

Post-typhoid dyspepsia / by Anthony Bassler.

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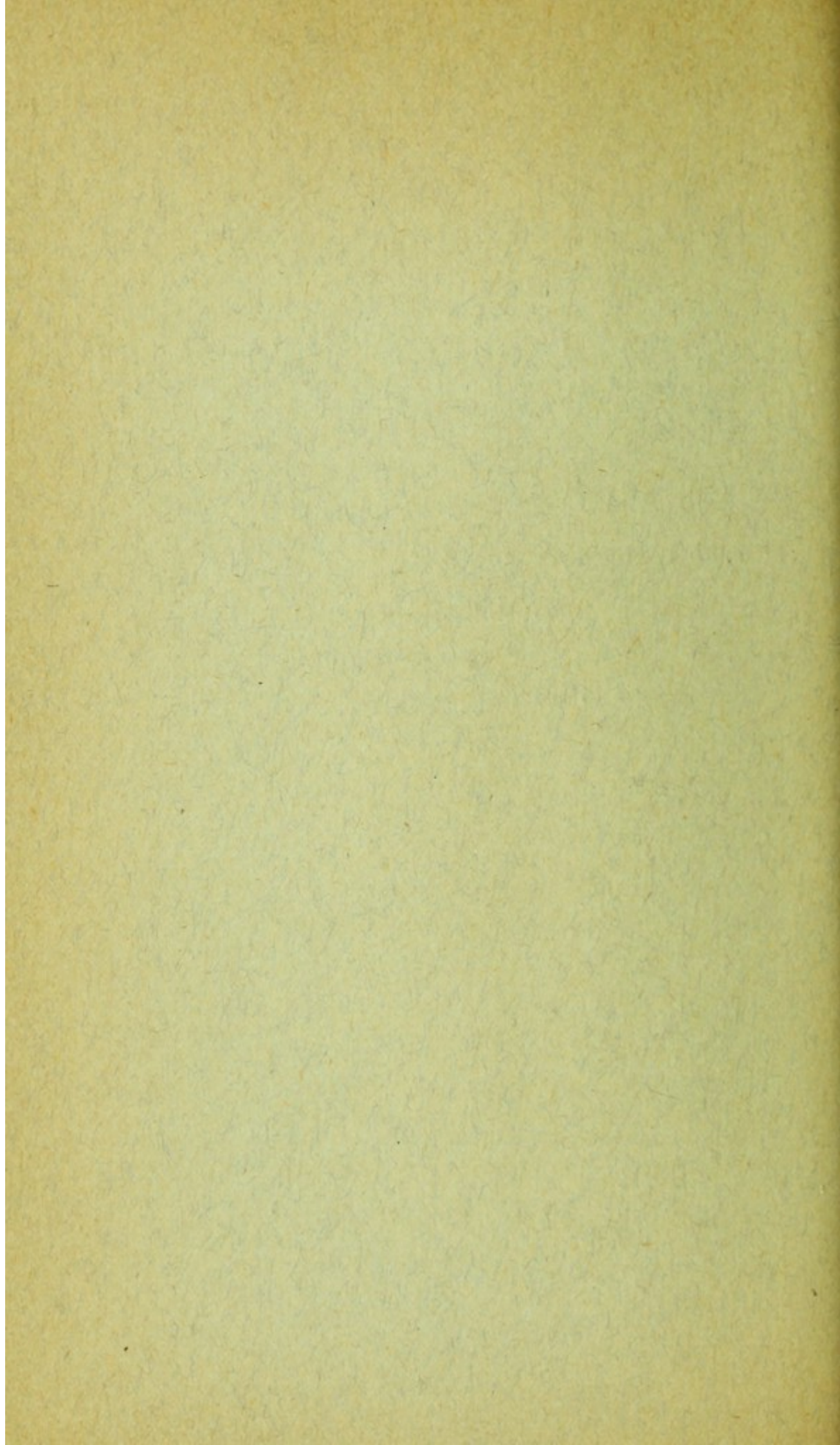
Post-Typhoid Dyspepsia

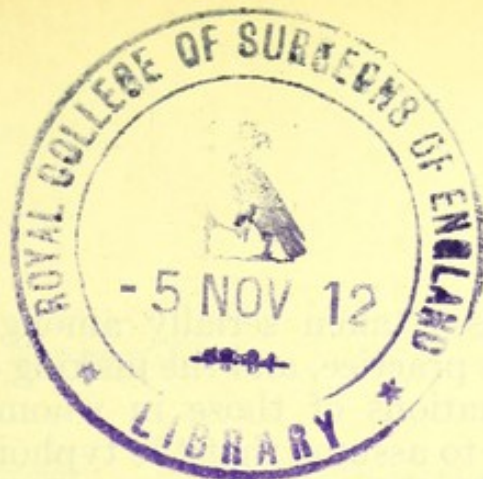
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POST-TYPHOID DYSPEPSIA.*

BY ANTHONY BASSLER, M.D., NEW YORK.

SINCE the time that typhoid was separated from typhus fever, the medical world has been littered with a consideration of this protean disease. The contributions on typhoid fever from American sources alone comprise one of the instructive eras in the development of medicine in America. Judging from the very thorough knowledge that we have of this disease in the way of the bacillus typhosus, the modes of infection, pathology, symptomatology, complications, sequelæ, prophylaxis and treatment, it would seem as if the last words have been said. And still, within recent years, new facts pertaining to the disease have been pointed out, among which are states of a chronic type following the acute course of the infection, most notably metastatic deposits, chronic gall-bladder infections, and to which the general term of the "typhoid-carrier" has been given.

It occurred to me several years ago that in the histories of patients mentioning states of chronic disturbances in the digestive tract there presented a history of a previous typhoid fever in more or less association with the beginning of the dyspeptic symptoms in a considerable proportion. In this connection, the recent advances drawing attention to the persistency of the typhoid bacillus in stools and urines of patients who had been infected, led to the analyses of the histories of

* Read before the New York Academy of Medicine, April 16, 1912.

472 cases taken serially among those I saw in private practice, and the making of stool and urine examinations of those in whom it seemed warranted to assume that the typhoid fever had to do with the dyspepsia then present; and also to a comparing of the number of instances of typhoid fever with the two commoner diagnoses presented in the past histories of the cases, namely, appendicitis and pneumonia. In these 472 cases, there were 132 instances in which various diseases and conditions were connected with the history in certain suggestive, causative ways. Of these, there were 24 instances of typhoid fever; 17 who had had appendicitis, and 10 who had had pneumonia. There were 4 who had had typhoid, appendicitis and pneumonia closely enough associated that differentiation was not possible; and 76 cases with other states not considered in this article.

In analyzing these histories it is apparent that in 15 instances of typhoid fever the dyspeptic symptoms began within two years after the attack. In 6, the beginning dyspeptic symptoms closely followed the fever and had continued with an average duration of the dyspeptic history of over eighteen years. In 9, the dyspeptic symptoms began within two years following the attack of the fever, with an average duration of the continuation of a dyspeptic history of eleven years and six months. Averaging these, it is seen that in the 15 cases which began with dyspeptic symptoms following the typhoid in a short space of time, to consider a connection between them, the average duration of the dyspeptic history at the time the cases were seen by me was fourteen years and nine months, and that in 24 instances of typhoid fever states of chronic dyspepsia followed in $62\frac{1}{2}\%$.

In 472 case histories:

TYPHOID FEVER.

Case Number.	How long ago contracted.	Dyspeptic symptoms begun.	Age now.	Typhoid bacilli in stools.
1.	21 years.	11 years after.	40	None, 3 examinations.
2.	24 "	11 years after.	40	None, 5 "
3.	10 "	1 year after.	29	None, 7 "
4.	7 "	Continuous.	36	Present periodically, 13 "
5.	4 "	2 years after.	28	Present periodically, 9 "
6.	22 "	1 year after.	49	None, 6 "
7.	13 "	5 years after.	36	None, 5 "
8.	20 "	1 year after.	42	None, 3 "
9.	11 "	1 year after.	23	Present periodically, 9 "
10.	13 "	9 years after.	28	None, 5 "
11.	36 "	Continuous.	50	Present constantly, 11 "
12.	16 "	Continuous.	32	None, 6 "
13.	2 "	1 year after.	36	Present periodically, 14 "
14.	14 "	10 years after.	24	None, 4 "
15.	21 "	18 years after.	54	None, 5 "
16.	2 "	6 months after.	22	Present constantly, 7 "
17.	19 "	1 year after.	29	None, 5 "
18.	5 "	Continuous.	26	Present periodically, 10 "
19.	35 "	8 years after.	44	None, 7 "
20.	27 " }	5 years after second.	38	None, 15 "
21.	8 " }			
21.	15 "	13 years after.	30	None, 3 "
22.	16 "	1 year after.	48	None, 8 "
23.	10 "	Continuous.	51	Present constantly, 8 "
24.	16 "	Continuous.	35	Present periodically, 16 "

APPENDICITIS.

Case number.	How long ago contracted.	Dyspeptic symptoms begun.	Age now.
25.	12 years.	5 years before.	32
26.	2 years.	1 year after.	26
27.	8 years.	Continuous.	47
28.	14 years.	26 years before.	58
29.	10 years.	1 year before.	43
30.	9 years.	Continuous.	30
31.	1 year.	Continuous.	39
32.	11 years.	4 years before.	29
33.	2 years.	2 months after.	19
34.	11 years.	10 years after.	31

Case number.	How long ago contracted.	Dyspeptic symptoms begun.	Age now.
	(Two operations on appendix.)		
35.	2 years.	Continuous.	40
36.	20 years.	6 years after.	44
37.	10 years.	2 years after.	43
38.	6 years.	5 years after.	40
	(Appendectomy at once.)		
39.	7 years.	3 years before.	30
40.	7 years.	6 years after.	33
	(Operated upon.)		
41.	12 years.	5 years after.	38

PNEUMONIA.

Case number.	How long ago contracted.	Dyspeptic symptoms begun.	Age now.
42.	39 years	15 years after.	55
43.	20 "	3 years after.	30
44.	3 "	7 years before.	47
45.	10 "	9 years after.	30
46.	6 "	14 years before.	65
47.	7 "	6 years after.	24
48.	12 "	19 years and indefinite.	31
49.	20 "	19 years after.	43
50.	18 "	13 years after.	54
51.	14 "	10 years after.	34
52.	10 "	9½ years after.	31

One, — Typhoid and appendicitis close enough together that differentiation was not possible.

One, — Typhoid twice and pneumonia once close enough together that differentiation was not possible.

Two had both typhoid and pneumonia close enough together that differentiation was not possible.

Miscellaneous conditions having a close association with the beginning of dyspeptic symptoms given in the order met with in the history files: Ptomaine poisoning, mental shock, acute indiscretion of diet, exhaustive labor, syphilis, pregnancy and post-partum, alcoholism, chronic intestinal putrefaction, definite cases of gallstones, poly-arthritis, rheumatism, splanchnoptosia, pulmonary tuberculosis, acute gastritis, gynecological conditions, occupational, tropical intestinal disorders, heart, kidney and liver conditions, gastric ulcer, malaria, trauma, diabetes, neurasthenia, la grippe, erysipelas and peritonitis. Total, 76 cases.

Leaving out of consideration the miscellaneous conditions mentioned as the cause of dyspepsia, it is interesting to compare the above with appendicitis and pneumonia. Since we are well aware that dyspepsia commonly follows attacks of acute appendicitis and accompanies the chronic, it is interesting to observe that in the 13 unoper-

ated cases in which a definite diagnosis of appendicitis had been made at some time previous, states of dyspepsia close enough associated with the history of appendicitis to be considered in any causative way existed in only 6 cases, a percentage of slightly over 30, about half of that following typhoid fever. It therefore seems reasonable to assume, even deducting from this limited number of cases, that states of chronic indigestion follow typhoid fever twice as frequently as disease of the appendix. In the pneumonia cases, there was no instance shorter than three years after at which the dyspeptic symptoms began, and, with our knowledge of life's history of the pneumococcus, this entire set of cases may be considered as having no etiological connection with dyspepsia.

In the great majority of cases of typhoid fever, and perhaps always, the typhoid bacilli are present in the gall bladder during the attack, and the bacilli may persist in the gall bladder after the attack for an indefinite period — instances of as long as eighteen years are on record. Herewith is reported a case in which they were recovered thirty-six years after, and others at shorter intervals. It is known also that typhoid bacilli may occur in the gall bladder as a local infection without the patient ever having had typhoid fever as we usually see it to be in a clinical way. From the above statistics and close study of many other histories of dyspepsia cases, it seems warranted to assume that the general term "post-typhoid dyspepsia" is worthy of consideration, and that not a few of the cases of typhoid fever present this following the acute course, which lasts from two to seven weeks, in persons who at that time acquire a general immunity which banishes the bacilli from the blood

and causes a cessation of general symptoms although the germs still persist in some regions of the body, notably in the gall bladder and intestines. It is known also that there is a proportion of instances in those who have been exposed to the ingestion of typhoid bacilli in which no reaction occurs with any disturbance to health, but whose blood serum nevertheless displays a marked agglutinating action on the typhoid bacilli. Whether or not intestinal ulceration is present or absent from these cases cannot be determined, although it is probable that a proportion are affected, since typhoid ulcers have been found in people dying suddenly of heart failure who had been in apparent good health. It has been stated that the typhoid bacilli in cases in which no obvious illness follows their ingestion always disappear, and that in 95% of the acute typhoid cases the bacilli disappear rapidly and that health is restored. It seems to me that these beliefs can be questioned as being too conservative. It is interesting in this connection to consider in what locality the typhoid bacilli may be latent in the cases which have apparently recovered from the fever and in which one or two examinations of the stools do not show their presence later than three weeks after defervescence. In this is bound up the interesting subject of chronic bacilli carriers, and the question as to whether in such instances the bacilli are only present in the gall bladder and whether or not they are constant denizens of the bacteriology of the intestinal canal. This differentiation in the majority of instances is impossible by ordinary physical and in a few even careful examinations of the feces. It seems reasonable to believe that in those who excrete the typhoid bacilli in a periodical way (and even here irregu-

larity may exist for a time) years after the acute disease are probably gall-bladder cases. A close study of all kinds of infections of the gall bladder will often give a distinct suggestion of periodicity, due probably to the gall bladder requiring a certain length of time for complete emptying and thus discharging large quantities of its contained bacilli into the intestine, or certain states of digestion or general health causing a proliferation of the gall-bladder bacilli.

Mayer has pointed out that in addition to the lesions of the biliary tract the typhoid carriers may suffer from frequently repeated attacks of intestinal catarrh. In the 15 cases whose dyspeptic symptoms followed within two years after the typhoid fever, I recovered the typhoid bacilli in 9, 6 of them being present in the feces in periodical ways and 3 continuous. In only 1, however, was it possible to make a diagnosis of a chronic enteritis by the history and the examination of the feces in its mucous content and crystals. It seems probable, therefore, that all of the others were either chronic gall-bladder infections, chronic infections of the bacteriology in the intestine, some infected focus of a chronic type in the lymphatic structures of the intestine, or combinations of these.

In these cases, 307 analyses of the urine were made for the typhoid bacilli and in none of them were they found, although the colon bacillus was regularly found present in Case No. 19. What was of still greater interest was that in the 29 Widal tests of the blood, 2 in each case and 3 in Case No. 16, not one gave a reaction that could be considered positive in a diagnostic way. This is not only of interest in showing that this simple to perform test (as compared to the feces examinations) is of no value in the diagnosis of these chronic

typhoid conditions, but is of moment in showing that this reaction may not persist as long after typhoid fever as we have believed. The nearest approach to a reaction was seen in Case No. 23, in which in two examinations it was present in dilutions of 1:20. Leucocyte counts were made in all, and in none was this distinctive in any way.

In the 6 cases in which the typhoid bacillus could not be obtained from the stools, other diagnoses were apparent to account for the dyspeptic symptoms. In 5 of these, chronic excessive putrefaction in the intestine due to a high content of the *B. coli communis* was apparent, and it may be that an attack of typhoid infection can cause a change in the running state of the bacteriology of the intestinal content in that direction. All of these were distinct indolic cases with a bacteriology characteristically Gram negative, in 4 as high as I have ever seen.

The technic employed in the separation of the typhoid bacilli from the other bacteria of the stools was made in each stool examination by three methods: those of Conradi-Drigalski, endo fuchsin-agar, and the Bendick method. The typhoid bacilli were tested further by staining with the Gram method, grown in glucose-agar stab, litmus milk and on potato.

In 4 of the 6 cases that were probably chronic gall-bladder infections, a definite tenderness to deep pressure existed in the region of the gall bladder, although none had any demonstrable enlargement or firm feel to the liver. In the 3 that seemed to be infections in the intestines, there were no local tendernesses that I could elicit. In all of the 9 cases the history of more or less headaches was a feature, and seven complained of attacks of "biliousness," these coming on at intervals of every few weeks in the beginning

of their dyspeptic history, and after that more often. In 3 the attacks began with an intense headache, followed shortly by nausea and finally vomiting for several hours, the whole seizure lasting about a day. A feature with all of them was a shortage of ability to withstand mental effort or physical strain. They were always tired and became excessively nervous, with physical exhaustion when attempting these. The bowels moved normally in all, only one being constipated, and she only occasionally. The majority of them were subnourished and showed evidences of gastro-intestinal atony and ptosis. Two, however, were very well nourished, with organs in good tone and position. All of the cases in whom the typhoid bacilli were recovered from the stools claimed that they had not had the dyspeptic symptoms before their attacks of typhoid fever.

Of distinct interest were 4 of the cases who submitted themselves to a length of treatment for their conditions. Two of these were probably gall-bladder cases, and two probably intestinal ones. These were established on a diet arranged according to their work, age and physical condition, given $7\frac{1}{2}$ gr. (0.5 gm.) of urotropin before the meals and 1-10 gr. (0.0065 gm.) of calomel one hour afterward, and treated for six weeks with autogenous typhoid vaccine up to 400 million at a dose every fourth day, and then a stock typhoid vaccine for six weeks up to 400 million at a dose at the same intervals. At the end of the third month, although all claimed to have been improved in their symptoms, further examinations of their stools were made, and in each the typhoid bacilli were still present. It seems to me as if we would have to turn to surgical drainage of the gall bladder in those cases in which we can reason-

ably assume that the gall bladder is the source of the infection. For if we desire to render them symptom free in a logical and permanent way, and consider the public health of others in a general way insofar as typhoid fever is concerned, more than what medical treatment can now accomplish seems necessary in these cases.



