The bacteria in the alimentary canal, in virtue of the high fermentative activity which many of them possess, carry out protein cleavage to a point beyond that which the body demands for its own nutritional needs, and certain of the products which thus arise are actually harmful to the host. But this has always been so, ever since bacteria took up their residence in the alimentary canal of animals, and it has been necessary for animals to evolve some means of chemical antagonism to neutralize this harmful effect. We are still in comparative ignorance of the precise chemical mechanism by which this permanent menace is met in health, but that it is so met is certain, for in health we remain untouched. It is conjectured that one of the functions of the thyroid gland is to deal with this matter. A number of observations by different workers have shown that the activity of this gland is excited by the presence of bacterial toxins, and it has been asserted by certain observers that thyroidectomized animals are abnormally susceptible to infective processes. It is possible that the antitoxic functions of the thyroid extend to chemical poisons other than bacterial toxins. Be this as it may, it is clear that the body in some way compensates the ordinary activities of bacteria in the intestinal tract, so that no harm results. Only when such bacterial decompositions are excessive, and when faecal retention affords unusual opportunities for the absorption of the products, are symptoms manifested.

Thanks to the labours of chemists, our knowledge of the protein decomposition products is fairly extensive, though our knowledge as to their toxicity is incomplete. Most of them appear to be of feeble toxic powers, but it must not too hastily be assumed from animal experiment that such substances as, for example, indol are so harmless as they would appear. I am very far from agreeing with the recent hypothesis suggested by Metchnikoff as to the rôle of indol and allied substances in the causation of arterial degeneration, for the evidence put forward seems to be wholly insufficient. But we can to some extent measure the absorption of indol by the excretion of indican in the urine and correlate its excess with clinical phenomena. The excessive indicanuria in conditions of mental depression and melancholia, for a knowledge of which I have to thank Mr. Mackenzie Wallis, may be of great pathological significance, and may outweigh any negative evidence derived from animal experiment.

There is also a possibility, for the suggestion of which I again owe my thanks to Mr. Wallis, that excessive bacterial activity in the intestine may have its negative as well as its positive influence. Thus, still to take indol as an instance, it is known that this substance is derived from the tryptophane element in proteins. If tryptophane is an important element in tissue nutrition, it may be that excessive splitting up of this substance in the intestine by bacteria may lead to tissue starvation in an important detail. An element in a supposed alimentary toxæmia may possibly be due not so much to the positively toxic action of the derivative as to the absence of the needful substance from which it has been derived. I must leave such considerations to those who follow in this discussion.

Bacteria, however, may do more than provoke merely fermentative changes in the medium in which they are growing. They produce, in virtue of their own intrinsic metabolism, substances of unknown chemical composition which are deleterious to their hosts, and which we must be content to term "toxins" in the absence of more exact knowledge. It is established that a very few—bacteria produce soluble toxins which pass out into the surrounding medium without any disintegration of the bacterial body. These are the pre-eminently "toxic" bacteria, of which the diphtheria and tetanus bacilli are the commonest examples, and they lie outside our present subject. Indeed, almost the only essentially toxic bacterium which could be dragged into the discussion is van Ermengem's *B. botulinus*, present in certain ham poisonings, though *B. pyocyaneus* might perhaps also be included. But the great majority of pathogenic bacteria, and practically all those which are normally saprophytic in the alimentary canal, form no soluble toxins; so far as they possess toxic properties, these reside in the bacterial protoplasm itself and are liberated only on its dissolution—so-called "endotoxins."

Bacteria are, of course, constantly undergoing dissolution in the alimentary canal, and one cannot dispute the possibility of harmful effects from such endotoxins on absorption. But we are in almost complete ignorance of the extent to which alimentary toxæmia rests on such a basis. So far as concerns the normal intestinal flora, I should be disposed to believe that poisoning by bacterial endotoxins plays a comparatively negligible part in the process. As soon as evidence is brought forward showing this to be the case I shall be ready to accept it, but so far there is none, and it is probable that the endotoxins of most of the normal inhabitants of the alimentary canal are of very feeble virulence, though those of the colon bacillus may perhaps prove of some importance. This does not, of course, apply to cases of infective enteritis, in which the absorption of bacterial toxins plays a large part in the production of the constitutional symptoms.

To sum up what I have said as to the part played by bacteria in alimentary toxæmia, it seems to me reasonable to lay down the following propositions:

1. In an important group of cases the poisoning is by foreign proteins, as such, the defect lying in the liver, which ought to shield the body from their effects. With this bacteria have, as a rule, nothing to do.
2. The main effect of bacterial activity in the production of alimentary toxæmia lies in their ability to carry out protein cleavages beyond the capacity of the ordinary digestive ferments, with the formation of products which, when in excess, the body is unable to neutralize.
3. There is at present little evidence that true toxins, derived from the ordinary flora of the gut, soluble or intracellular, play much part in alimentary toxæmia.

ALIMENTARY TOXAEMIA : ITS SYMPTOMS AND TREATMENT.

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THE doctrine of intestinal autointoxication was originated by Bouchard, and has formed the foundation for far-reaching speculations. There is little evidence that the normal products of intestinal fermentation and putrefaction are capable of producing disease, and if acetic and butyric acids, indol, skatol, and the like are harmful their effects must not be exaggerated. Sulphuretted hydrogen is only present normally in traces, and much larger quantities may be injected into the rectum without hurt, although admittedly this gas is more harmful when formed in the small intestine or the stomach. Ammonia is converted into harmless urea, while volatile fatty acids and acetic and butyric acids only produce local stimulation, even when present in quantity. Acetone, diacetic acid, and beta-oxybutyric acid are present in the urine, together with various symptoms of disturbed digestion, but this does not prove that the symptoms are due to them. The first two are found in the urine of starving animals with no digestive disturbances, and merely indicate the breaking down of tissue protein. Ptomaines may be present in excess in intestinal contents without any toxæmic results, as, for example, in cystinuria. On the other hand, there is a widespread belief, not without evidence in its favour, that many diseases depend upon the absorption of poisons from the intestine, either introduced with food or formed from it by the fermentative or putrefactive processes in the alimentary tract.

FOOD POISONS.

In former days food poisoning referred to the accidents that result from the ingestion of substances rendered harmful by the poisons contained in them, but modern research has added to these the effects of microbes and the toxins due to their activity. These last are exclusively produced from animal food; the danger of vegetable food depends solely upon its natural poisons or its accidental contamination by microbes in the water with which it has been washed or in the dust lying on its surface.

Vegetable Food Poisons.

Among the best known poisons from vegetables are those contained in many kinds of mushrooms, and the poisonous effect varies not only with each kind, but with season and place, some being poisonous at one time of the year and not at others, some dangerous in warm countries but harmless in colder climates; some of these poisons are destroyed by boiling, the water in which the mushrooms are cooked becoming highly poisonous.

Phalline, the poison of *Amanita phalloides*, determines destruction of the blood cells and causes choleric symptoms, with vertigo, delirium and somnolence that may pass into coma, convulsions, and death; or final fatal icterus may occur after a remission of the symptoms has taken place, while should the patient recover, convalescence is slow and the myocardium and kidneys may remain damaged.

Muscarine, the poison of *Amanita muscaria*, causes nervous symptoms resembling alcoholic intoxication with cramps, convulsions, contracted pupils, rapid small pulse, and death in collapse, or recovery may follow a prolonged sleep.

Flour is sometimes poisonous by the accidental admixture of the seeds of tares, cow-wheat, smut, ergotized rye or vetch with the meal of wheat, maize or rye. Adulteration from tares and cow-wheat gives rise to nausea, vomiting, colic, disturbed vision, vertigo, somnolence, and lassitude, which disappear in a few hours. Cow-wheat may also produce convulsions. Smut contains a poison called "githagin," which may determine intense colic with diarrhoea and haemorrhage from the bowels. Ergotized rye causes nausea, vomiting, lassitude, with constipation or diarrhoea, and subsequently local symptoms such as cramps of certain muscles or gangrene of the fingers and toes; vetch poisoning induces spasmodic paraplegia with disorders of sensation and tremors.

Potatoes sometimes produce poisonous symptoms from the presence of solanine, causing slight vertigo, headache, vomiting, dilated pupils, and rapid action of the heart.

Animal Food Poisons.

The putrefaction of animal food leads to the production of (1) ptomaines and (2) leucomaines. The first are formed by putrefaction, the second by the excessive destruction of protein.

Certain poisons are developed spontaneously and apart from microbial infection—for example, oxybetaine from meat and putrefying bones, choline from herring pickle, muscarine from decayed fish, methylgadinine from putrid horseflesh, and ptomatropine from salted sturgeon.

On the other hand, the *Bacillus botulinus* and other microbes of the type Aerttryck and the type Gaertner cause a poison, botulin, in putrid ham. Other poisons are formed by the paratyphoid bacillus, by enterococcus and proteus, while the *Bacillus coli* may under certain circumstances enter the blood and make the meat poisonous—as, for example, in luted animals. Meat preserved in various ways, especially in cold storage, seems to be liable to undergo poisonous decomposition. This is also true of gelatine, cream, and Rouennaise ducks; these last are killed without loss of blood by puncturing the medulla, and are then beaten so as to effect the diffusion through their tissues of anything absorbed from the intestine. Certain fish, the herring and the conger, and certain roes are at times poisonous; mussels contain a poison which has been called mytilotoxine (Virchow and Brieger) and mytilocongestine (Richet), while oysters sometimes contain similar poisons. It is not known how these poisons are produced; by some (Thesen) it is thought that they are formed in sea water. Young veal is highly poisonous when the cow has suffered from septic fever or puerperal metritis. The meat of animals suffering from septic diseases, metritis, pneumonia, and typhus is undoubtedly poisonous. Hunted game is sometimes also poisonous from the intestinal microbes entering the blood.

Preserved meat, potted meat, sausages and the like, may be infected before being packed or during preparation, and in hot weather the microbes develop and produce poisons. Eggs may be contaminated in the oviduct, and when used in confectionery—especially when mixed with such favourable media for the cultivation of microbes as sugar, milk, and gelatine—may develop poisons. Cheese is also occasionally the seat of poisonous putrefaction.

There is little doubt that certain foods become poisonous from the absence of some principles normally present, but which have been removed or disappear when the food has been kept too long. Of these beri-beri, although only known to Western medicine in recent years, is the example in which this relation has been most clearly established. The belief that it depended upon some dietetic defect was maintained as long ago as 1878 by Wernich, supported later by Van Leent, and the practical value of the doctrine was demonstrated by Takaki, who, by the reform of the diet of the Japanese navy, wholly eradicated the disease from that service. But it was Braddon who was the first to show the connexion between the consumption of white rice and the prevalence of the disease, and the conclusive proof has been worked out by Fletcher, Stanton, and Fraser, who have shown that rice completely denuded of its pericarp, if used as an exclusive diet, causes beri-beri, while the disease may be prevented by mixing a proportion of the "cleanings" with the diet, and conversely that the disease does not arise when imperfectly husked rice is consumed.

Although the position of pellagra as a food disease is disputed, the belief is general in the United States, where its incidence has extended widely during the last few years, that this spread is due to the fact that maize, which was formerly consumed where it was grown, is now transported long distances and stored for use. The cases that have been observed in Scotland are also thought by some to be due to an analogous change in oatmeal.

Scurvy or scorbutus has been long recognized as related to insufficient diet, and should be included among diseases due to dietetic error or defect, for the suggestion that it is caused by a poison formed in tainted meat receives small support from the experience of those who have seen most of the disease. This view impressed itself upon the mind of Captain Robert Scott, as in the second year of his former Antarctic expedition, when he had plenty of fresh seal meat, the disease disappeared; but fresh meat contains the necessary principle. There is strong evidence of the value of varied diet, of fresh or preserved vegetables, and lime juice; the use of the last article coincided with the practical disappearance of the disease from the British navy.

Barlow's disease is another result of dietetic defect, as, like scurvy, it depends upon the want of some principle usually present in fresh milk but absent in patent and preserved foods. Cases have been alleged to occur in children who were fed on breast milk; but the breast milk may have been defective owing to the impaired health of the mother.

Speaking generally, the results of food poisons may be classified into slight, medium, and severe: (1) In slight cases there may be only some digestive trouble, nausea, vomiting, colic, headache, pain in the back, with recovery in two or three days, or there may be urticaria without digestive trouble, or simply diarrhoea. (2) In medium cases, in addition to the symptoms already described, there may be a persistently dirty tongue, slow convalescence, a disposition to relapse, accompanied by constipation and enfeebled digestion lasting for weeks. (3) Severe cases may present (a) the typhoid state, with high temperature, epistaxis, diarrhoea, gurgling in the right iliac fossa with enlarged spleen, such cases often giving rise to considerable difficulty in diagnosis; (b) the choleric form type, with violent vomiting and purging, pains, cramps, dyspnoea, thready pulse, sweating, purpuric and scarlatiniform eruptions, low temperature, collapse and perhaps death, or a reaction with return of diarrhoea and vomiting, followed by gradual improvement, slow convalescence, and prolonged weakness; (c) the icteric type, characterized by jaundice which may be malignant in form and results especially if the kidneys are diseased. Many doubtful cases of disease are due to these toxic infections.

Botulism is a special form in which, after two or three days of colicky pain, vomiting and diarrhoea set in with dryness of the skin and mucous membrane. There may be constipation. External and internal ophthalmoplegia may occur. There is no fever, but there is difficulty in deglutition owing to the dryness of the pharynx. These symptoms may gradually disappear and slow convalescence follow, or death may result from cachexia,

bronchopneumonia, or asphyxia from food sticking in the pharynx.

In Captain Ejnar Mikkelsen's book, *Lost in the Arctic*, which describes his adventures and hardships during his expedition to Greenland, he tells how, when hard pressed by hunger, he and his companion were driven to eat the liver of freshly-killed dogs, and suffered in consequence. Their symptoms were peculiar, as they consisted of an uncontrollable desire to sleep, their sleep lasting ten or twelve hours; when they awoke they suffered from a splitting headache; some days later the skin peeled, and Mikkelsen says his companion Iversen had "big sores all over his body." In an earlier part of the book it is stated that a dog was made ill by eating dog's liver, but somnolence was not a symptom. The livers, when eaten by Mikkelsen and his companion, were cut in slices and "fried in water," so that the meat was imperfectly boiled. The poison was probably toxic albumose, which was all the more active as the meat was freshly killed and only partially cooked.

Sir Lauder Brunton, Dr. A. C. Farquharson, and others have drawn attention to the symptoms produced by food poisons which in many cases resemble the action of a paralysing narcotic, but Farquharson attributes this effect, not to a direct soporific poison, but to dilatation of the gastro-intestinal blood vessels with consequent anaemia of the brain.

Many skin eruptions are attributed to the absorption of intestinal poisons—for example, erythematous rashes, urticaria, pemphigus or pemphigoid eruptions, and pruritus. The effects of fish, shellfish, and certain fruit in causing erythematous eruptions are well known. Coffee may cause pruritus and sweating of the palms of the hands and soles of the feet. A hospital patient had many recurrent attacks of herpes iris, with large bullae in the mouth and pharynx and over the elbows and knees, which invariably followed a bout of beer drinking.

Cyclic or periodic vomiting, or food fever, is a condition which occurs in childhood, from the second or third year onward up to puberty, and is attributed by most writers who have concerned themselves about it to disturbances of digestion, especially the digestion of carbohydrates. It does not always cease at puberty or at the age at which puberty generally occurs. A case of this kind, under my care a few years ago, was a lad of 18, as undeveloped as a boy of 12; he weighed only 4 st. 9 lb.; his attacks of vomiting occurred every few weeks, and were accompanied by severe headaches; he was quite unable to earn his living. At the urgent request of his father his abdomen was opened and gastro-enterostomy was performed, without any definite condition being discovered or any improvement resulting. Acetonuria has been observed in this affection, but there is little likelihood that the cause is acetone poisoning.

It is doubtful whether there is any sound foundation for Nothnagel's view that certain cases of infantile marasmus, cachexia, and pernicious anaemia result from atrophy of the intestinal mucous membrane. It is possible that pernicious anaemia may result from the absorption of poisons from some part of the alimentary tract which destroy the blood, but the atrophy of the stomach or intestinal mucous membrane is probably associated or secondary (Knud Faber). The relation of pernicious anaemia to intestinal intoxication, although possible, has by no means been proved, and Schmidt says he has not met with sufficiently severe fermentation or putrefaction to support this theory. Chlorosis and leukaemia have also been attributed to intestinal intoxication, but without any adequate proof, and there are now few persons who would support Sir Andrew Clark's view that chlorosis is due to intestinal poisons, and may be cured more efficiently by purgatives than by iron.

Certain affections of muscles may result from severe digestive disorder, as in the cases of polymyositis acuta described by Senator and Albu.

Oxaluria or oxalaemia may be fairly reckoned to be of alimentary origin, for oxalic acid is introduced preformed in the food, or is the product of carbohydrate fermentation in the stomach which may occur when the hydrochloric acid is deficient; hence the old Edinburgh treatment by nitro-hydrochloric acid is well founded.

Arterio-sclerosis is one of the conditions attributed to

auto-intoxication, but the argument seems to be that, as only a small percentage of cases give a Wassermann reaction, they are not all due to syphilis; and if not to syphilis they must be due to auto-intoxication (Aufrecht), which only shows how comprehensive this doctrine may become. Indeed, Combe makes it play the same universal part that others assign to uric acid, while Tissier, in an equally wide generalization, maintains that the decomposition of animal protein in the intestine prepares a favourable soil for the growth of all pathogenic bacteria, and is therefore the source of most diseases.

It is sufficient comment on these theories to say that, however plausible, they do not rest upon a solid foundation.

POISONING BY FAECAL STASIS.

It is tolerably certain that there is little evidence in favour of the popular notion that mere faecal retention favours the production in the bowel of fermentative or putrefactive changes giving rise to poisons which are absorbed into the blood and cause various pathological symptoms.

Simple constipation does not lead to any increase of decomposition in the faeces, and infrequent evacuation of the bowels is consistent with perfectly good health and longevity. But it is a not uncommon cause of chronic intestinal catarrh which may go on to ulceration and even perforation, and it is to these inflammatory consequences that must be attributed the various symptoms which are associated with constipation. Faecal retention is not inconsistent with a daily action of the bowels, as in a case reported by Maunaberg of a public official who asserted most positively that he had always made a point of getting a daily evacuation, but who died of perforative peritonitis caused by a large number of enormous faecal masses nearly as hard as stones.

While, therefore, we may be reasonably sceptical about "copraemia," "faecal fever," and other alleged results of faecal fermentation or putrefaction, nothing is more certain than that many symptoms may result from constipation where alterations in the wall of the bowel have taken place. Nor is it inconsistent with this view that thorough emptying of the bowel should be followed by temporary relief to these symptoms, but at the same time we should expect that the simple evacuation of the pelvic colon would not effect a cure.

Without inflammatory changes the natural protective agencies existing in the alimentary canal can keep us safely from such toxins as are formed in the bowel; so long as the several organs concerned are structurally healthy the intestinal contents are practically outside the body and are powerless to injure us.

PROTECTIVE AGENCIES.

Against the long catalogue of supposed evil influences of intestinal microbes and toxins must be set the very real protective agencies by which under physiological conditions their entrance into the circulation is prevented. The general harmlessness of pathogenic microbes and their toxins in the digestive tract is supported by strong evidence. The healthy mucous membrane presents such a barrier by its relative impermeability that their diffusion takes place slowly, and time is allowed for the action of the digestive secretions which destroy the microbes and neutralize the toxins. This action is effected most efficiently in the stomach and small intestine, less so in the large intestine and rectum; but dogs and rabbits can withstand the rectal injection of cultures of diphtheria and tetanus, of which a fractional dose would be fatal if injected under the skin or into the circulation. Mere quantitative variations in the amount of the digestive secretions do not appear to interfere with this protective action, but in *achylia* infective microbes and toxins are not destroyed in the stomach, and pass rapidly into the duodenum. Even here they have to run the gauntlet of the pancreatic and intestinal digestion, which possesses almost equal destructive and neutralizing properties.

The bile has a much weaker action, its antitoxic effect being apparently due to its saponifying power as an artificial solution of soda palmitate possesses the same antitoxic coefficient. Roger attributes the antitoxic action of the bile to its power of modifying pathogenic bacteria so

that they no longer secrete toxins; but he maintains that so far from neutralizing toxins already formed, it actually increases their toxicity. Should these poisons enter the portal circulation, in spite of the destructive neutralizing or attenuating effects of the digestive secretions and the barrier of the mucous membrane, the liver itself possesses the power of arresting poisons or of converting them into harmless or less poisonous substances. Further, the intestinal mucus which coats the lining of the bowel delays absorption and prolonged contact with the mucous membrane neutralizes toxins.

Finally, the ordinary intestinal microbes are able to neutralize the toxins secreted by allied species and can inhibit the putrefactive power of others, while they can also neutralize toxins although to a much less extent than the digestive juices.

Inflammation of the mucous membrane of the bowel favours the absorption of toxins and its permeability seems to be normally greater in infants and young animals as shown by experiment. It is claimed that diphtheria antitoxin may be administered successfully to infants by rectal injection (Chantemesse) and that other antitoxins and vaccines may be so employed.

Further, it is found that various vegetable toxalbumens are not completely destroyed by the digestive juices but diffuse through the intestinal wall into the blood, and it is said that tuberculin differs in this respect from the other microbial toxins, behaving like these toxalbumens.

TREATMENT.

The treatment of these conditions cannot be discussed in detail, but the principles may be laid down.

In the first place, it is essential that food should be wholesome, that articles of doubtful nature should be avoided, that preserved foods, especially animal foods, should not be eaten when fresh can be obtained, and that as far as possible all food should be prepared by submitting it to cooking at a temperature of not less than that of boiling water. The eating of all uncooked food is attended by a certain amount of risk, and although we all run this risk at times it is unwise to eat such food when we know little about its origin or handling. For example, many English travellers suffer from eating fruit and salads in hotter countries, and even at home oysters are justly shunned unless we know their source.

In the second place, all food should be eaten slowly, properly masticated, and mixed with saliva. Defective teeth should be repaired or replaced, and the mouth kept in a healthy condition.

Provided these conditions are all fulfilled the stomach, if healthy, may be trusted to perform its share, but no doubt Trousseau's traveller was right who concluded each meal in strange places by taking a few drops of hydrochloric acid to reinforce the action of the gastric juice.

By all means let the diet be a mixed one of carbohydrate, protein, and fat, and consist of such things as are suited to the individual, avoiding excess and remembering that while every stomach can digest small quantities of most things no stomach can digest large quantities of everything. In youth amounts of food can be assimilated with advantage that in middle age tend to obesity and gout, and in later life to disordered digestion and many evil consequences. Moreover, mode of life must be allowed to regulate the quantity of our food, and as we become less active we should eat less.

Alcohol should be regarded with doubt and suspicion, for, while capable of conferring a certain amount of pleasure and at times of promoting appetite and even digestion, its general effect is to slow the digestive process and in large quantities to check it and to inhibit those protective agencies upon which our safety depends.

While due time must be given to meals and resting afterwards assists digestion, daily exercise favours absorption, maintains the circulation of the blood and lymph, prevents constipation and promotes elimination by the lungs and skin.

When alimentary toxins have been absorbed and passed into the circulation, the most rational treatment would be the administration of an antidote if we possessed one; failing that, we should try to prevent the further formation of poisons, and to favour their elimination.

Nature endeavours to get rid of the poison by setting up diarrhoea, vomiting and sweating, more rarely by diuresis, all of which may be imitated, guided, or controlled.

The means by which we may endeavour to provoke the prevention of the further formation of poisons and the destruction and elimination of those already present may be by (A) cutting off the supply of material; (B) reinforcing the digestive juices; (C) bacterial action; (D) drugs; (E) hydrotherapeutics.

A.

We seek to cut off the supply of further material for the formation of poisons by simplifying the diet. In acute conditions, after washing out the stomach, we may give nothing but hot water, or the same with the addition of a little milk or milk diluted with lime water, soda water, or barley water; in subacute and chronic conditions it is best to rely on lacto-farinaceous diet. As a general rule eggs, milk, and cheese are allowed, but some forbid even these, and give only vegetable protein, carbohydrates, and fat (Tissier). In all cases it is better that the food should be varied, well cooked, and moderate in quantity.

Fasting is a perfectly sound method of diminishing toxins if combined with copious draughts of water or mineral water (Guelpa).

Fruit "cures"—such as the strawberry cure that enabled Linnaeus to get rid of his gout, the grape cure and the like—probably act partly as fasting cures, partly by supplying a non-toxic and moderately purgative diet. The advice of Albu to take the juice of two or three fresh lemons three times a day, and to abstain from milk and fatty food, is based on similar principles.

The so-called Salisbury diet, which consists of three small meals of finely minced lean meat, each followed at an interval of two hours by half a pint to a pint of hot water, is a starvation diet, and acts probably very much like fasting, while the hot water promotes elimination.

Schroth's dry diet is also a cure by inanition, but the rational basis of the plan in all its details is not apparent, as after four or five days' abstinence from all drink and all food except five or six small dry rolls, the patient is encouraged to take a litre or a litre and a half of light wine, which, as might be expected, often causes intoxication; but the results of the Lindewiese cure, as reported by a recent observer (F. Sandoz), are on the whole not unfavourable.

B.

Physiology teaches us how to stimulate the secretion of the various digestive juices as the whole series depends for its efficient working on the proper initial start. Careful mastication not only divides the food mechanically and mixes it with the saliva, but stimulates the salivary glands to secrete and the saliva in turn provokes the flow of gastric juice and thus the other secretions. Therefore the habit of hasty eating and imperfect mastication from any cause may impair the whole process of digestion, while conversely due attention at this stage is calculated to ensure the complete carrying out of the entire function. As we have seen that these digestive agencies are the most powerful means we possess for destroying or neutralizing alimentary toxins, the importance of this point can hardly be exaggerated, although to chew food until it is tasteless and remove the residue from the mouth, as recommended by Mr. Fletcher, goes beyond what is reasonably necessary.

C.

We have seen that the ordinary intestinal bacteria afford a protective agency which, although weaker than the digestive juices, checks the action of putrefactive and pathogenic bacteria and neutralizes the toxins formed by them. Of the ordinary bacteria which play this useful part the best known is the lactic acid bacillus—a normal inhabitant of the intestine which has been made famous by Metchnikoff, but the value of butter-milk in remedying the results of a debauch has long been known in the North of Ireland. Bulgarian sour milk or Yoghourt when properly prepared is a pleasant article of food which promotes the normal action of the bowel, and the use of this or of butter-milk should be encouraged.

D.

As the stomach is the place where the food first rests and remains longest, it possesses the most efficacious antitoxic properties. The healthy stomach is sterile, and all food during its stay within its cavity is sterilized as a result of peptic digestion, and this is carried out in the presence of sufficient hydrochloric acid and pepsin. Pepsin is deficient in only the rarest instances, but the supply of hydrochloric acid may be defective or altogether absent. Under these circumstances the administration of hydrochloric or nitro-hydrochloric acid is indicated, but should be in larger doses than those commonly administered. Ten or fifteen drops of hydrochloric acid (*B.P.*) may be added to half a pint of sweetened water, and this quantity used as a drink during or after each meal. Mercurials, especially blue pill or calomel, act as antiseptics in the stomach and duodenum, but most of the so-called intestinal antiseptics have not justified the claims made for them, and it is doubtful whether any antiseptic drug in practicable doses can preserve its bactericidal capacity after passing a short distance down the intestine.

Purgatives unquestionably eliminate toxins, and are among the most powerful means we possess, but many act only on the pelvic colon and rectum, and do not clear the whole length of the large intestine. Senna possesses this valuable property, and one of the best aperients for chronic faecal stasis is an infusion of senna pods taken every night at bedtime.

E.

The practice of drinking large quantities of water, especially of the milder mineral waters, has much in its favour. Unmineralized water is apt to stay in the stomach and so to cause discomfort, but this does not seem to be true of hot water, which many people take on rising and again at bedtime. Small quantities of salines, especially magnesium chloride or sodium phosphate or sulphate, may be added to the water with advantage. This helps elimination by preventing the drying of the faeces in the colon, and stimulates peristalsis. To ensure complete evacuation of the large bowel irrigations or ascending douches are among the most efficient means, but there is a risk of inducing atony of the bowel when their quantity is excessive or their use is continued too long. The "entero-cleaner" of Anton Brosch is the most elaborate of ascending douches, the chief advantage claimed for it being that, as it is administered under water, the external pressure supports the bowel and prevents over-distension. When the above described methods have had a fair trial, and there is reason to believe in serious pathological changes in the colon permitting the continued absorption of toxins, its exclusion or removal should be considered.

CONCLUSIONS.

1. Under physiological conditions the natural protective agencies in the alimentary system are sufficient to shield the body from the dangers of poisons formed therein or of those introduced with the food, provided that these are not in overwhelming amount.
2. Infrequent or incomplete evacuation of the colon is not in itself a cause of disease.
3. Such morbid symptoms as arise result from a breakdown in the protective machinery, in consequence of functional defects or pathological changes in the organs concerned.
4. Wholesome food in reasonable quantities may consist of animal or vegetable protein, carbohydrates, and fat in due proportion, and the evidence does not justify the entire exclusion of animal protein, as being directly or indirectly the cause of toxæmia.
5. When toxæmia is present treatment should be based on the principles of eliminating the poison already present, preventing any further introduction and reinforcing the natural protective agencies.
6. Where there is extensive disease of the wall of the colon the exclusion or removal of this organ is a justifiable proceeding after a reasonable trial has been given to medical methods.

CONSEQUENCES AND TREATMENT FROM A
SURGICAL POINT OF VIEW.

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I WILL endeavour to give a brief summary of the surgical treatment of the auto-intoxication and other effects which result from chronic intestinal stasis. The causation of stasis and the consequent infection of the gastro-intestinal contents are due to improper feeding in early life, and subsequently to the prolonged assumption of the erect posture of the trunk.

The changes that result in the drainage scheme are evolutionary in nature, and simply mechanical in origin. To meet the tendency to downward displacement of the viscera which results from an accumulation of faecal matter, at first in the large intestine, and later in the small intestine and stomach, membranes or mesenteries develop. These bands represent the crystallization of resistances. They form, in the first instance, as thickenings on the peritoneum, and later as distinct membranes or mesenteries, which support the viscera and tend to obviate their prolapse. Some observers regard these new developments as being congenital in origin, while others consider that they are inflammatory products. Against both these hypotheses are the facts that they do not exist at birth, that they can be seen in every stage of development during the lifetime of the individual, that they vary distinctly with occupations, that they commence at that point in the mesentery which is most distant from the bowel, and are obviously developed for the purpose of securing that particular portion of the intestine more closely to the abdominal wall, that they appear only on one surface of the mesentery, and that surface is the one along which the greater amount of strain is exerted, and that they correspond exactly in situation and direction to the lines of force. Besides this, they occur in the layers of peritoneum in which the main lymphatic trunks do not run, while they do not develop on such peritoneal surfaces as are chiefly in relation with the lymphatic glands and vessels.

At their commencement they serve a useful purpose. Later they tend to impair the function of the part and consequently to shorten life. This is a common characteristic of such evolutionary changes as develop during the lifetime of the individual.

While the effluent through any portion of the intestine may be controlled by some mechanical means applied externally, as, for instance, by a band, membrane, or appendix, the contents of the intestine may also be dammed back merely by the accumulation of material in the bowel beyond. This is illustrated by the puddling and obstruction in the much elongated pelvic colon seen so frequently in tuberculosis and rheumatoid arthritis in young people, and again in the delay of the contents of the ileum, and their consequent infection when material is accumulated in a large dilated caecum occupying the true pelvis, or occasionally when the caecum is fixed immovably in the iliac fossa. In any case the result is the same—namely, stagnation of the intestinal contents and the changes consequent on such delay.

The result of this stasis of the intestinal contents manifests itself in two distinct ways, which may or may not be obviously associated clinically. The mechanical results of such delay in the small intestine are interference with the effluent from the duodenum and consequent distension changes in the duodenum, more especially in its first part. This obstruction produces engorgement of, and later inflammatory changes in, the mucous membrane of the bowel, and may finally cause ulceration and perforation, or cicatrization and stenosis of the first part of the duodenum. Spasm of the pylorus follows on distension of the duodenum, and this brings about dilatation of the stomach with hypertrophy of its walls. Because of the strain exerted by the heavy contents of the stomach, often increased by the weight of an habitually loaded transverse colon, engorgement, inflammation, ulceration, and finally cancerous infection of the mucous membrane may develop along the lesser curvature at the site of greatest strain. This area of excessive tension approximates to the