of a rest cure, the stomach remained in practically the same position as before.

As regards the development of the abdominal muscles, reliance must be placed upon massage and the practice of "abdominal exercises." Electrical treatment may undoubtedly be of help here, but its results are probably inferior in value to the spontaneous muscular contractions which exercises provoke. Hydrotherapeutic measures—for example, the Scottish douche—are also of service in increasing the tone not only of the abdominal wall, but also, in all probability, that of the blood vessels of the splanchnic area as well. The question of artificial substitutes for the abdominal wall will always arise.

The argument sometimes used against them—especially by masseurs—that they demoralize the abdominal muscles by doing their work for them, is, I believe, fallacious. On the contrary, by preventing over-stretching of the weakened abdominal wall, they are undoubtedly of service, pending the restoration of the natural means of support. They are therefore of great temporary utility, at least. It would be going too far into details to consider the relative merits of the different forms of artificial support which have been devised, but there is certainly no lack of choice—from the simple plaster strapping of Achilles Rose to the latest belt-corset of the artist in orthopaedic appliances. I think it will be generally agreed, however, that the ideal abdominal belt remains yet to be invented. It is simple enough to point out what is wanted—the exercise of pressure from the lower part of the abdomen upwards and somewhat backwards, but the difficulty is to carry this principle into practice. In cases of "pendulous belly" in stout subjects it is quite simple, but most patients with gastroptosis who require treatment are, *ex hypothesi*, thin, with prominent iliac bones which tend to carry off the pressure from the hypogastric region where we most wish to apply it. The problem is a purely mechanical one, and the use of a suitable pad helps to overcome it, but the results often leave much to be desired.

Having by the adoption of the above means restored the patient to a condition of comparative well-being, the next problem is how to keep him so. It is here that our real difficulties begin, for the neurasthenic state, which, as has been repeatedly pointed out, underlies the gastroptosis in the great majority of instances, is due either to an inborn feebleness of constitution or to unfavourable factors in the environment, of which the former is wholly and the latter often very largely beyond our power to alter. Much may be done, however, by suitable regulation of the patient's life and habits. Overstrain—mental, physical, and emotional—must be avoided so far as is possible. The importance of rest must be enjoined, particularly rest after meals, which greatly facilitates the labours of the stomach. Frequent holidays are advantageous. The diet must be so regulated as to contain a maximum of nutriment in a minimum of bulk, and all voluminous and innutritious foods forbidden. It is particularly important, as in all cases of gastric atony, that the meals should be dry. Meanwhile, the further development of the abdominal wall must be persevered with by the persistent practice of abdominal exercises, coupled, perhaps, with the use of auto-massage, and, until a sufficient degree of natural support has been attained, the belt must not be left off.

The use of drugs may be confined to the occasional administration of bromides, or of such as are required for the relief of any particular symptoms. Suitable aperients may be necessary to supplement the use of enemata.

In those cases in which the carrying out of a thorough rest cure is impossible, the above means may be used as best calculated to palliate the symptoms, but it will be readily understood that a patient with gastroptosis whose symptoms have been sufficiently severe to call for treatment is rarely able to return to a condition of full activity. He must walk softly and be sparing of his nervous output, as otherwise a relapse is almost certain to ensue. It would not be an exaggeration, indeed, to say that his whole life must partake of the nature of a more or less modified rest-cure.

Up to this point we have been concerned with the things to be done in the treatment of gastroptosis; may I conclude by enumerating three things which I think should not be done? First, patients with gastroptosis should not be overdieted—which, of course, is just the

reverse of being overfed. Secondly, their stomachs should not be washed out. Thirdly—and *pace* Mr. Eve—they should not be operated upon. They should not be overdieted, because if that is done their nutrition is apt to suffer, and the maintenance of a high degree of nutrition is the first essential of successful treatment. They should not be washed out, because there is no real gastric stagnation, and the introduction of large quantities of fluid is apt to overdistend the already atonic stomach. They should not be operated upon, because the mere replacement of the stomach, even if this be permanently possible by operation, is treating a symptom and not the real disease, and because most patients with gastroptosis are neurasthenic, and no operation on a neurasthenic subject is advisable without very good reason.

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A CASE OF CEREBRO-SPINAL RHINORRHOEA WITH DOUBLE OPTIC ATROPHY.

By NORMAN H. PIKE, M.B., B.S. LOND.,

SURGEON FOR THE EYE, EAR, NOSE, AND THROAT, THE GENERAL
HOSPITAL, CHELTENHAM.

AN undoubted case of the spontaneous escape of cerebro-spinal fluid from the nose, known as "cerebro-spinal rhinorrhoea," is of sufficient interest to record.

A woman aged 22 first attended among my out-patients on June 1st, 1909, complaining of a constant watery discharge from her right nostril.

History.

Up to the age of 12 she was a strong, healthy girl, and could see well. From 12 to 13 she had a severe illness, keeping her bed for nearly twelve months. The chief symptoms were severe headache, vomiting, drowsiness, frequent convulsions in which she was unconscious, and stiffness at the back of the neck. The eyes became prominent, and at the end of the illness she was quite blind.

After getting up she continued to have severe attacks of pain in the head, and fits of an epileptiform character; they were often nocturnal, and occurred about every three weeks to one month, but in the year 1908 they were less frequent—about every three months. Mr. Arthur Cardew, who attended her during her illness, told me that there was some pyrexia for the first few days, but that afterwards the illness ran an apyrexial course.

Family History.

The only interesting fact about the family history that I have discovered is that her younger sister has suspiciously notched incisor teeth and a few scars at the angles of the mouth. Her general expression, too, suggests congenital syphilis.

Present Condition.

She is a moderately well developed girl with a prominent head, measuring round the forehead 55½ cm. Mentally she is dull; her speech is slow and the voice has a monotonous character. She is quite blind and has no perception of light. There is no ocular paralysis, but marked nystagmoid movements of the eyeballs of an ocular character. The pupils are equal, moderately dilated, and react slightly to a strong light. Both discs are white and have slightly irregular edges. The lamina cribrosa is indistinct and the vessels are tortuous, the condition being one of double post-neuritic atrophy.

The nose is somewhat swollen externally at the tip. By anterior rhinoscopy nothing abnormal is seen, except that on the right side, descending between the septum and middle-turbinal and extending into the inferior meatus, there is an oedematous polypoid tumour. From this right nostril there is a constant dripping of a clear watery fluid. There is no discharge from the left nostril. With the patient in the Killian position the fluid is seen to collect between the middle turbinal and the septum, and the tumour has probably been formed by the continual soaking of the spongy tissues. There is no supuration in the nose and no signs of any sinus trouble. Transillumination shows good infraorbital reflexes on both sides.

During the patient's attendances at the hospital the following facts were ascertained about this fluid:

1. It was always from the right side, and was a constant drip, as in epistaxis.
2. It dropped at the rate of from 3 to 8 drops a minute, and was measured on several occasions, the amounts varying from 5vj to 3j in an hour and a half—that is, from 12 to 16 oz. in twenty-four hours. It drops faster with the head forward.
3. It was ascertained from the mother that when asleep she makes frequent swallowing movements, the fluid apparently running into the naso-pharynx.
4. She uses many handkerchiefs a day, and these when dried are found to be quite pliable and not at all stiff.
5. Cocaine and adrenalin applied had no effect on the flow.
6. The fluid has never ceased during her attendances, and has now been constant for over nine months. Since its advent the

head pains have been less and she has only on one occasion (February 7th, 1910) had a fit, after which she was unconscious for about two hours.

Treatment.

I removed the polypoid tumour, but this has since re-formed; apart from this there has been no intranasal treatment. Potassium bromide has been given when the head pains were bad.

Report on Fluid.

Dr. Rupert Collins, honorary pathologist to the hospital, very kindly examined several specimens of the fluid, and reported as under:

"Various specimens from the nasal discharge of K. P. have been examined by me at different periods.

"The first specimens contained a considerable amount of mucin, as shown by a precipitate with acetic acid in the cold, and gave no reduction with Fehling's solution. When centrifugalized a deposit of leucocytes and nasal epithelium was obtained. All this pointed to the condition being a nasal hydropyorrhea. Later specimens were different, sometimes clear, sometimes turbid a few hours after being passed owing to the enormous number of micro-organisms present.

"The analysis of the clear specimens was as follows: Acetic acid in the cold yielded no precipitate whatever, showing absence of mucin. Heating and acidulating with acetic acid gave a precipitate showing the presence of proteid. If the fluid was saturated with $MgSO_4$ and filtered it no longer gave any reaction for proteid, showing that the proteid present was a globulin. The fluid reduced Fehling's solution, and did not ferment with yeast. I twice tried to obtain crystals of pyrocatechin by evaporating and extracting with alcohol, but failed to do so.

"The fluid was quite clear, like water, specific gravity 1006, reaction alkaline. On centrifugalization a few leucocytes only. It was rich in chlorides, and contained a trace of sulphates. Two cultures were taken by letting the fluid drip straight into culture tubes of agar-agar, and both grew cocci and bacilli luxuriously. The fluid was always collected by letting it drip into a sterile flask, but it invariably was turbid with micro-organisms in six hours, and had accordingly to be examined at once. I found the fluid to be turbid on two occasions as it dripped from the nose when I was present.

"I conclude that this is a case of cerebral rhinorrhoea with a septic condition of the mucous membrane of the nose which caused the presence at intervals of mucin in the fluid. Why the fluid did not reduce Fehling at first I do not know; there was no difficulty in getting plentiful reduction in later specimens, and I can only conclude pyrocatechin was not present in the first two specimens tested."

I also failed to obtain a reduction of Fehling's solution from the first specimens, but later obtained a definite reduction. With regard to the turbidity, I found that the fluid collected shortly after the use of an antiseptic nasal douche remained clear for several days.

It is due to the excellent monograph, *The Cerebro-spinal Fluid, its Spontaneous Escape from the Nose*, published in 1899 by StClair Thomson, that the true nature of these cases has been recognized. He records one case of his own and twenty others collected from the literature both English and foreign.

He divides them into 9 undoubted and 12 probable cases. In 17 there were cerebral symptoms, and in 8 eye symptoms. The flow was from the left side in 10 cases, right side in 4, both sides in 1; the side was not mentioned in 5.

The cerebral symptoms presented a great similarity in most of the cases, the most marked being severe headache, drowsiness, vomiting, delirium, convulsions, and coma. A striking point was that in all the cases there was an absence of fever, and that the headache and other cerebral symptoms were relieved by the flow.

In these respects my case agrees, also in the eye symptoms, there being always either a neuritis or a post-neuritic atrophy. The pathology suggested by StClair Thomson is a meningitis serosa, giving rise to idiopathic internal hydrocephalus, optic atrophy being due to a pressure on the optic chiasma, caused by the dilated third ventricle.

The condition with which "cerebro-spinal rhinorrhoea" will be most easily confounded is that known as "nasal hydropyorrhea." But there are such marked clinical differences in the two conditions that I felt from the first that I was dealing with a true case of cerebro-spinal rhinorrhoea, in spite of Dr. Collins's first reports on the analysis of the fluid, and so, at my request, he very kindly examined further specimens, and our perseverance was rewarded by finding the Fehling-reducing substance in the fluid.

The true nature of the condition is established by the analysis of the fluid, the main characteristics being that cerebro-spinal fluid is a perfectly clear, transparent, tasteless fluid, with no sediment. It has a low specific gravity,

contains no mucin, but a trace of globulin, and has a substance which reduces Fehling's solution, but does not ferment with yeast and therefore is not a true sugar. This reducing substance can be obtained in a crystalline form resembling pyrocatechin.

I would suggest that in some of the cases classed by StClair Thomson as "probable," if a repeated examination of the fluid had been made, this sugar-reducing substance would have been eventually found. Notably in two cases recorded by Priestley Smith in the *Ophthalmic Review*, 1883, both with double optic atrophy, and in neither of which was a reducing substance found. In both of these cases StClair Thomson comments that it is "difficult to lay aside the hypothesis of cerebro-spinal fluid, but not tenable in view of analysis."

Anyway my case is of special interest from the fact that some specimens did not show the reducing substance, while others did. One must remember that we are dealing with a diseased condition which may alter the character of the cerebro-spinal fluid; in general paralysis of the insane there is no reducing substance.

The case was shown at a meeting of the Gloucester Branch of the British Medical Association in October, 1909.

THE DIAGNOSTIC VALUE OF THE LEUCOCYTOSIS OCCURRING IN WHOOPING-COUGH.

BY

HUGH T. ASHBY, B.A., M.B.CAMB., M.R.C.P.LOND.

The following observations on the blood of children suffering from whooping-cough during an epidemic were made in the early part of this year in the out-patient department of the Manchester Children's Hospital. The blood from nearly 100 cases was examined, and a differential count of the white blood cells made in each case. The age of the children varied from about 9 months to 8 or 9 years. The blood was taken from cases in various stages of the disease so far as possible, and the blood of the same case was examined repeatedly during the progress of the disease and during the convalescence. The number of red blood cells and the haemoglobin percentage were estimated occasionally, but yielded no results of special significance; patients suffered from no more than a "slight anaemia." The blood was taken from the ear and was stained by Leishman's method or by eosin and methylene blue after fixing in formalin vapour.

In a fair proportion of cases the number of leucocytes per c.mm. was estimated, and in every case where this was done there was a leucocytosis of between 15,000 and 30,000 white blood cells per c.mm. The number of white blood cells in children suffering from bronchitis and other febrile chest cases was also estimated, but in only rare instances did the numbers reach such a height as that observed in whooping-cough. The results, therefore, confirm those made by Fröblich¹ in 1897, who examined the blood of 55 children suffering from whooping-cough, and found a leucocytosis varying from 7,000 to 40,000 white blood cells per c.mm. This leucocytosis in itself is a valuable aid in the diagnosis of a suspicious case of whooping-cough. In examining the blood of a child suffering from whooping-cough the preponderance of lymphocytes over the polymorphonuclear cells is at once noticed, and in nearly every case which had a well-marked whoop the lymphocytes formed about 60 per cent. of the total white blood cells, the rest of the white cells being made up of polymorphonuclear cells with an occasional eosinophile cell. The excessive lymphocytes were nearly all of the small variety, but there was at the same time a greater proportion of large lymphocytes than is found per c.mm. in normal blood, and in some cases these cells amounted to 10 per cent. of the lymphocytes present. This excess of lymphocytes was found in cases which had not reached the spasmodic or whooping stage of the disease, and also in mild cases, when it was difficult to make a definite diagnosis from the history the parent gave.

An interesting example of this happened when I was examining the blood of a child aged $3\frac{1}{2}$ years, who had