DISCUSSION

The response to treatment in these cases is very varied. Case 1 appears to justify Sutton's claims, since the attack lasted only about seven days without the subsequent development of heart disease. In Case 2 a second course of treatment was necessary. The movements only subsided during the sixth week after the onset of the attack. It is therefore possible that, in the absence of treatment of any kind, the attack would have come to an end by then. Case 3 was under observation during two separate attacks, one treated with T.A.B., the other by the usual sedative measures. The attack treated with T.A.B. lasted about two weeks, that treated by conservative methods two months. This case therefore appears to confirm the claim that the new treatment cuts short the duration of choreic movements. On the other hand, it demonstrates that there is the same liability to recurrence with either method of treatment. Finally, Case 4, whilst bearing out the effect on the choreic movements, shows in a rather tragic manner that the pyrexial treatment has no retarding influence on the course of the cardiac complications. It is even possible that it had an accelerating effect.

CONCLUSIONS

The conclusions to be drawn from the results are that the treatment of chorea by artificial pyrexia (1) shortens the duration of choreic movements, (2) does not prevent recurrence, (3) appears to have no beneficial action on rheumatic carditis.

In view of these conclusions it is clear that the method requires exhaustive trial before it can be generally adopted. Its grave danger lies in the tendency to produce a false impression of cure by abolishing the movements, which in themselves are merely a symptom, when serious changes may be occurring in the heart. We do not allow a child with acute rheumatism out of bed as soon as the joint swellings have subsided under the influence of salicylates. In the same way it is undesirable to allow a child out of bed two weeks after the onset of an attack of chorea unless it can be conclusively proved that the treatment which cuts short the choreic movements also cuts short any accompanying carditis. When these facts are borne in mind it is clear that the use of artificial pyrexia has a very limited place in the treatment of chorea.

REFERENCES

- ¹ Findlay, L.: *The Rheumatic Infection in Childhood*, 1931, p. 50.
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- ³ *Journ. Amer. Med. Assoc.*, August, 1931, xcvi, 299.
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TREATMENT OF CHOREA BY INDUCED
PYREXIA

BY

HUXLEY FISH, M.R.C.P.

ASSISTANT MEDICAL OFFICER, HIGH WOOD HOSPITAL FOR CHILDREN,
BRENTWOOD, ESSEX

In the *British Medical Journal* of June 10th, 1933, Dr. Donald Bateman gives a description of the treatment of Sydenham's chorea by pyrexia induced by intravenous injections of a triple typhoid vaccine. The following is an account of a case treated by this method. The author intends to apply the treatment to a series of cases, but in the absence of any other suitable ones the experience gained from the first seems worthy of publication.

CASE RECORD

The patient was a girl, aged 9 years. She had had two previous attacks of chorea. The first one, starting in December, 1930, lasted four months. The second, starting in

December, 1931, lasted ten months, five of which were spent in a hospital and three in a convalescent home in the country. The present attack started in January, 1933, two months after her return from the above-mentioned convalescent home. The child's mother stated that this attack was worse than either of the other two. When admitted to High Wood Hospital on June 28th, 1933, the child had been continuously in bed for six months—three at home, and three in a general hospital.

State on Admission.—Choreiform movements of the upper part of the body were severe. The child grimaced, waved her arms about continuously, and made writhing movements of her trunk from side to side. Voluntary movements were very clumsily performed. She could not hold a pencil in the writing position, and was unable to put on her bed jacket. There were no movements of her legs, in which there was slight loss of muscular power. Both knee-jerks were absent. Speech was very little affected, except that the child was reluctant to talk, and never did so spontaneously. The condition of the heart was as follows. The apex beat was in the fifth intercostal space, 7 cm. from the mid-sternal line (in the nipple line). The area of cardiac dullness was normal. A soft systolic murmur was faintly audible in the mitral area, and conducted towards the axilla.

Treatment.—The first injection of vaccine was given ten days after admission. During this time no appreciable change took place in the child's condition. She was kept lying down in bed all the time on an open-air balcony. The vaccine used contained 1,000 million typhoid and 500 million each of paratyphoid A and B per cubic centimetre. (This is slightly weaker than Dr. Bateman's vaccine, which contained 750 million each of paratyphoid A and B.) It was administered intravenously, three hours after breakfast on each occasion. The details of the management of the pyrexia were the same as those described by Dr. Bateman, with the exceptions that no aspirin was administered and the intervals between successive treatments were longer. The child was nursed between blankets, and hot-water bottles were used to help raise the temperature. The first injection was 0.05 c.cm. As this produced only a moderate pyrexia (103° F. rectal), 0.1 c.cm. was given next time. The temperature now rose very rapidly to 106°, and in spite of tepid sponging it reached 107° before it was finally brought down with ice-packs. The third injection was the same quantity as the second, and this time the temperature rose rather less rapidly to 105°, and was then successfully controlled with ice and sponging. For the fourth injection 0.15 c.cm. was given, and 105° was again reached. The interval between the first and second injection was two days. After the second injection, which produced a temperature of 107°, it was thought advisable to watch the child's general condition and the state of her heart for a few days before continuing. As no ill effects were observed, the third injection was given ten days after the second, and the fourth three days after the third. During the early stages of the pyrexia the child was very fretful and irritable. She complained of headache, and vomited from one to three times. Later she became drowsy, except during the last bout of pyrexia, when she was so restless and excitable throughout that considerable difficulty was experienced in taking the temperatures. During the second bout a rigor occurred. Sweating was not a very prominent symptom, which probably accounts for the rapidity with which the temperature rose. On the morning following each bout the child felt quite well.

Effect on the Heart.—An examination of the heart was made before and after each bout, and also at the height of the pyrexia. During the first and third bouts there was no evidence of cardiac disturbance beyond a tachycardia, which was not out of proportion to the pyrexia. During the second bout, at the height of the pyrexia, there was slight cardiac dilatation, the apex beat being palpable about one centimetre outside the nipple line. It had returned to its normal position the following morning. During the fourth bout there was again slight dilatation, and the child complained of pain over the praecordium. On the following day the dilatation was still present, although the pain had gone. The murmur at the apex was louder and better conducted than it had been previously. This, however, was presumably only due to a relative incompetence of the mitral valve, for on the next day the apex beat had returned to its normal position, and the murmur was only faintly audible.

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

W. N. PICKLES, M.D.

AYS GARTH

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