is another and perhaps less familiar example of the sort of rapid representation of clinical features that may sometimes be witnessed on the moribund heart. It is taken from a paper by Waller and Reid,[9] and shows the rapid development of true pulsus alternans ending with ventricular fibrillation in the case of the excised and therefore moribund heart of a cat.

Here is another figure (Fig. 11) taken from the same paper showing the electrical indications of *a.-v.* dissociation, and another (Fig. 12) showing the electrical indications of true alternation of the ventricular beat. But the hour has struck and I must reserve any further consideration of *a.-v.* dissociation for the next lecture.

ALIMENTARY TOXÆMIA:

A SUMMARY AND REPLY.[1]

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IT is obviously impossible to present in a quarter of an hour any adequate summary of a debate in which 60 speakers have taken part, but the speeches of the pathologists, chemists, and bacteriologists have probably interested the society most, for in an inquiry as to what clinical symptoms can with justice be attributed to alimentary toxæmia the first thing is to understand how the poisons are formed and how it is that they affect the body. There has been much said to show that it is not the formation of poisons which chiefly matters. Probably they are continually being formed from the contents of the alimentary canal, or they might even occur as endotoxins derived from dead bacteria, but no harm follows so long as the constantly active defences of the body against such poisons are in full play. But the liver may fail us, the thyroid may fail us, mechanisms in the intestinal wall may fail us, or other unknown defences may fail, and then poisoning symptoms will occur. It will be a great step forward when, with the help of experimental pathologists, we are able to say which cases of supposed alimentary toxæmia are really caused by disease of the liver, disease of thyroid, or disease of some other organ, as the case may be, or which are due to the formation of abnormal poisons or to an excess of normal poisons in the alimentary canal. Then sometimes it may be that the symptoms ascribed to alimentary toxæmia are really due not to the formation of more poison but to the fact that the wall of the bowel being diseased the poison is absorbed more readily than in health, or, again, some alteration in the intestinal contents may render rapid solution and consequent absorption of the poison easy.

Considering next the bacteria in the intestine, it is necessary to point out that if a person is poisoned by absorption from the intestine it does not follow that the poison is produced by bacteria; it may be that it is one of the normal results of the splitting of proteins, but that in all healthy subjects the poison is destroyed by the liver, which, however, is not performing this normal function in the person suffering from poisoning. We only know for certain that definite micro-organisms lead to poisoning when specific micro-organisms introduced from without cause specific diseases—e.g., in the case of typhoid, dysentery, cholera, and Gaertner poisoning; but many of those who have taken part in this discussion strongly suspect that under certain circumstances the micro-organisms constantly present in the alimentary canal lead to poisoning. But there is, I think, a consensus of opinion that the phrase "alimentary toxæmia" should be limited to the absorption of chemical poisons produced in the alimentary tract, and should not include the passage into the blood of bacteria themselves, although this probably occurs more often than is usually thought.

Which micro-organisms originate symptoms that are commonly included under the term alimentary toxæmia we do not know, even in comparatively simple cases. For example, we do not know the micro-organism which is the cause of enterogenous cyanosis; nor do we know whether, if the symptoms are not due to failure of protection against poisons, they are due to excessive number of bacteria, unusual supply of material for their growth, or absence of bacteria which are antagonistic to them; nor do we know whether the bacteria form the poisons from their medium, from their intrinsic metabolism, or from endotoxins liberated at their death. Although it has clearly appeared that we have a considerable knowledge of the bacterial flora of the human alimentary canal, it must be confessed that this discussion has shed very little light on the bacteriology of alimentary toxæmia originating in the gastro-intestinal tract, but we know more about that of pyorrhœa alveolaris, although even here we often only surmise which is the offending organism by the rather unsatisfactory method of the opsonic index. Probably among the intestinal bacteria the bacillus aminophilus intestinalis and its allies will most repay for the research, for much work has been done on them, but it certainly apppears that much of the statements of the French school on the intestinal flora need serious revision.

As the subject of this debate has been alimentary toxæmia it was to be hoped that from it we should learn something definite as to the toxins concerned. Unfortunately we have not. It is well known that the view of the Metchnikoff school is that poisons are manufactured in the intestine, especially the large, by micro-organisms, and these poisons produce the symptoms commonly comprehended in the term alimentary toxæmia, and that indol is one of the most important of these poisons, but although that view has been upheld by some speakers it has been severely criticised. More than one authority on bacteriology thinks it gratuitous to regard indol as the main toxic product of putrefactive organisms, and tells us that the conclusion drawn by Distaso that the flora of constipation is an index of intoxication is entirely unsupported by the evidence he adduces. I must confess it seems to me that the case for indol has not been proved, and that there are serious defects in the work of the French school on the bacteriological and biochemical properties of the intestinal flora. If the debate has done no other good it has helped to make widely known that much of the bacteriological and biochemical work which has been done upon the flora of the large intestine and the poisons found there does not satisfy the standards of scientific accuracy. But there is a general impression that the poisons of alimentary toxæmia may in many instances be comparatively simple chemical bodies derived from the proteins of the food. It is known that the ammonia present in the portal vein is derived from putrefaction in the large intestine. Tyrosine and histidine may be converted into poisonous substances by putrefactive bacteria, and experiments have been quoted which suggest that when certain of these poisons are absorbed arteriosclerotic changes in the vessels and disease of the kidney may follow. Confirmation of these experiments will be looked forward to with great interest. Of the poisons formed in the alimentary canal probably the study of derivatives, of tyrosine, leucine, and histidine will most repay further investigation.

In the clinical part of the discussion the dental surgeons who contributed to it seemed to be on surer ground than other speakers. Disease of the gums leading to periodontal disease is by far the most fruitful source of oral sepsis. Large numbers of people owe ill health to it, and it is often overlooked, and especially we are inclined to forget that mouth-breathing in children leads to persistent marginal gingivitis, and that this disease is more intractable and serious in its results in those who are mouth-breathers, from which it follows that not only must the teeth be treated but often it will be necessary to treat nasal obstruction also. Different speakers have laid a varying amount of stress on the use of autogenous vaccines. Probably the most important part of the treatment is local, and here much judgment is required. Some remove teeth needlessly, some do not remove enough of them, but in the more severe examples the intelligent use of autogenous vaccines may be of distinct help. The question has been raised as to whether in a few cases pyorrhœa alveolaris is not secondary to a toxæmia arising from some parts of the alimentary tract. It is quite possible that this may be so, or, at any rate, that such local pyorrhœa is increased by the fact that the general resistance of the body is lowered by an alimentary toxæmia of other origin, but obviously the question can only be settled by a discussion of each case in

[9] Philosophical Transactions, 1885. The examples referred to in the text are Tracings 1, 2, 3, and 4 of that paper, not here reproduced.

[1] A paper read at a special meeting of Fellows of the Royal Society of Medicine on May 7th, 1913, at the close of a debate on Alimentary Toxæmia.

which it may be raised, and such suggestions may do harm if they lead to a widespread belief that the local treatment of the disease of the gums is unimportant. Mention has been made of the many ways in which it is conceivably possible that pyorrhœa alveolaris might affect the general health, and it is clear that the opinion of most speakers is that nearly always it is by direct absorption from the diseased gums. Further, it must not be forgotten that the tonsils may be the source of chronic toxæmia.

It appears that in this country not much attention has been paid to the relationship between alimentary toxæmia and diseases of the eye, but this discussion has been of great use in directing attention to the question. Certainly some diseases of the eye may be due to pyorrhœa alveolaris. What others are due to other forms of alimentary toxæmia can only be decided by future clinical observation which avoids the error of ascribing changes to alimentary toxæmia because no other cause can be found, but the extremely interesting question as to whether failure of accommodative power may not often be due to intestinal toxæmia needs further investigation. If the remarkable figures quoted by one speaker are confirmed a great advance in our knowledge will have taken place.

It is generally allowed that the mere absorption of a poison introduced from without—e.g., morphine—from the alimentary canal is not enough to constitute alimentary toxæmia, and the striking urticaria which follows the ingestion of shellfish, copaiba, &c., in some persons is most likely an example of anaphylactic phenomena following the absorption of foreign proteins, and therefore not an instance of alimentary toxæmia. The purpura often associated with cirrhosis of the liver is probably an alimentary toxæmia in which the poison absorbed from the intestine is able to act because the protective influence of the liver is, owing to its disease, in abeyance. Henoch's purpura may be an example of alimentary toxæmia, but we really know nothing of its pathology. The dark-blue tint of the skin in enterogenous cyanosis is almost certainly due to alimentary toxæmia, and so may be the brown cutaneous pigment so often seen in those who have disease of the intestines. It seems as though increase of knowledge will show that several abnormalities of the skin may be ascribed to toxins formed in the alimentary canal.

We now pass to the most controversial part of the subject. It has been urged by several speakers that in consequence of the erect posture of man the intestines tend to drop; that to overcome this peritoneal bands are evolved; that they produce kinks; that these lead to stagnation of the intestinal contents; this leads to increased putrefaction; this to the formation of poisons, and that these, passing into the general circulation, cause definite symptoms and predispose to many diseases. Observers are by no means agreed as to the frequency with which these bands are found, nor, it is said, are they evolved as a result of the erect posture of an individual, because they may be found in the human fœtus, and are in reality mere expressions of a normal and healthy fœtal process. The question will, it seems to me, have to be solved by anatomists. Are these bands to be found in all animals in whom the body is usually vertical—e.g., apes, gibbons, and penguins? Are there evidences of them in animals whose bodies approach the vertical—e.g., giraffes? Are there what might be called reverse bands in bats that spend so much of their time head downwards? Here I might anticipate and point out that if these bands are present in all animals that adopt the erect attitude it would be of great interest to know whether such animals have intestinal stasis.

Passing to the question of kinks, it seems from listening to this debate that those who have laid great stress on an ileal kink as a cause of intestinal stasis have not estimated at its full value the strength and activity of the ileocæcal sphincter, and that much of the supposed holding back of the intestinal contents at the end of the ileum is normal, and that when abnormal it is often due to spasm of the ileocæcal muscle.

Many of the statements about kinks and stasis claim support from X ray examinations after bismuth or barium sulphate, but we have been told that sometimes food is given too soon after a bismuth meal when we wish to know if there is gastric stasis; that sometimes so much bismuth is given that its weight may invalidate our observations; that allowance is not made for normal variations in the average time taken in the passage of a bismuth meal, anything differing from the average being taken as abnormal; that sometimes the bismuth mixture lags in the stomach because it is nasty, although in the same patient it will leave the stomach normally if it is palatable; that the so-called writhing of the duodenum is nothing else than the normal peristalsis and segmentation rendered more clearly visible than usual by the great quantity of bismuth-containing chyme present; that the X rays do not show the duodenum to be dilated in cases of duodenal ulcer; and that many of the kinks which the X rays are supposed to show depend upon the fact that the X ray picture is taken in a single plane. It is clear that as the X ray work on this subject has been so severely criticised it will have to be repeated by independent observers before we can decide upon its value.

Those who believe that evolutionary peritoneal bands often lead to intestinal stasis because they produce kinks, especially a kink at the last part of the ileum, think that as a further result a kink forms at the end of the duodenum; this and the stomach dilate, and either may become ulcerated. This debate has not provided any statistical evidence to show in what proportion of sufferers from duodenal or gastric ulcers such kinks are found, nor has any experimental evidence been brought forward to show that in animals such ulcers form as a result of an artificially-induced ileal kink; and we have heard one speaker say that intestinal stasis never leads to gastric stasis, nor to duodenal kinking, dilatation, or ulceration, and that duodenal ulcers are associated with an unusually rapid passage of chyme out of the stomach and through the whole of the small intestine. It seems that the onus of proving their contention lies with those who believe that the above-mentioned phenomena follow intestinal stasis.

I do not think that in a short summary such as this anything can with advantage be said about the more remote effects—e.g., tubercle, cancer, rheumatoid arthritis, arterio-sclerosis, disease of the heart and kidney—which have been ascribed to intestinal stasis, for up to the present no statistical or experimental evidence has been brought forward for such a view. It may turn out to be correct. I hope it will, for I should like to see such an extremely brilliant suggestion shown to be true; but up to the present no adequate evidence has been brought forward for this supposition, which, if it is to be accepted, must have in its favour not one or two isolated cases, or a mere expression of opinion, but a large mass of incontrovertible experiments and statistics adduced by more than one observer. But whether or not future observation proves its truth there is no doubt that we owe a debt to the author of it, for if it is correct it marks a new era in medical thought, and even if the evidence for it fails it has made us think hard.

Nor do I think that time allows me to say much about treatment. We have seen that the ways by which intestinal poisons may be formed are so various, and the faults of our mechanism which may allow them to get into our bodies are so numerous, that proper treatment is in our present ignorance often impossible—indeed, almost our only treatment, surgical or medical, is the very primitive plan of keeping the bowels well cleared out. Sometimes, indeed, nature does that for us, for, as has been pointed out, severe alimentary toxæmia may exist with a diarrhœa which keeps the intestines almost empty. On the other hand, many people whose bowels are rarely open have not alimentary toxæmia. It is agreed that in the vast majority of cases medical treatment suffices; and what has been said about treatment in this discussion has come chiefly from the surgeons. Removing the colon has been mentioned, but a surgeon of wide experience tells us he has never seen a case in which he considered it justifiable. Short-circuiting the ileum into the sigmoid, too, has been discussed. Some say it succeeds, but it is clear it often fails to cure, and some surgeons prefer the simpler operation of appendicostomy. But if the cases that now seem to some to justify surgical treatment had been treated in the first stages by proper medical means surgical interference would not be necessary, so that, when this is widely appreciated, cases ought never to become so ill that surgical treatment will be contemplated, and we may hope that one result of this discussion will be that we shall keep "the drainage scheme" of our patients in sufficiently good order as to render surgical interference unnecessary.

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