

Effects on Chorea.—After each bout of pyrexia there was a definite decrease in the spontaneous movements and an increased facility in performing voluntary movements. It is noteworthy that this improvement did not reach its maximum till the second day after the pyrexia. During the ten days' interval between the second and third injections not only was there no further improvement after the second day, but towards the end of this period a slight relapse set in. This was probably attributable to a visit of the child's parents. Two days after the final injection involuntary spontaneous movements had disappeared, except for an occasional twitching of the angles of the mouth, and of the tongue when protruded. Control of voluntary movements was normal. The child could write legibly, and do up buttons with one hand. The effect of the treatment on the paresis was much less obvious than on the movements. There was still weakness of the legs, and the knee-jerks were still absent.

DISCUSSION

In my opinion there is no doubt that, in the above case, the duration of chorea was cut short by induced pyrexia. The attack had continued for six months without showing any signs of abatement, although the child was in bed all this time. It might be suggested that the cure was due to some special factor or factors in the environment of an open-air sanatorium, but the absence of improvement, either before the treatment or between the second and third bouts, makes this hypothesis untenable. Further, to those who would say that so drastic a treatment is unjustifiable in a disease recognized to be self-limited, the author would point out that this child had previously had an attack of chorea which lasted for ten months, although it was stated to have been milder than the present one. The present attack was almost completely cured in three weeks from the start of the treatment; and this period could presumably have been made shorter still by reducing the intervals between the injections of vaccine. Relief from so many months' recumbency was surely an end worth achieving.

When we consider the place of pyrexia in the treatment of chorea in general the question becomes a very different one. Cases as persistent as the one described are the exception, and in many instances the disease clears up after a few weeks of well-managed institutional treatment. Further, it is not always the severest cases that take the longest time to get better. The method has definite disadvantages, of which the most obvious is the discomfort it causes the patient. In the case described this amounted to considerable suffering. Also, there is certainly a risk, though only slight, of the temperature becoming so high that the child's life is endangered. With regard to the heart, there are several points to be considered. In the present case, specially chosen with early cardiac involvement, the pyrexia does not appear to have given rise to any permanent extension of the lesion. The fact that slight dilatation did occur, however, would make one chary of applying the treatment to any case with well-marked valvular disease. Another question is whether a long period of rest in bed is not advisable for every case of chorea, irrespective of the duration of the movements. Besides helping to arrest the progress of endocarditis already present, might not this rest prevent the appearance of the cardiac murmur which is so frequently discovered after the chorea has subsided? In our present state of ignorance of the pathology of rheumatic carditis and its relation to chorea, the practice described by Dr. Bateman of discharging cases of chorea after three weeks in hospital would seem to involve considerable risk. On the other hand, if these cases are to be kept recumbent for a long time after the movements have ceased the value of the treatment seems doubtful.

CONCLUSIONS

Although it is unjustifiable to dogmatize from a single case, the author would tentatively suggest the following

types of chorea as suitable for treatment by induced pyrexia.

1. Cases with no signs of cardiac rheumatism which show little improvement after four weeks in bed in an institution.

2. Cases with slight cardiac rheumatism in which a period of rest longer than four weeks in proportion to the severity of the heart lesion has produced little improvement.

3. Cases of choreic relapse in which the previous attack or attacks have been of long duration (the heart again being only slightly affected).

The treatment would appear to be unsuitable for cases with extensive valvular disease, especially if active rheumatic carditis or any degree of cardiac failure be present.

With regard to the technique, I would like to suggest the following modifications of the method described by Dr. Bateman. (1) The principle of doubling the dose of vaccine for each new injection may lead to dangerous reactions in some cases. If only one and a half times the first dose is given for the second injection some idea can be obtained from the reaction to this as to how easily the patient acquires immunity to the vaccine, and the rate of increasing the doses may be gauged from this. (2) By spacing the injections with a day's interval between each, instead of giving them daily, it should be easier to assess the degree of improvement obtained after each one; and possibly a smaller total number of injections would prove necessary. For those who do not approve of Dr. Bateman's practice of getting the patients up after two weeks in bed a slight increase in the total duration of the treatment would not matter. With regard to the importance of careful observation and attention to the patient during the course of the pyrexia I am in complete agreement with Dr. Bateman, and would like to re-emphasize this point.

I wish to thank Dr. A. G. L. Reade, medical superintendent of High Wood Hospital, for his consent to the publication of this case.

"BORNHOLM" DISEASE

ACCOUNT OF A YORKSHIRE OUTBREAK

BY

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The following is an account of an acute specific infectious disorder of definite and characteristic syndrome, which hitherto has apparently escaped recognition in this country, though it is often recorded elsewhere. As the symptoms, though of brief duration, are initially alarming and perplexing, this paper is written in the belief that it will aid the general practitioner, to whose purview the disease is likely to be confined, in recognizing cases and according them formal diagnosis.

In the early morning of July 24th, 1933, I was called to a farmhouse to see a small boy, 2½ years of age, who had been quite well and lively whilst being dressed, but was then suddenly attacked with pain in the upper abdomen, sweated profusely, and was thought to have had a fit. When he was seen the pain was not so acute, but he seemed limp and ill, and his temperature was 96° F. His respirations were rapid, and appeared to be painful, and he resented palpation of his abdomen. My partner, Dr. Dean Dunbar, saw him shortly afterwards in a return of the acute symptoms, and suggested that the condition was a painful spasm of the diaphragm. We saw him together at 3 p.m. His face was then flushed, his temperature was 101°, and his respirations were 60 per minute; his *alae nasi* were working, and he had an expiratory grunt and a short cough. Previous to this the case was frankly undiagnosed. The possibility of the ingestion of some poisonous substance, or of an acute abdominal condition, was suggested, but the absence of vomiting

made either unlikely. Now, however—quite justifiably, as I think—we considered the condition a commencing acute pneumonia. At 6 p.m. he seemed better; his temperature was 100°, his respirations were 48, and he had no pain. The next morning he was standing on the window sill, and greeted me cheerily as I came up the garden path. His temperature was 98°, and he seemed normal in every respect. On the 26th he had a return of the symptoms of two days before, with the addition of a pain in the back, which prevented him raising himself from the bed. On the 27th he had violent nasal catarrh, and his evening temperature was 99.8°, but after this date he recovered completely.

In addition to this patient the household consisted of his father and mother and his three brothers, two of whom, aged 4 and 6, were attacked respectively on the 25th and 26th, and two little girls, contacts, aged 5 and 7, on the 27th. It would be tedious to describe all these cases, as they differed very little from the first, but the following is a summary of the symptoms.

1. Onset with very acute upper abdominal pain, which occurred in spasms, and was accompanied by profuse sweating. All the children placed their hands with the centre of the palm over the xiphisternum when asked to indicate the site.

2. Rapid, shallow respiration, obviously extremely painful. Pain on yawning and deep breathing between the spasms. No abnormal signs in the chest.

3. Fever up to 103°, rapidly subsiding, but tending to rise again with a return of the pain on the third day.

4. Absence of vomiting or diarrhoea. Tongues coated, three being of the white strawberry variety.

5. No faucial injection, but nasal catarrh in one child and unilateral conjunctivitis in another.

6. No rashes.

7. Complete recovery in from four to six days, and an absence of sequelae.

On August 3rd the father of the boys, a man of 33, had a sharp pain in his left shoulder at 9.30 p.m. After a good night he awoke with pain at his costal margins and down his arms, but was able to work. In the evening he had a profuse sweat, and the pain disappeared. He had no pain during the next day, the 5th, but in the afternoon of the 6th it returned in the lower ribs, being especially noticeable on deep breathing. He had a very bad night, breathing being rapid at intervals, and accompanied by pain in the epigastrium and lower ribs. On the 7th he was still feeling dizzy and ill, and had definite tenderness over both costal margins. His temperature was normal, although he stated that during the night he was feeling "burning hot" and had an intense headache.

The probable infecting case was a small girl, who visited the family of boys on July 20th, and who spent the afternoon lying on the sofa and crying with pain in her abdomen, but was said to have recovered on the following day. This child came from the York neighbourhood, but all inquiries have failed to elicit information of similar cases. An interval of four days separated this case from that of the first small boy, which suggests a short incubation period.

COMMENTARY

It was clear that we were dealing with an infectious malady of which we had had no previous experience, and which was identical with one described in Scandinavian countries and named after the island of Bornholm in the Baltic, a frequent nidus of the disease. Articles by Dr. Ejnar Sylvest¹ (Copenhagen) gave me the clue to its identification. He considers that in these cases there is an infiltration of the muscles, and names the disease "myositis acuta epidemica." He states that it is more prevalent in the summer and autumn, and estimates that as many as 3,000 cases occurred in Copenhagen during 1930. Looking up his references I find that the disease was first described in the United States in 1888 by Dabney,² who suggested rather tentatively that it was a form of dengue, and stated that its local name was "devil's grip." In 1923 references were made to the same disease by Payne and Armstrong,³ who named it "epidemic transient diaphragmatic spasm"—a very good descriptive title—and by Hangar, McCoy, and Frantz,⁴

Torrey,⁵ and Greene.⁶ In 1924 three epidemics of a somewhat similar nature, but accompanied by a pleuritic rub, were recorded as "epidemic pleurisy" by Bruce Williamson,⁷ Lloyd,⁸ and Attlee, Amsler, and Beaumont.⁹ Beyond these no references have been found in English medical publications.

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PRIMARY THROMBOSIS OF THE SUBCLAVIAN VEIN

BY

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Mr. J. Cosbie Ross's article in the *British Medical Journal* of September 16th gives a résumé of the characteristics of primary axillary vein thrombosis, and usefully summarizes the theories which have been propounded as to its causation.

The following notes are on two cases I have seen of primary thrombosis of the subclavian vein, and possibly of that part of the axillary vein which is impalpable. I have seen or heard of similar cases only amongst undergraduates who row, although the proportion of my patients who engage in other forms of athletics has been larger than that of rowing men.

CASE I

A healthy-looking and powerfully built undergraduate of about 13 stone, aged 19, in his second year of rowing, came to see me because his friends in the boathouse had for some days been "ragging" him that his "right arm was growing as big as his thigh"; an observation which might have been disturbing, had not the absence of pain or other symptom for a time permitted a not unnatural pride in what he took to be a further development of his bulging musculature. However, the phenomenon soon began to call for explanation, which I was asked to provide. There was uniform and considerable swelling from fingers to shoulder, slight cyanosis of skin, but no pitting or tenderness on pressure. The radial pulse was good. Superficial veins were prominent over the right pectoral region and axillary chest wall. No thrombosed vein could be palpated, nothing abnormal could be felt or seen in the axilla, and x-ray examination was negative. There was no evidence of any associated pathological condition and no constitutional disturbance. I reported the condition to the boy's parents, who took him to London for further opinion, but beyond hearing that he had been forbidden to row again, I lost touch with the case. At the present time he is said to be well.

CASE II

In May, 1927, a muscular and healthy-looking undergraduate, aged 19 and weighing over 12 stone, at the end of a year's rowing at Cambridge, woke up one morning to find his left arm swollen and rather cyanosed. He rowed the same afternoon, but next morning the arm ached considerably and swelling had increased. I found his left arm cyanosed and evenly swollen from fingers to shoulder. It did not pit on pressure. There were dilated veins over the pectoral region and antero-lateral chest wall. The radial pulse was good. No thrombosis of the axillary vein could be felt. A report on the skiagram stated that there were old tuberculous glands in the chest and some evidence that the trouble was not quiescent. I found a small rise of temperature (98.8°)