a short time previously, in which the upper arms were pressed to the sides, the forearms bent at the elbows, the fingers bent at the knuckles, with the thumbs inside the palms. The feet were also contracted, and there were twitchings about the mouth. During the entire attack he was quite conscious—indeed, rather alarmed at the appearance of his hands, exclaiming, quite excitedly, as if watching something entirely outside himself, "Look! Look at my hands!" Bathing in hot water had relieved the contracture and the pain. He was very flushed and sweating when I entered, sitting propped up in bed, carefully holding his hands under the bedclothes lest another

* Read before the Spring meeting of the Belfast Branch of the British Medical Association.

attack should develop. On questioning him he stated that prior to the "fit" he had felt his feet and hands "prinkling." The temperature was normal. Pulse regular and strong, 78 per minute. The tongue was clean, and the bowels had been moved the day previously. He was now complaining of nothing except pains in his loins and legs.

Second Day.—At 4 a.m. he had another similar, but shorter paroxysm, which was again relieved by hot water bathing. Temperature normal, pulse 78. During the day he had another slight seizure. I examined the urine for albumen, with negative results. He was rather excited in conversation, and complained of want of sleep which he said he had suffered from for four nights. Accordingly I prescribed: Pot. brom. gr. xv, chloral hyd. gr. v, in a mixture to be given every hour till five such doses were taken if required. This had little effect, producing only slight snatches of sleep, from which he waked up very excitedly. Hiccough occurred at intervals.

Third Day.—No further paroxysm. Temperature normal, pulse 80. Delirium deepening, with muttering incoherency. Hiccough almost constant.

Fourth Day.—Sir Wm. Whittall was called in consultation, and mapped out the stomach, the lower curvature reaching below the umbilicus. Pulse 78, temperature normal. Conscious, but incoherent. Hiccough constant. Recognizing the necessity of getting rid of any toxin which might have been accumulating in the stomach, but fearing lest the introduction of a stomach tube might precipitate another paroxysm, it was decided to give him water freely in order to wash out his stomach automatically by emesis. Accordingly pint after pint was given (measured) till six were swallowed, and, as no vomiting occurred, a pint of mustard and warm water was administered—7 pints in all at one time, without result.

Fifth Day.—Muttering delirium and hiccough continued; morning temperature normal, evening ditto 98.8°. Having given a dose of Purgin, which cleared the bowels of a copious dark green motion, I ordered enemata of glyco-thymoline and bovine to be given at 4-hour intervals, as it was evident nothing was being absorbed by the stomach.

Sixth Day.—No further paroxysm; muttering delirium and hiccough continued. Evening temperature 99°; pulse 88.

Seventh Day.—*Death.*—Vomited at 4 a.m., after which for half an hour he rallied somewhat, but gradually lapsed into his former state. There was a right external strabismus, but no inequality of the pupils. Gradually he sank into a comatose condition, and died at 4.30 p.m. without regaining consciousness. Rigor mortis set in less than two hours. Trousseau's sign was only slightly present. Chvostek's sign was absent.

In two particulars this case differed from any other of which I can find any record:

1. Hiccough, which was persistent and constant towards the end, resisting all treatment.
2. (Which I regard as being by far the most important) The peculiar mental condition associated with the case. When the first paroxysm passed off his mind was perfectly clear, though a little excited, but gradually he became more and more delirious and incoherent until the stage of complete coma was reached on the seventh day when he died unconscious.

Regarding Trousseau's sign (in itself a monumental witness to his keen and careful powers of observation), this seems to be regarded more as a curious phenomenon than as of any practical utility; and tested after it is definitely known that a paroxysm of tetany has occurred, it is like a man who consults the weather-glass merely to corroborate the fact that rain has just fallen. I suggest, then, that if the sign was sought for in cases where tetany might reasonably be feared (not in dilatation of the stomach merely, but in rickets, etc.), it might prove a valuable diagnostic token, as indicating a coming storm, rather than a confirmation of the storm that has passed.

That tetany as a complication of gastric dilatation is comparatively rare is admitted by all observers; thus Berlitzheimer⁶ believed in 1897 that only 11 real cases had been recorded. But Sievers⁷ in 1898 gave an account of 27 fatal cases, while Parsons⁸ says no case had been reported in Ireland up to 1894. Since then, however, the recorded cases have considerably increased, and it may be fairly assumed that cases were rarer in point of observation than in point of fact. Further, they seem to be almost as fatal as they are rare. Soltan Fenwick⁹ collected a series of 26 cases in which the mortality was 70 per cent., which is, perhaps, below the general average.

Regarding its etiology, the two main theories which obtain most acceptance are, that it is due to (1) toxæmia or to (2) reflex irritation. But neither of these two theories will alone explain the phenomena, while both together would seem to satisfy all requirements.

As indicating an auto-intoxication, with the origin of the toxin in the alimentary canal, it is a significant fact that nearly every case recorded (including ordinary tetany associated with rickets, etc.) has had some gastro-intestinal disorder. This was pointed out by Hauser,¹⁰ but long previously—in 1875—by Haddon¹¹ in a paper read before the annual

meeting of the Association. Bouveret and Devic found that if hydrochloric acid was allowed to act on syntonin, and alcohol on the resulting product, and this final product introduced into the stomachs of animals, symptoms were produced in all respects like tetany. Further, in the case of which I have given notes, the peculiar mental condition seems to point unmistakably to auto-intoxication.

Oddo and Sarles, and Ewald¹² have found indican in the urine in some of these cases, which is formed by fermentation set up in the gastro-intestinal tract.

Lastly, it seems a significant fact, as first pointed out by Hauser¹³ in an article on tetany in children, that "there appeared to be a connexion between the appearance of gastro-intestinal symptoms and that of tetany."

For the theory of reflex irritation as a cause much might be said, but the question at once arises, Why does the nerve irritation commence in the hands and feet, and not produce general convulsions, as it does, for example, in the teething of childhood? This would be satisfactorily answered by presupposing as a cause some form of toxæmia which had a selective affinity or irritative effect on the delicate sensory nerve endings of the hands and feet, for it must be borne in mind that the first symptoms of the paroxysm is a tingling sensation in the extremities. As supporting this, it may be mentioned that Ross and Bury¹⁴ believe that a neuritis may be induced by auto-intoxication from ptomaines.

Weiss in adults, and Bonome and Cervesato¹⁵ in children, have demonstrated the fact that in severe cases, inflammatory and degenerative changes take place in the anterior cornua of the spinal cord—a condition which was shrewdly suspected by Parsons¹⁶ in 1894, because of the muscular atrophy which sometimes sets in, in tetany of children.

As supporting the combined theory of toxæmia and reflex irritation, the remarkable clinical resemblance which exists between strychnine poisoning and tetany is of deep significance. In the former there is a well-recognized poison at work, and we know its mode of action thoroughly. A poison is introduced into the system with a strong affinity for nerve tissue, analogous to the toxin of tetany. After an interval more or less prolonged, and generally excited by some irritation, a tonic spasm occurs, which passes away for a time, only to again recur upon some fresh irritation. In this, tetany maintains its clinical resemblance to strychnine poisoning, since in nearly every recorded case, vomiting or some other irritation has preceded the paroxysm.

The pathology of tetany in gastric dilatation might be pictured thus: The stomach, narrowed at its pyloric end by the cicatrization of an old ulcer, is gradually dilated into a mere bag, with scarcely any digestive power, and thus becomes a suitable nidus for fermentative toxins which gradually become absorbed into the system, producing a condition of nervous irritability both directly and reflexly—directly by acting on the delicate sensory nerve ends of the extremities, and reflexly as a result of this on the motor nerve cells in the cord producing the characteristic spasm of the extremities, upon any determining cause sufficient to upset the unstable equilibrium of the nervous system.

As to treatment, lavage has fallen into disrepute because this operation has sometimes precipitated an attack. Oddo¹⁷ recommends calomel as a purge every other day and benzonaphthol in $\frac{1}{2}$ gr. doses every two hours as an intestinal antiseptic. If the patient survives the attack, Mayo Robson¹⁸ strongly recommends operation for the relief of the pyloric stenosis, which is the primary cause of the disease, and is present in about 92 per cent. of these cases. This is certainly the only treatment which has up to the present given any hopeful results, and has the further advantage of curing the attendant constantly-recurring and sometimes ever-present indigestion.

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THE annual meeting of the Asylum Workers' Association will be held at 20, Hanover Square, W., on Friday, May 19th, at 4 p.m., under the presidency of Sir John Batty Tuke, M.P.