A Post-Graduate Lecture ON HYPERCHLORHYDRIA AND ITS COMPLICATIONS.

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BY

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Two reasons weighed with me in selecting this subject as the one on which to speak to you this afternoon: the one was that there has been a good deal of writing about it during the past twelve months, and a very marked difference of opinion expressed regarding it; while the second was that I had myself written on this subject as long ago as 1903,¹ and that I had not written on it since then.

When I made the communication referred to before the Edinburgh Medico-Chirurgical Society it met with two lines of hostile criticism: the one was that the condition could not be differentiated from other gastric disorders; and the other that it was purely a neurotic manifestation. More recent writings, especially by some surgeons, have again called in question or denied the existence of such a condition as hyperchlorhydria, and have attributed the symptoms referred to that condition to ulceration either of the stomach or of the duodenum.

We may with advantage approach the consideration of this important practical question from the physiological standpoint, and may on physiological grounds postulate this—namely, that

The Secretion of HCl Varies in Amount.

Pawlow and others have, by their experimental researches on dogs, shown conclusively that the amount of secretion of HCl can be varied—can be stimulated, can be controlled, and can even be inhibited by the administration of certain substances. In the human subject there is no more reason to doubt that the HCl secretion of the stomach varies in amount in different persons, or in the same person at different times, than that variations occur in every other gland in the body, whether it be salivary gland, liver, or mamma. It seems, indeed, unnecessary to argue this point—the proposition is recognized as a fundamental truth whenever the activity of the various glands of different individuals is considered. If this be true the further proposition must also be true—namely, that variation in secretion means that it may be below or above a certain standard in quantity or in potency—which of these it may be is of no importance to the argument.

When the secretion of HCl is under the standard it is hypochlorhydria, when over it is hyperchlorhydria. It is with the latter we are to deal at present.

The Use of HCl.

There is, however, the other secretion of the stomach—namely, pepsinogen. It is one of those interesting substances which only attains its acme of potency when acting in consort with another substance. This is the great purpose to which HCl is applied. It is said that digestion never fails from deficiency in pepsinogen, because of its great power even in small quantity; that, in fact, when gastric digestion fails, it does so from an insufficient supply of HCl, and not of pepsinogen.

The Stimulus to HCl.

It is well known that the secretion of these two substances is for the disintegration and digestion of flesh and other proteid foods. Pawlow has established our knowledge of this action, and has shown how savoury flesh foods stimulate the secretion of HCl. In the perfectly normal condition the amount of secretion is in direct ratio to the work which is required—that is, to the amount and kind of proteid taken. It surely puts no great strain on our credulity to believe that that physiological balance can be disturbed—that there can be too much as well as too little; yet we are told from some quarters that our belief in over-secretion is in a mere Will-o'-the-wisp—an *ignis fatuus*.

I therefore ask your attention to the following cases as helping us to form a judgement on this question. I begin with what may appropriately be called acute cases.

ACUTE CASES OF HYPERCHLORHYDRIA.

The symptoms of hyperchlorhydria not infrequently come on somewhat acutely, in strong, vigorous persons who have not suffered previously from digestive disturbance of any kind. As examples of this I may mention two cases:

CASE I.

A gentleman, aged 37, who was sent to me in November, 1908, had suffered for one month from pain and flatulence. The pain came on about two hours after breakfast, two hours after lunch, and two hours after an eight o'clock dinner. He had sometimes been awakened in the night with the pain. The pain lately had become almost constant, but he had noticed that taking food put it away for a time. The bowels had been constipated from the onset of the symptoms, and he had for this taken an occasional laxative. The tongue was furred. On examination of the abdomen, succussion showed the lower border of the stomach to be at the level of the umbilicus. There was nothing to note in his other systems. He had no worries, and he led the open-air life of a country gentleman. "Indigestion was common in his family, and his father died of it."

I advised a considerable reduction in the amount of food, wine, and malt liquor he was taking, and gave him belladonna and soda to take thrice daily. He saw me again at the end of a week and of a fortnight; he had no pain since he began the medicine, and by the latter date the dilatation of the stomach had entirely disappeared. I met him a few days ago, when he assured me that he had been perfectly well, and had had no return of his symptoms since he had seen me nearly two years ago.

CASE II.

A retired officer of the R.A.M.C., aged 54, consulted me in August, 1909. He had been attacked by pain between three and four weeks before, two hours or more after lunch, and it had lasted for a couple of hours. This pain kept recurring about two hours after taking food. He dieted himself very strictly, and found that the pain kept away if he lived on milk, biscuits, and lightly boiled eggs. He was, as a result of this meagre diet, getting weak and out of condition. The pain sometimes kept him awake at night. He found that taking hot milk removed the pain. The stomach was not dilated. There were no further points I need refer to.

I gave the patient belladonna and sodium bicarbonate, and a laxative pill of cascara, belladonna and nux vomica; I took off the restrictions on his feeding, but advised that he should have red flesh and chicken minced. The belladonna and soda were to be taken after food, as soon as the discomfort began. He reported himself to me in the end of September, and assured me that the medicine had very promptly removed his symptoms.

He kindly reported himself on September 21st, 1910, when passing through Edinburgh, and informed me that he had had occasional threatenings of a return of his symptoms, but found that he had the remedy in the drugs I had prescribed.

In cases of this kind the diagnosis is so simple that neither for purposes of diagnosis or treatment is it necessary to obtain the gastric contents. If you do remove them, as one does sometimes, you will find that they contain much free HCl.

I have selected these two cases as I have seen them so recently, and as there has been sufficient lapse of time since I saw them first to judge of the results.

CASES OF HYPERCHLORHYDRIA OF LONGER DURATION.

I select the following cases for the illustration of this class:

CASE III.

The patient was a medical man, aged 47, who consulted me in June, 1907. He had suffered off and on with acidity for twenty years. The attack from which he was suffering when he came to me had lasted for two weeks, and was unusually severe. Pain came on about two hours after food, and might continue up to the taking of the next meal; taking food relieved the pain. There was no vomiting, and the bowels moved daily and freely. There was no gastric dilatation, but as there was something in his stomach it was drawn off, and was found to be intensely acid from free HCl, while the material removed consisted of fluid and starch. The diet was regulated, and I recommended belladonna before food and bicarbonate of soda after food, as soon as the pain threatened. Ten days later he wrote saying how much better he was, and that, "although the weather has been simply atrocious, I have not suffered any more." I saw this patient a few weeks ago, when he assured me that he had been able to restrain his old enemy as soon as there was a threatening of reappearance. He repudiated the suggestion of having either a gastric or a duodenal ulcer.

CASE IV.

Another medical man, aged 35, consulted me in November, 1908. He had not suffered so long as our preceding friend, but was finding his energy sapped and life burdened by his recurring

attacks. He was cured, and has become an able exponent of the clinical manifestations of hyperchlorhydria, and a curer of the condition in others.

I select these cases as they were in medical men.

I now ask your attention more definitely to the symptoms of this disorder.

THE SYMPTOMS OF HYPERCHLORHYDRIA.

In approaching the consideration of the symptoms, I would preface it with a few general remarks on what is recognized as acid dyspepsia, where the symptoms are burning pain at the cardiac orifice of the stomach, acid eructations, water-brash, usually flatulence and constipation. The prevalent idea that these symptoms are due to the presence of other acids—as lactic or butyric—caused by the presence of fermentative processes in the stomach, is a mere myth. The acid which is present, and present in abundance, is HCl, secreted by the oxyntic cells of the stomach. In early adult life the symptoms are often such as have just been indicated. In other cases there is a sense of dragging or weariness, referred to the stomach, or a *hunger-discomfort*, coming on two hours or less after breakfast perhaps, and removed by taking a glass of milk, a drink of cold water or soda water, or a cup of tea.

In other cases, and more commonly somewhat later in life, much gastric discomfort, or definite pain comes on from one or two hours after a meal; and this is associated with mental depression, and great difficulty, amounting in some instances to complete incapacity for mental or physical effort. The misery of the hyperchlorhydric leads him to reduce and reduce his feeding in the hope of freeing himself from his depression, and he becomes thin and haggard in appearance. Sometimes he gets relief by vomiting, more commonly he does not vomit. He often gets relief by the eructation of some mouthfuls of very acrid stuff. His pain lasts for an hour or two, and if it lasts up to his next meal, the taking of it relieves him for some time, the duration of the relief depending upon the composition of the new meal. Some patients tell you that they relieve their symptoms by taking sodium bicarbonate, others by taking milk or by drinking water.

On removing the gastric contents, they are found to be intensely acid and to contain much free HCl. The contents consist of a slightly opalescent fluid, and a deposit consisting almost entirely of the remains of farinaceous food, giving a strong starch reaction, and microscopically seem to consist of starch grains and granules. No trace of milk curd, of white of egg, or of fish or flesh fibre may be found, although the finding of the latter depends upon the amount of these foods taken at the previous meal, and the time which has elapsed before the stomach contents are withdrawn.

The retention of broken-down cereal food in an intensely acid fluid is the usual characteristic of the material removed from the stomach, when the symptoms are present in these cases.

That this hyperacid fluid in the stomach is the cause of the pain and depression is proved by the immediate relief of the symptoms when it is withdrawn.

A like effect on the symptoms is produced by a sufficient dose of an alkali, and the best is probably bicarbonate of soda, without peppermint. The symptoms are also promptly relieved by taking milk or raw egg. Whenever in fact the free acid is sufficiently diluted, or fixed in combination with proteid, or neutralized by an alkali, or removed mechanically, complete relief is obtained.

PATHOGENY.

The question of pathogeny is beyond my present object. In persons subject to the condition, unsuitable feeding or drinking, fatigue, worry, chill, may determine an attack. To call it "neurotic" is a mere euphemism to denote the unknown or the misunderstood. The view I expressed seven years ago, that it was a manifestation of an *acid dyscrasia*, has been confirmed by the additional years of critical observation.

THE TREATMENT.

The dietetic treatment depends upon various considerations—mere reduction in quantity may be sufficient. Speedy relief may be obtained by a proteid dietary which

can be rapidly digested and is not too highly seasoned. I have a simple test for digestive conditions in Scotland, namely, that if porridge and milk cause acidity the digestion is not right. As regards drugs, sodium bicarbonate not only counteracts the free HCl, but, as Pawlow has shown, it inhibits acid secretion. For the inhibition of secretion various drugs can be used, but belladonna stands at the head of the list in my experience.

CONCLUSIONS.

I began by giving you notes of a few cases of acute and chronic hyperchlorhydria, and indicating to you the results of treatment in them. I have outlined the symptoms which characterize the condition; I have indicated how easy it is to remove them promptly, and the line to be followed if more permanent relief is aimed at. I could multiply these cases were it necessary. To me there is no doubt that we have in hyperchlorhydria a clinical entity, capable of exact diagnosis, and highly amenable to intelligent medical treatment.

THE COMPLICATIONS OF HYPERCHLORHYDRIA.

In the next place, I seek to draw your attention to what may be quite appropriately called the complications of hyperchlorhydria. And here we get into deeply interesting and very important questions, where your knowledge of gastric and abdominal examination, of skill in getting a clear history of symptoms from your patient, will determine the correctness of your differential diagnosis. In considering what we term the complications of hyperchlorhydria, it must not, of course, be assumed that all the different conditions which we look at as complicating hyperchlorhydria are necessarily and always associated with it, or that they do not occur apart from it. A tabulated list of important complications may be made as follows:—

Complications.

1. Acute gastric or duodenal ulcer.
2. Pyloric spasm.
3. Chronic gastric ulcer.
4. Chronic duodenal ulcer.
5. Gastric dilatation—
 - (a) Of fundus;
 - (b) Ptoxis of lower border (including other causes leading to the lower border being below the level of the pyloric outlet).
6. Gastropnoxis.

1. Acute Gastric or Duodenal Ulcer.

I need not occupy your time by dwelling on the symptoms of acute gastric ulcer. Its association with hyperacidity is well known, and the most enterprising surgeon does not suggest that such ulcer demands immediate surgical measures. In fact, acute gastric ulcer, as we know it clinically, and as it has recently been studied experimentally in animals by Dr. Charles Bolton,² heals rapidly under suitable conditions. That what is true of acute gastric ulcer should also be true of acute duodenal ulcer seems to me a rational corollary. That healing of duodenal ulcer takes place without surgical interference cannot be questioned. I may mention, as illustrating this fact, the case of a medical student, sent to me by one of my surgical colleagues, who had a history of hyperchlorhydria, and also evidence of duodenal ulcer, as indicated by pain and melaena on two occasions at least. Rest in bed and appropriate dietetic and medicinal treatment led to the disappearance of all the symptoms, and he has continued free from any recurrence for a number of months. Another patient—a woman—was sent into my ward with a history of having had a very severe illness, due to duodenal ulcer, over a year before, from which, under the care of a doctor in Morayshire, she had completely recovered. Before admission to the infirmary symptoms returned which again indicated the existence of duodenal ulcer. We tried dietetic and medicinal treatment for some weeks, but without effecting a cure. She was accordingly operated on. The scar of the healed ulcer was seen on the anterior wall of the duodenum, near the pylorus. A gastro-enterostomy was performed, and the patient made an excellent recovery. Whether she is thereby rendered safe from developing another ulcer is a point on which we do not have much evidence. The case, which is one of several, proved not only that duodenal ulcer may heal without operation, but that once having

had an ulcer does not secure the patient from having a second.

2. Pyloric Spasm.

Spasm of the pyloric sphincter, and sometimes of the antrum also, is a marked phenomenon in the more severe cases of hyperacidity. It is the cause of the severe pain in these cases. During the spasm the pyloric end of the stomach can be readily palpated. It is allayed by neutralizing the acid and by other means. Its persistence or recurrence leads to dilatation, and even to ptosis.

3. Chronic Gastric Ulcer.

Chronic ulcer means an ulcer the existence of which has been indicated by symptoms for weeks. The prominent symptom of gastric ulcer is pain after the ingestion of food, and its continuance until the stomach is emptied. Some time ago I had a man in my ward with hyperchlorhydria, for which he was successfully treated. Months afterwards he returned, not only with hyperacidity but with symptoms pointing to gastric ulcer having been present for some time. I at once recommended a gastro-enterostomy, which was performed. An ulcer with thickened edges was found some distance from the pylorus. He made a good recovery, but did not get the measure of relief that we anticipated. The ulcer itself was not dealt with, and it is a question for the surgeons to determine if it might not be wiser to do so.

4. Chronic Duodenal Ulcer.

The medical treatment of chronic duodenal ulcer is, to my mind, not worth spending time over, in the expectation that it will be healed by such measures. For both it and chronic gastric ulcer I strongly advise operation. Were it established that a serum treatment had been found which ensured the healing of chronic ulcerations, one's attitude towards this question would be changed.

5. Gastric Dilatation.

Under the name of "gastric dilatation" there is some confusion of idea, or the fact has not been apprehended that there are at least two conditions to which the name is applied. The first and commoner form of dilatation is characterized by enlargement of the fundus of the viscus with extension of the great curve upwards and to the left, and a corresponding increase in the antero-posterior diameter of the organ. This type of dilatation is met with in big feeders, and may sometimes be regarded merely as a big stomach. It may or may not be associated with symptoms calling for medical treatment. The other form of dilatation is that in which the inferior border of the stomach has fallen below the level of the pyloric outlet, when at the same time there is enlargement of the cavity and atony, although not immobility, of the muscular coat. This form can be conveniently called *ptosis of the inferior border*; there is ptosis or sagging of the great curve, without ptosis of the viscus as a whole, including the lesser curve.

In this connexion let me remind you that the position of the pyloric outlet moves somewhat with the position of the stomach; it moves somewhat to the right and slightly downwards, but it does not descend in proportion to the degree of ptosis of the inferior border. One result is that, when this form of ptosis occurs, the pyloric outlet is considerably above the lower border; and, as ptosis is always associated with considerable muscular atony, the stomach takes long to empty itself, and in many cases may never empty itself completely. This condition of things is prone to occur in hyperchlorhydria; it is the cause of much discomfort; the condition tends to increase, and the symptoms to become more severe. When recognised early, the checking of the hyperchlorhydria is followed by restoration of tone, and by recovery from the ptosis. With the recurrence of the hyperchlorhydria the ptosis, however, recurs also, with its accompanying miseries. In a patient whom I have seen from time to time during the last three years there have been several outbursts of hyperchlorhydria with accompanying ptosis, and a prominent feature in the case has been that with the recurrence of the hyperchlorhydria extraordinary quantities of fluid are poured out into the stomach. He has been entirely without symptoms for as long as eight months. When he is free of gastric symptoms there is a relaxation of dietetic rules; he is a busy and a hard-working man, who will

travel to London one night, transact business during the day, dine, and return to Edinburgh by the night mail, with the result that his symptoms may return. He can manage himself so well that he often cuts the attack short; but, if he does not succeed, I see him again, and I have warned him that these recurring attacks may leave his stomach in such a condition that he will ultimately require a gastro-enterostomy. In this case it is probable that there are pyloric or duodenal adhesions intensifying the mechanical difficulty, by keeping the pyloric outlet in a fixed position, and that consequently there is no movement of accommodation on its part. He is not desirous of having an operation, as he has a friend who, in spite of a gastro-enterostomy having been performed, has still to exercise caution in his dietetic habits.

This leads me to point out to you that this condition of ptosis of the lower border is more intractable, not only where there are adhesions or narrowing from old healed ulcer, but also in cases where there is congenital narrowing of the pyloric outlet, or where the pylorus is abnormally high in position. I saw a case of the last description in a medical man, who had suffered so long and so severely from digestive miseries that I thought it quite likely that his pylorus, which I determined was high in position, might be fixed in its high position by old adhesions, and yet at the operation it was shown that none existed. A gastro-enterostomy was, however, done, with marked benefit. In this patient, from the position of the pylorus to the lower border of the stomach, it was clear that the stomach could not empty itself properly, and the new opening afforded a channel which presumably enabled it to do so. Another patient, whom I saw some months ago, had a like sagging of the lower border, which had not yielded to treatment, and who also had symptoms indicating duodenal difficulty from previous duodenal lesion. I at once recommended gastro-enterostomy. At the operation old duodenal lesion with adhesions was found of such a kind that would have made an attempt at medical treatment ridiculous. She also did well, and was much benefited by the gastro-enterostomy. This patient had been diagnosed a year before as a neurasthenic; but blunders of this kind are no more to be taken as the standard of the physician's work than the blunder of opening the abdomen unnecessarily is to be taken as the standard of the surgeon's capacity. The aim of the physician ought ever to be to reduce the margin of error in internal diagnosis to vanishing proportions.

6. Gastropptosis.

In gastropptosis there is a falling down of the whole stomach, including the lesser curve. The greater part of the stomach is much below the level of the pyloric outlet, so that it is never emptied by way of that channel. I need not dwell upon the symptoms; there is no condition easier of diagnosis, and no condition which more lends itself to such methods of examination as I demonstrated to some of you in Ward 27 the other day. The precise limits of such stomachs can be traced, without either artificially blowing them up, or submitting them to x-ray examination after loading them with bismuth. I do not believe that we have any means of curing the inevitable symptoms of such a condition but by gastro-enterostomy.

CONCLUSION.

In conclusion, I would summarize the position by saying that, if chronic gastric and duodenal ulcer are excluded, the question of operation hangs upon, *first*, the determination of the position of the inferior border of the stomach to the pyloric outlet; and, *secondly*, upon the prospect of being able to restore the sagged lower border. The first of these conditions is, in my experience, easy of attainment; the second depends upon there being no natural or acquired barrier to the passage of food through the pylorus or the duodenum. Upon the accuracy of the answer to this last problem hangs the accuracy of the prognosis, and it will determine the important question whether it is wise or not to try to restore a proper relation of stomach axis to outlet. It must, however, be clearly stated that many cases are most amenable to suitable medical treatment. The attempt, however, to cure all cases of ptosis—either partial or complete—can only end in loss of prestige to the physician. Physical laws hold here as elsewhere; and there are physical barriers to the

emptying of some stomachs by their natural outlet—it is so impossible that it is simply fatuous to try to bring it about. Accuracy of diagnosis is not unattainable, and on such accuracy or inaccuracy the physician's prestige naturally depends. If he attempts to cure what correct diagnosis would tell him he cannot cure, and the patient is driven by force of his misery into the hands of the willing operator, only one result can follow—namely, discredit to himself and the art he professes.

REFERENCE.

¹ W. Russell: Gastric Hyperacidity—Hyperchlorhydria: Its Nature, Diagnosis, and Treatment. *BRITISH MEDICAL JOURNAL*, April 18th, 1903. ² Bolton, *Practitioner*, September, 1910, p. 297; *Journ. of Path. and Bact.*, April, 1910, p. 418.

THE SCIENCE COMMITTEE OF THE British Medical Association.

REPORT CXXIII.

THE DISTRIBUTION OF APPENDICITIS, WITH SOME OBSERVATIONS ON ITS RELATION TO DIET.

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DURING the past few decades, while attention has been so forcibly focussed on appendicitis, much has been written and many theories advanced as to its etiology. While there can be no doubt that infection is the final cause of the clinical condition met with, the underlying factors predisposing to this infection are as yet undetermined. The influence of age and sex are well known, but opinion is divided as to the influence of nationality or diet. Accuracy of diagnosis has until recently considerably influenced the statistics which have been available, and, further, only within the past few years has the disease been mentioned in national statistics. Even yet in some countries such information is not obtainable. These conditions explain the diversity of opinion which is held as to its frequency.

Sporadic references are to be found in the textbooks, the American authors stating that it is comparatively rare in the negro.

Murphy¹ states "Lucas-Championnière (1904), in an analysis of 22,000 patients among Roumanian peasants, found but 'one case of appendicitis'; they live mostly on vegetables. The Roumanians living in the city, chiefly on animal diet, are frequently affected—1 case of appendicitis among every 221 patients. The vegetarian diet of the Japanese and of the Indians of India seems to protect them against appendicitis."

That these are not the only striking evidences of the influence of diet is shown in the evidence from other countries mentioned below.

Hare² of Philadelphia states that "appendicitis is more frequent in the well-to-do than in the poorer classes." A comparison of the Poor Law hospitals with others in this country shows the same difference in incidence.

In observations previously recorded³ I have shown that intestinal sand and appendix concretions are allied in chemical constitution, and also that abnormal bodies sometimes found in the intestine and producing symptoms of disease are formed of compounds of saturated fatty acids.

These observations led to the conclusions that there are conditions in the intestines in which certain soaps or other insoluble compounds of fatty acids are not absorbed, or are excreted by the intestinal mucous membrane, and that with the presence of these conditions certain diseases of the alimentary tract are related. The fats and soaps found in these conditions were compounds of saturated fatty acids. The richness of these bodies in calcium and fatty acids, which are constituents of the excretion of intestinal mucous membrane, point to their originating in the intestine as a result of some excess in excretion or defect in absorption.

These calcium soaps, when formed in the intestine in excess, produce intestinal sand, and associated with this there are symptoms of colic with, under some circumstances, an associated form of mucous colitis, or, as the French authors call it, muco membranous entero-colitis.

On examination of the concretions, the frequency of which in appendicitis is undoubted, I have found that they consist of the same calcium soaps of saturated fatty acids as occur in intestinal sand. They are unquestionably formed in the appendix.⁴ Kelly states⁵:

The histological examination shows that the most important factor in the formation of the calculus is the mucus secreted by the glands of the mucous membrane. The mucus which is deposited in layers round the central nucleus becomes desiccated, and the lime salts are deposited secondarily. Frequently fragments of epithelium, a few leucocytes, and altered blood are found in the different layers. Ribbert noted that in favourable specimens stained with Weigert's fibrin stain, the outer layers of mucus may be seen to be directly continuous with the glands of Lieberkuhn. In one case, besides the two large enteroliths in the canal of the appendix, I found three smaller bodies embedded in the mucous membrane, which had evidently developed within gland lumina.

By suitable methods of staining I have shown that the above-mentioned soaps are found in this so-called mucus in the appendix, and further that they are actually deposited in large quantity in the submucosa of the appendix, thus cutting off the blood supply to the mucous membrane, and so rendering the invasion by organisms an easy matter.

The cause of these pathological changes is possibly an excess of saturated fats in the foodstuffs, giving rise to the formation of calcium soaps of more saturated fatty acid in the mucosa and submucosa than is normally the case. These soaps are not so easily absorbed or excreted as the soaps of the unsaturated fatty acids, and therefore act as foreign bodies in the wall, and at times in the appendix as a concretion.

The nature of the fat in the food determines to some extent the nature of the fats in the tissues. The absorbability of the various forms of fats is very different; for example, olive oil (containing unsaturated fatty acids) is absorbed to the extent of 98 per cent., while of stearin (saturated fatty acid) only from 9 to 14 per cent. is absorbed. Mixtures of the saturated and unsaturated compounds are absorbed in varying degree, depending upon the amount of unsaturated fatty acid present.⁶

In reference to the above investigations, I have endeavoured to find if the nature of the food fats has any relation to these pathological processes, whether an excess of saturated fat (as in animal fats, particularly beef and mutton) in the diet is associated with an increased incidence as compared with unsaturated fat (as in olive oil, butter, fish oil, etc.).

The difficulty of an inquiry such as this is obvious, and few deductions can be drawn from the statistics obtained. One point seems to be established, and that is, that diet plays a definite part in the production of the disease, and further, that animal food seems to be associated with an increased number of cases.

The following statistics and statements are quoted from a large mass of correspondence, and are of interest apart from any deductions which may be drawn from them. For comparison it may be stated that the death-rate in England from appendicitis is about 90 per million persons living. The names of the gentlemen to whom I am indebted for the information and statistics given below are mentioned in each case.

ABYSSINIA.

Dr. Frank Wakeman, Medical Officer to the British Legation, writes:

During a residence of eight years or so in this country and a practice among all classes of the population, I have never come across a single case of appendicitis, nor have my colleagues, with whom I have at one time and another discussed the subject, ever met with what could be described as authentic cases of the above malady. It is generally believed that this complaint, as well as enteric fever, does not exist in this country. Sporadic cases of gastric fever (so-called) are occasionally seen, but in no instance have the characteristic signs and symptoms peculiar to appendicitis ever been observed, and the disease usually runs a short course seldom attended with fatal results.