REMARKS

ON THE

SYMPTOMS, AND TREATMENT BY INTESTINAL
ANTISEPSIS, OF ENTERIC FEVER.

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I PROPOSE in this paper to give a brief summary of the symptoms and history, so far as I have myself observed them, of forty-six cases of enteric fever treated in hospital during the last fifteen years. As concerns treatment, I shall sketch briefly the methods employed, and contrast the results obtained by intestinal antiseptics with those following the ordinary expectant method. The duration, symptoms, treatment, and issue of these cases are represented as far as practicable in the tabular statement annexed.

The average age of my cases was 23.9 years, and, as we usually find in hospital statistics, the large majority were males. The febrile period, indicated in the synopsis by the dark horizontal line, expresses the time elapsing between the initial rigor or other first symptoms and the decline of temperature to the normal. A note of interrogation at the left end of the line implies that it was impossible to ascertain the date of first symptoms. For statistical purposes I add a week to the observed duration in these cases. In 38 cases which recovered and of which I have complete records the average duration of fever was 30.8 days, including any relapse or recrudescence that followed. Seventeen cases had a relapse or return of fever, usually slight. In some cases the disease was of great severity and long duration; for example, in Case 7 the pyrexia lasted 70 days; probably this may have been due to an intercurrent relapse. Excluding Cases 24 and 38, which I place in brackets, and which were not treated as cases of enteric fever, the average stay in hospital of the 44 cases was 46.7 days.

It is most convenient to recount special symptoms under the head of the various systems, as one would do in recording a single case. I commence with the integumentary system. Among 44 cases the typical rose rash was present in 27; in Case 30 desquamation followed; in Case 1 erysipelas formed a troublesome sequela. In 5 cases sudamina were observed, in 2 boils, and in 1 instance a carbuncle appeared during early convalescence, while 2 patients suffered from abscesses in the subcutaneous tissue.

Taking next the alimentary system, we had invariably dryness and whiteness of tongue and defective salivary secretion; in 1 case hæmorrhage from the gums occurred. In 10 instances marked tonsillitis or pharyngitis were present, and were much complained of by the patient; doubtless they existed in slighter degree in many other cases. In a private case I remember once seeing deep ulceration of tonsils and pharynx with separation of large sloughs, which reminded one of the sloughs from Peyer's patches. In 9 cases vomiting occurred, in Case 12 to such an extent as to endanger life. Loose stools of the typical character were noted in 33 cases. In Case 30 we had evidence that ulceration does not necessarily cause diarrhoea; the patient had persistent constipation, and the necropsy revealed unusually extensive ulceration. In 4 instances hæmorrhage from the bowel occurred in considerable amount. Tympanites was common, and in a few instances amounted to meteorism. Soreness on pressure in the right iliac fossa was observed in 19 cases. From the tenderness on pressure which frequently existed in the umbilical region I was convinced that the mesenteric glands were enlarged and sore. In Case 10 perforation and fatal peritonitis occurred after thirty days' treatment in hospital, and in spite of care and rest.

Under the respiratory system I shall take first the nasal chamber. We had 5 cases of epistaxis, in Case 2 so formidable as to imperil life, and to require the frequent use of ergotine. Bronchial catarrh occurred in 23 cases; to a severe extent in about 8. In 4 cases pneumonia occurred. Case 21 is a man

who was all but drowned in a dock; vitality was restored by artificial respiration, after which he vomited a large quantity of foetid dock water; then came on acute double pneumonia and left-sided pleurisy, with effusion, requiring thoracocentesis; while recovering from the pneumonia enteric fever set in, commencing about the twenty-sixth day after admission. Case 44 was admitted with influenza and acute double pneumonia of great severity. When recovering the patient was attacked on the twenty-seventh day after admission by enteric fever. In the former of these two cases, I assume that the enteric contagion was derived from the dock water. In the second case, it is difficult to explain. No typhoid case was in the ward at the time, and the sanitary arrangements of the infirmary are, I think, as perfect as they can be made. A possible explanation is that the first illness, which appeared to be merely pneumonia, was enteric fever without characteristic symptoms.

As regards the circulatory system, rapid pulse with low tension was invariable, with frequent diastolic murmurs. The first cardiac sound frequently became muffled and indistinct during the second week. One case of thrombosis occurred in the leg. As to the spleen, I confess myself of Dr. Fagge's opinion, that although enlargement is common, it is often difficult to detect, being masked by tympanites. In Case 30, the spleen weighed 1 lb. I have observed the organ to be tender on pressure during the first week, and to lose the tenderness later.

As regards the nervous system, headache was common, deafness also was frequent. Convulsions, which are rare in the adult, occurred in Case 24, accompanied by coma. Case 5 had severe muscular cramps, and Cases 7 and 43 complained respectively of pedal and crural neuralgia during convalescence. Nocturnal delirium was frequent. I had only two cases of the temporary imbecility which sometimes follows the disease; patient No. 39 became imbecile during the interval between the first defervescence and the relapse. He was a public-house keeper, who had taken much spirits. Case 44 was partially imbecile, also during the later stages of the disease. Both recovered perfectly.

As to the renal system, we had seven cases of albuminuria and one of hæmaturia.

The subject of treatment is more practical and more interesting. In the synopsis I have put down every case I had, but in the consideration of treatment I must exclude the two in brackets, namely, Case 24, in which case enteric fever was first diagnosed at the necropsy, and Case 38, a patient who, after treatment at his own house by a general practitioner, was brought to the infirmary during a relapse, and speedily showed symptoms of perforation. The remaining 44 cases I divide into two groups of 22 each. The one group was treated by the expectant method; among these cases 4 deaths occurred. The other group was treated by antiseptics, with the object of securing intestinal antiseptics; of this group every case recovered. In most respects all these cases received the same treatment. They were kept in the recumbent position until temperature had been normal for ten or twelve days. All were put on a light diet; milk and lime water, and if there was but little flatulence or diarrhoea, beef-tea was given. Alcohol was administered when the first cardiac sound became muffled and the pulse soft, the amount, 2, 3, 4, rarely 6 ounces of brandy, in small divided doses with food. It was always reduced in quantity and abandoned as convalescence advanced and the pulse strengthened. Hyperpyrexia was not frequent, but when occurring it was treated sometimes by tepid baths, but more frequently by cold sponging in the bed and by the administration of 5-grain doses of quinine. If the night rise of temperature was not beyond 104° we did not interfere. In enteric fever I prefer sponging with spirit and water to the use of the bath, it disturbs and exhausts the patient less. I always avoid the use of antipyrin and antifebrin.

During convalescence the patient is kept for a considerable time on a light diet, only foods which are easily and quickly digested being given. By degrees, increasing amounts of farinaceous food, meat juices, fish, eggs, and meat are in succession allowed. Two or three instances falling under my own observation of evil results following too early a return to a meat diet have caused me to regard it with dread; for example, I remember a clergyman who took a substantial meal

of roast mutton two or three days after his temperature had fallen; he did this in opposition to the advice of his medical attendants. Gastric and intestinal dyspepsia followed, and death occurred in three days. Case 38 in my list was treated at patient's own house by a practitioner, but when convalescing, in opposition to the doctor's advice, he resumed a heavy meat diet, and refused to remain in bed. Severe dyspepsia, flatus, perforation, and death followed.

As regards the constipation which so often occurs during convalescence, I allow it to continue as long as seven or eight days, provided the intestinal antiseptic is being taken. No harm seems to result. If it is needful to interfere, a glycerine enema is used. Any special symptoms of course are treated.

All of the 44 cases were treated on the principles just stated, but in addition, as I have said, half the total number took regularly a drug which was intended to produce intestinal antiseptics. The results of this latter treatment have been satisfactory, but before stating them I wish to say I tried several antiseptics, namely, salicylate of soda, creasote, naphthalin, and alpha-naphthol. Six cases were treated by the first drug; all recovered, but still I believed the salicylate to have a nauseating and depressing influence. The average stay in hospital was prolonged to fifty-six days. I have, therefore, abandoned the use of the salicylate in enteric fever. I have observed no bad results from the use of creasote, naphthalin, and alpha-naphthol. The average stay in hospital of 16 cases treated by one or other of these was forty-two days; the average stay in hospital of the 22 cases, including those treated by salicylate, was forty-six days. As to the cases treated by the expectant method, of whom, as I

have said, 4 died, the average stay in hospital of the remaining 18 who recovered was 52.1 days.

The average duration of pyrexia of the 22 cases treated by antiseptics was 25.3 days. From the same number treated by the expectant method I deduct 4 who died and 2 whose charts have been lost. The average duration of pyrexia of the remaining 16 is 37.9 days. The most striking point in the comparison of the two groups is the immunity from relapse enjoyed by the cases which were treated by intestinal antiseptics. Of the 22 cases the average days of relapse were 1.8, while among the 16 cases who recovered under expectant treatment and whose charts are preserved; the average days of relapse were 9.

Stated in tabular form the total results are as follows:

	Deaths.	Days of Fever.	Days of Relapse.	Days in Hospital.
Expectant treatment ...	4	37.9	9	52
Intestinal antiseptics ...	—	25.3	1.8	46

Watching these cases from day to day I have been much impressed by the apparent good effects of the intestinal antiseptic treatment. It is obviously a rational method. There is considerable evidence that such bodies as chlorine, creasote, naphthalin, iodine, iodoform, and alpha- and beta-naphthol are destructive to septic and poisonous compounds and organisms found in the intestinal canal. It is, therefore, antecedently probable that they would be of service. I am, how-

Synopsis of 46 Consecutive Cases of Enteric Fever.

No.	Initials.	Age.	Sex.	Duration of Fever and relapses in weeks.	Rash.	Diarrhoea.	Sore Throat.	Spleen enlarged.	Miscellaneous Symptoms.	Treatment.	Days in Hospital.	Result.
				1 2 3 4 5 6 7 8 9 10 11 12								
1	H.W.	22	F					x	Albuminuria, Erysipelas.	Salicylates	101	R
2	A.S.	26	M					x	Epistaxis, Deaf.	do.	57	R
3	I.O.	18	M					x	Sudamina.	do.	35	R
4	H.B.K.	20	M					x		do.	41	R
5	D.C.	34	M					x	Hæmorrhage, Cramps.	do.	74	R
6	T.B.	32	M					x		do.	27	R
7	S.O.	17	F					x	Neuralgia in feet.	Expectant	67	R
8	M.B.	24	M	Chart lost				x		do.	7	D
9	E.R.	21	M					x	Deaf, Vomiting.	do. & Quinine	48	R
10	A.E.	32	M					x	Deaf, Perforation.	do	31	D
11	I.McC.	26	M					x		Expectant	20	R
12	P.J.	29	M	Chart lost				x	Vomiting, extreme prostration.	do. & Quinine	51	R
13	C.W.	22	F	do.				x	Vomiting.	do.	47	D
14	A.K.	16	M	do.				x		Expectant	26	R
15	I.S.	16	M					x	Sudamina	do.	35	R
16	A.P.B.	20	M					x		do.	24	R
17	I.B.	3	M					x	Convulsions, Vomiting.	do.	81	R
18	B.T.	13	M					x	Sudamina.	do.	63	R
19	I.M.	34	M					x		do.	49	R
20	I.H.R.	39	M					x	Vomiting, Albuminuria.	do.	19	R
21	J.Q.	26	M					x	Pneu., Pleurisy, Epistaxis, Thromb., Boils.	do.	81	R
22	G.F.	16	M					x	Epistaxis, Vomiting.	do.	81	R
23	C.T.	16	M					x		do.	82	R
24	P.O.	28	M					x	Convulsions.	do.	3	D
25	P.A.	20	M					x	Sudamina.	Creasote	35	R
26	B.McD.	38	M					x	Epistaxis, Deaf.	Expectant	62	R
27	M.K.	21	M					x	Epistaxis, Vomiting.	do.	48	R
28	P.F.	17	M					x	Hæmorrhage, Pneumonia, Boils, Abscess.	do.	76	R
29	I.C.	21	M					x	Deaf, Hæmorrhage from mouth.	do.	25	R
30	W.J.	22	M					x	Pneumonia, Albuminuria.	do.	13	D
31	G.K.	13	M					x	Desquamation.	Naphthalin	51	R
32	H.K.	18	F					x		do.	58	R
33	A.D.	16	F					x		do.	36	R
34	I.B.	50	M					x		do.	35	R
35	M.McL.	24	F					x	Albuminuria, Vomiting, Sudamina.	do.	35	R
36	I.R.	18	F					x	Deaf.	do.	35	R
37	W.T.	17	M					x	Hæmaturia and Albuminuria.	do.	47	R
38	L.S.	39	M	came in with perforation during a relapse (spirit drinker)				x		do.	52	R
39	I.G.	40	M					x		do.	5	D
40	G.N.	28	M					x	Hæmorrhage, Anal Abscess, Deaf.	Naphth. in part	80	R
41	R.H.	17	M					x		Naphthalin	34	R
42	A.E.	13	F					x		do.	28	R
43	T.W.	50	M					x	Deaf.	do.	28	R
44	R.F.	22	M					x	Vomiting, Crural Neuralgia.	do.	48	R
45	M.B.	30	F					x	Pneumonia, Albuminuria.	do.	49	R
46	W.O.W.	17	M					x	Deaf.	do. in part.	31	R

ever, fully aware that the number of my own cases is too small for the drawing of a definite conclusion; they only become of value when used to support the larger experience of others. Dr. Wolff, of Philadelphia, for example, has treated 100 cases with naphthalin; the average febrile period was 24.4 days, and the mortality 2 per cent., after excluding deaths from heart diseases and phthisis, and those of patients brought in moribund. Dr. Gramshaw treated 116 cases with iodine and carbolic acid, and had no deaths. Bouchard states that from the year 1854 to 1885 the mortality from enteric fever at the Lariboisière Hospital was 21.1 per cent. Since 1885, the treatment pursued has been quinine with intestinal antiseptics, and the mortality has fallen to 11.7 per cent. I could adduce many more evidences of the value of intestinal antiseptics in the treatment of enteric fever, but I do not wish to lengthen this paper. I may add that I administer the naphthalin in the form of pill; six or eight pills, each containing 3 or 4 grains of the drug, are given daily. Alpha-naphthol I have also given in pill in similar amounts.

I fear that a certain mortality will always attend enteric fever, but, as the use of antiseptics has lessened the danger arising from external injury to the body, so the experiments of others on a large scale, and my own very limited experience encourage me to hope that intestinal antiseptics may similarly lessen the mortality of enteric fever.

REMARKS

ON

THE SURGICAL TREATMENT OF GENERAL PARALYSIS OF THE INSANE.

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IN submitting the following record of our experience in the surgical treatment of general paralysis in the Stirling Asylum, we consider it proper to state our reasons for the performance of these operations. After carefully considering the subject from different aspects, we concluded that some further acquaintance with the operation and its results was desirable. The pathological conditions usually found and generally believed to exist in the disease appeared to give countenance to the supposition that surgical interference might interrupt or permanently relieve the affection. The operation had been shown by the previous experience of others to be unattended with any danger, provided, of course, that it were carefully performed, and that ordinary attention was afterwards given to dressings and nursing.

Briefly stated, we considered that the operation was justified (1) because the pathology of the disease seemed to mark it as capable of relief by surgical operation; (2) because further acquaintance with the results of operative interference was desirable in the present state of our knowledge; (3) because of the proved safety of operative treatment.

General paralysis is a disease of the brain cortex, chiefly affecting the fronto-parietal regions. The disease in most cases is limited to areas on the vertex of those regions. Whether it is regarded as of nervous origin, or looked upon as a primary inflammatory affection of the non-nervous elements of the cortex, the limitation of affection is capable of explanation on the generally accepted theory, of which Meynert¹ is the chief expounder—that certain portions of the cortex may be the seat of functional hyperæmia, while other (even neighbouring) areas remain non-hyperæmic. It is more than likely that these portions of the cortex in front of the fissure of Rolando are chiefly concerned in what we call, for want of a better name, the higher mental processes. The experiments of Goltz and of Ferrier seem to bear this out and to strengthen the view. We may, in addition, cite the weighty authority of Ross.² Further, it is believed by many

psychologists that the motor areas of the cortex underlie, to a great extent, the higher mental processes. If now we consider general paralysis in the light of a mental, or, more properly, a mental motor affection of the brain cortex, we come *a posteriori* to a plausible explanation of why that particular region is more chiefly affected.

Whether the disease is of primary nervous origin or not does not appear to be of very essential importance except in so far as it directly bears upon the results expected from surgical interference. Some authorities hold that the disease is a degeneration of the nerve elements of the cortex, and that all the phenomena are explicable and ought to be assigned to that cause. The chief modern upholders of that view would appear to be Drs. Clouston and Savage.³ Other authorities again hold that the affection is primarily a vascular one—that is, 'inflammatory, and that the nerve cells are secondarily affected. We believe we are justified in placing Dr. Bevan Lewis⁴ among the upholders of this theory. We have been unable to decide to which view Mickle⁵ inclines, as his judicial summing up of authorities on this point is a little confusing. The following quotation, however, epitomises his opinion, "on the whole, we may view general paralysis as essentially commencing with hyperæmia and ending with chronic cortical degenerative cerebritis."

Granting that the disease is primarily nervous, the result of morbid over-activity of the cortical cells would be hyperæmia of special localities, for, as Meynert⁶ points out, not only is the anterior cortex a vasomotor centre, but each region of the cortex controls its own blood supply, so that when an associated area is active, it is at the same time hyperæmic. If, however, we adopt the theory of primary vascular affection, we see that, as a result of this congestion, the nerve cells, owing to their unique method of blood supply, through the perivascular canals, must suffer (as they actually do) and be thrown into a state of morbid over-activity, which ought, according to the advocates of the nervous theory, to augment the existing state of congestion of the blood vessels. We know on the authority of Duret⁷ that in certain cases of cerebral concussion the resulting vascular paralysis may pass on to inflammatory congestion. Therefore, whichever view of the origin of this disease is entertained, it must be acknowledged that there is vascular inflammatory congestion. It must be admitted that if the origin of the disease is a primary nervous degeneration followed secondarily by inflammation, surgical treatment would not be so hopeful, for, while relieving symptoms, it could hardly be expected to do more than check the inherent degeneration of protoplasmic vitality. It seems probable, however, that there is something more than passive inflammatory congestion of the blood vessels of the affected region to be considered. The condition presents a more active pathological aspect than can be accounted for by mere passive congestion, and points to the presence of an inflammatory stimulant.

Dr. Bevan Lewis says that the earliest indication of morbid change is certainly presented by the vascular tissues. There is, first of all, a true paralysis of the tunica muscularis of the cortical arteries (pial vessels), accompanied by extensive protoplasmic segmentation and nuclear proliferation. There follows, as a consequence, a transfusion of the fluid contents of the vessels into the perivascular canals, and from thence into the pia-arachnoid spaces into which these canals open. Finally, there is a diapedesis of leucocytes into the perivascular canals and the above-mentioned spaces.

Is this, then, a true primary active inflammatory condition of the vascular tissues or a passive congestion, the result of a vasomotor paralysis due to primary affection of the cortical cells which have undoubtedly a vasomotor function? Our opinion is that if such a vasomotor paralysis could be admitted to account for the inflammatory state, death would ensue more speedily than it does from increasing paralysis.

The cause of such an inflammation is as yet unknown, but, according to the latest researches, there is probably always present in inflammation an organism which produces or accompanies the characteristic phenomena which we recognise

³ *Journal of Mental Science*, vol. xxxvii, p. 495.

⁴ *Textbook*, p. 439.

⁵ *General Paralysis*, p. 342.

⁶ *Psychiatry*, p. 216.

⁷ *Traumatismes Cérébraux*, t. 56.

¹ *Psychiatry*, p. 214.

² *Diseases of the Nervous System*, vol. ii, p. 346.