PEPTIC ULCER

A NEW APPROACH TO ITS CAUSATION, PREVENTION, AND ARREST, BASED ON HUMAN EVOLUTION

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PREFACE

IT would probably be fair to say at the present time that all authorities who have studied the causation of peptic ulcer agree on one point: that it is not properly understood. Most consider that the causes are likely to prove multiple, and to include the 'stress' of living in a modern, civilized environment. Many consider that the chief cause lies in the aggressive nature of the gastric acid becoming exaggerated, or in the defensive elements in the gastric and duodenal membranes becoming deficient.

The present work, based on the Darwinian theory of evolution and certain accepted physiological and biochemical facts in digestion, takes the very opposite view to all this—that the causation of peptic ulcer is not multiple but basically single; that it is only slightly related to stress; and that it does not lie in the gastric acid becoming too aggressive or in the defensive elements against it too weak, but that it lies in a third factor, which is operating unobtrusively in many parts of the world and is almost alone responsible—interference with the natural buffering of the gastric acid by the food, through the removal of protein in the refining of carbohydrates. The argument will be supported by a large accumulation of evidence from many countries, and also from prisoner-of-war camps in Japan and Russia during the last war, not so far used in this connexion.

This approach points to dietetic measures in cases of peptic ulceration that are almost the exact opposite of those advised to-day. Since the latter are considered by many authorities to be useless in influencing the course of the disease, and since they certainly fail to prevent the sacrificing of 30,000 human stomachs in operating theatres in this country annually, it is considered that there is reason for hope in the new approach.

This book reflects not only thirty years of intermittent thought, and many years of practice as a physician, but also several thousand hours toil in the accumulation of evidence. In connexion with the latter the author is under a profound obligation to the numerous medical authorities in many countries whose kindness has made the work possible. He hopes for their sake that the result may prove to be a significant contribution to the

understanding of this disease, and to the correct dietetic prevention and arrest of it. It must be emphasized, however, that the responsibility for the interpretation of the facts supplied is borne solely by the author.

The author is also indebted to the Admiralty, for permission to publish the work; to the Librarian of the British Medical Association, Mr. F. M. Sutherland, for great help over the references, and to the Association itself for providing these valuable facilities for its members; and finally to his secretary, Miss M. Handel, for her unstinted labours in typing both the manuscript and a massive correspondence.

November, 1962 T. L. CLEAVE

FOREWORD

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PEPTIC ulcer poses as one of the important unsolved problems of present-day medicine and makes appreciable demands on the hospital services. A challenging aspect of this problem is the changing incidence of peptic ulcer in the present century and its varying prevalence in different countries. For these marked differences there must be a cause. Clinical experience has suggested that the cause may be multiple, and may include anxiety, smoking, and fatigue as environmental factors, with the parietal cell mass and the blood groups as helping to determine the hereditary tendency. But there is also the possibility that these may only be modifying factors in a deeper, central cause, as put forward in this work. Surgeon Captain Cleave has brought together the available data on 'protein-stripping' in basic foods and the prevalence of ulcer. I personally accept the thesis that this may be an important factor in ulcer production, not necessarily connected with the hydrochloric acid, though this is the obvious link.

There is a remarkable facility in human nature for considering that because something is done universally it must be right. All the time it is necessary to re-examine modern so-called civilized practices. In an article some years ago I wrote that "gastric and duodenal ulcers can occur in lower animals, but only rarely in the natural state. Natural selection would tend to eliminate animals prone to develop ulceration from environmental factors. In man new personal habits, new foods, new drinks, new methods of cooking and food preparation, and changing feeding habits have come under the guise of civilization, but the forces of natural selection have become blunted by medical science."

I believe that Surgeon Captain Cleave's thesis underlines an aspect of modern nutrition which is important not only in relation to ulcer but possibly to many other conditions as well, more even than he himself has postulated. The remedy advanced in this work is a simple and practical one, and could change the present attitude to the treatment of peptic ulcer in a most radical manner.

PEPTIC ULCER

CHAPTER I

The Law of Adaptation

A COMPANION work to the present one, dealing with varicose veins,[1] began by drawing attention to recent statements by Huxley[2, 3] that Darwin, a hundred years after the publication of his *Origin of Species*, held first place in the biological field throughout the world, both by demonstrating that evolution must have occurred and by discovering the method by which it could and did occur, and that it was not sufficiently realized that the spectacular discoveries of genetics provide to-day a firm foundation for a theory which was originally based on pure deduction. The present writer then expressed his opinion that it was remarkable that in the year 1959, the centenary of Darwin's work, modern medicine should be making so little practical use of the theory of evolution, and he attempted to show that the prevailing ideas on the causation of varicocele, varicose veins, and to some extent femoral thrombosis, provided a striking example of this neglect. In the present volume he attempts to show that this neglect is equally striking in the case of peptic ulcer.

As in the companion work, it is first necessary to state that the side of the Darwinian theory chiefly involved is that which concerns the remarkable adaptation in all species to their natural environment. This adaptation, universal in extent and profound in degree, excited Darwin's imagination from an early date, and it was actually on his explanation for it—the struggle for existence, with survival of the fittest—that his theory of evolution was primarily based. Various explanations had been offered in the past for this adaptation, which had excited the imagination of others long before Darwin, but none so logically linked the adaptation with the *time* factor as his did. This latter factor is likewise of great importance to the present work, where the necessity will be stressed of an adequate period of *time* for adaptation to take place in a species to any unnatural (i.e., new)

feature in the environment, so that any danger in the feature should be assessed by how long it has been there.

It is fortunate that though there may still be differences of opinion over exactly how adaptation in species takes place, there are none over the fact that it does take place, for it is only with the latter fact that we are concerned here.

The adaptation to their environment which is constantly taking place in all species, and which at times has attained stages bordering on the miraculous, will be referred to in these pages as the 'Law of Adaptation', an expression that will be found to confer many advantages.

The law of adaptation is not absolute. Nature—that is to say, a 3000-million-year span of evolution—has not led to completely perfect adaptation in an organism to its natural environment, and evolution is still taking place. Congenital malformations, therefore, do occur. Normally the struggle for existence, i.e., the evolutionary process, keeps these failures very rare indeed. It is a matter of common observation that amongst wild creatures commonly consumed as human food, such as herrings, rabbits, or wood-pigeons, any congenital malformation is a very great rarity. In Man, however, under civilized conditions, these malformations have been allowed to become more prominent. Even under these conditions, however, the struggle for existence has prevented any congenital defect exceeding 5 per 1000 live births, the highest rates being the following:—[4, 5]

	Rate per 1000 *Live Births*
Spina bifida meningocele	2·0
Congenital malformations of the heart	2·8
Cleft palate, hare-lip	1·5
Pyloric stenosis (not certainly a true congenital malformation)	3·3
Club-foot	4·2
Congenital idiocy (including mongolism)	2·1

Most other congenital defects show much lower figures than these.

It follows from the above that before postulating an exception to the law of adaptation and attributing an ailment to a congenital defect, the frequency of occurrence, or incidence, of the ailment should be considered. If the incidence is many times as great as any known congenital defect, then, quite apart from other considerations, such a cause for the ailment becomes extraordinarily improbable. For example, in a companion work the

author has pointed out that the incidence of varicose veins is about twenty times as great, which fact alone, quite apart from the absence of the condition in non-Westernized peoples, enables any view of a congenital cause to be attacked at its very core. A similar attack will be made in the present volume against the possible view of a congenital element in the causation of peptic ulcer, the incidence of which is also about twenty times as great as any known congenital defect, and especially will this attack be sustained in the chapter on possible hereditary factors in the causation.

The above is only one example of the value of the law of adaptation in the elucidation of the causes of disease, but, indeed, its value will be seen at every stage of the argument now to be presented.

With regard to the term 'Nature', as used above, it may be added here that there is nothing unscientific, and much that is convenient, in this term, provided it be realized that it does nothing more than personify the total span of evolutionary adaptation on this planet. So compact an expression will therefore be made use of in the present work from time to time and much space will be saved.

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CHAPTER II

The Law of Adaptation and the Role of Hydrochloric Acid in the Production of Peptic Ulcer

FAILURE to appreciate the law of adaptation leads, in the author's opinion, to grievous misconceptions at the present time about the part played by the gastric hydrochloric acid in the production of peptic ulcer. Though the virtual confinement of peptic ulcer to the stomach and duodenum, which are the only parts of the intestinal canal that are normally exposed to this acid, points to the acid being undoubtedly the *agent* involved in the production of the ulcer, an effort will be made to show that the law now under consideration excludes it altogether from being *a primary cause.*

Yet to-day there is a widely held opinion that this acid is itself the primary cause of peptic ulceration. Another opinion widely held is that the primary cause admittedly lies elsewhere and is to be found in the 'stress' of living under modern conditions. It will be shown, however, that this second opinion is just as incompatible with the law of adaptation as is the first.

Meanwhile, to discuss the former view, it appears at first sight surprising that anyone should hold that the gastric acid is the primary cause of peptic ulcer, considering that it has been well observed[1] that this substance is natural to the stomach of every vertebrate. In point of fact the holders of this view march under not too clearly defined a banner. For example, a recent leader in the *Lancet*,[2] dealing with this subject, stated the following: "Anyone seeking in a court of law to prove 'acid aggression' responsible for peptic ulceration would be dismissed after two minutes' cross-examination with a stern admonition not to waste the court's time. But on the unjudicial assumption of guilt until innocence is proved, the hypothesis has served doctors and their patients extraordinarily well." Here the acid is not made primarily responsible, yet the first article in the same issue, on the pathogenesis of ulcer, begins with the following:[3] "The inescapable equation of ulcer aetiology—acid-pepsin aggression

versus mucosal defence—allows for certain differences in the pathogenesis of duodenal and gastric ulcer. In duodenal-ulcer patients, gastric secretion is characteristically higher, the acid-bearing (fundic) area of the stomach larger, the gastric mucosa thicker, and the parietal-cell mass greater than is usual in normal people (Cox, 1952; Card and Marks, 1959). Gastric-ulcer patients, on the other hand, often have normal or subnormal secretion and atrophic mucosal changes. It is thus understandable that an increase in acid and pepsin should be incriminated as the dominant aetiological factor in duodenal ulcer, and impaired mucosal resistance as the underlying cause of gastric ulcer (Illingworth, 1956)."

These words express perfectly the view perhaps most widely held at the present time, in which increased acid aggression and decreased mucosal defence are exalted for all practical purposes to the status of *primary causes* of peptic ulceration. In each case Nature is made at fault and the law of adaptation ignored.

In the author's opinion, far from serving the doctors and their patients extraordinarily well, the above view has served them extraordinarily badly, since it has distracted attention from a third factor, interference with the natural buffering of the gastric acid by the food, which the author will attempt to show holds the secret of the essential cause of peptic ulcer, and which, unlike the above view, never comes into conflict with the law of adaptation.

Meanwhile, to continue the discussion of the former view, much is made by this school of the factor 'hyperchlorhydria'. Any level of acidity in the stomach above the average is necessarily considered to increase the danger of the 'acid aggression' and therefore to constitute a possible threat to health. Strongly supporting this view are the feelings of actual sufferers from dyspepsia and peptic ulcer. Hyperacidity, heartburn, acid eructations, or even plain 'acidity'—what do all these terms from countless advertisements in the lay press indicate but that the actual feelings of the sufferers themselves are overwhelmingly behind the view that the acid is the cause of the trouble, particularly an excess of acid ?

In conformity with the opinion that the hydrochloric acid is the dominant cause of peptic ulcer, all medical and surgical treatments to-day are aimed at controlling this acid, either buffering it with alkalis, or preventing its production by cutting

the vagus nerves that control its secretion, or actually removing the stomach by operation. The first of these operative measures disquiets, and the last appals, those who have a reverence for the human body, however necessary such steps may become in the late stages of the disease, where scarring of the membranous surfaces of the stomach and duodenum upsets the delicate balance that normally obtains there, so that relapse is almost inevitable with any alternative treatment. Yet if the law of adaptation were deeply understood and applied to the subject of the gastric acid, it is contended that events would seldom arrive at a pass where these measures were required.

For the law of adaptation indicates that the production of hydrochloric acid must be just as perfectly attuned to the requirements of the individual as is, for example, the power in his arms or his legs or any other part of his anatomy. Far from constituting a liability, the production of large amounts of acid in the stomach, passing under the term 'hyperchlorhydria', should be regarded as a most necessary asset. A powerful machine is useless unless it receives the right amount of fuel to run it. A powerful man is equally reduced to impotence unless his stomach produces the right quantity of acid to secure the digestion of the large amounts of food his energies require.

As already stated, evidence will be produced later to show that it is not the production of acid that is at fault in the causation of peptic ulcer, *but its imperfect buffering by the altered foods of our present civilization*, aided by disregard of the instinct of appetite. But this evidence must wait, for the obvious first reply of those who incriminate the acid as the primary cause is that its production might be at fault for congenital reasons, just as the body can exhibit other congenital errors, like hare-lip and club-foot.

In the opening chapter, however, the incidences of various congenital deformities were set out in detail and it was seen that not one of them exceeded 5 per 1000. Contrast these incidences with that of peptic ulcer, which in this country is now so great that about 10 per cent of men and 4 per cent of women develop clinical evidence of the disease at some period during their lives, the post-mortem evidence being nearer 20 per cent for each sex.[4, 5] Thus, the incidence of peptic ulcer is over 20 times as great as that of any known congenital abnormality, and this fact alone, quite apart from other objections, such as the age of

onset, makes a congenitally installed hyperchlorhydria as the cause of the disease improbable in the extreme.

In short, the view that the gastric hydrochloric acid is the primary cause of peptic ulcer is incompatible with the law of adaptation, and this incompatibility cannot be overcome by postulating the presence of a congenital abnormality.

The view that the hydrochloric acid is the primary cause of peptic ulceration is also quite incompatible with the historical and racial incidence of the disease, but the discussion of these two important pieces of evidence must await the consideration of other possible causes, one of which will now be examined.

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CHAPTER III

The Law of Adaptation and the Role of Stress in the Production of Peptic Ulcer

To incriminate stress as the primary cause of any disease in our present civilization, including the disease of peptic ulceration, is almost always incompatible with the law of adaptation. For what does evolution involve but a struggle for existence that has continued from the dawn of creation, and is still continuing? And if this struggle for existence does not represent the quintessence of stress, what does? In the ceaseless killing of one individual by another stress exists in its extreme form, and to compare with this ruthless slaughter the irritations of a welfare state is quite unacceptable to anyone who thinks deeply on the subject. Certainly, if there is one thing that every single organism on this planet knows about, and is equipped to deal with, after a remorseless struggle extending over several thousand million years, it is stress in all its shapes and forms.

Stress as a primary factor in the production of peptic ulcer is also quite incompatible with the historical incidence of the disease. For all authorities are agreed that the very high incidence of peptic ulcer in Westernized countries, certainly as regards duodenal ulcer, is relatively of recent origin, first becoming conspicuous in this country at the beginning of the present century, and becoming steadily more so ever since. Many references could be given in support of this. For example, Brinton, in writing the first book on peptic ulcer in 1857, based on 1200 autopsies, in which he attempted to discriminate between cancerous degeneration of the margin of an ulcer and the ulceration of a gastric cancer, made no mention of duodenal ulcer at all. To quote from Ogilvie:[1] "The most striking fact about duodenal ulcer is that it is, in Europe and America at any rate, a new disease. Occasional cases were reported in the nineteenth century, but the text books of medicine and surgery made no mention of it till about the year 1900. From the beginning of the present century it has assumed an increasing importance. In the 1901 edition of Osler's *Text-book of Medicine*, the author records that he had twenty-five cases of

peptic ulceration in his wards in nine years. He remarks that 'duodenal ulcer is less common than gastric ulcer'. In the 1947 edition it is said that 'There are more duodenal than gastric ulcers, in the proportion of four to one.' In the First World War, as is well known, 709 men were discharged in the first 17 months owing to peptic ulceration. Duodenal ulcer is not mentioned in the official history. In the Second World War, 23,594 men were discharged owing to peptic ulceration in the first 28 months."

The steady rise in perforations and hæmatemeses, which are events that cannot easily be misinterpreted, refutes the idea that this rapid increase is the product of better medical technique, such as radiographic diagnosis. This is summarized by Backett:[2] "Various indices have been used which are likely to reflect the increase. They include mortality rates (Morris and Titmuss, 1944), and the reports of perforations and hæmatemesis (Avery Jones, 1947, 1955, 1957), and perusal of ancient medical records for perforations (Jennings, 1940), and an examination of discharges from the British Armies in two world wars (Morris *et al.*, 1944). Though of varying value all these analyses indicate a rapid increase."

The evidence need not be continued. And to suppose that stress should have arrived on the scene in this country only since the beginning of the present century is to suppose the impossible. Do any of the trials in our present social system compare with the poverty and suffering that attended the industrial revolution, and explosive increase in population, that occurred in this country during the preceding century? That is why it was stated above that stress as a primary cause of peptic ulcer is not only incompatible with the law of adaptation—it is also incompatible with the historical incidence of the disease. The time may be approaching when, to quote from the *British Medical Journal*,[3] the facile explanation that peptic ulcer is a disease due to mental stress may seem as remote from the truth as the view that malaria is caused by vapours arising from the swamps.

Nor must sight be lost of the serious effect on national and personal morale of any doctrine that lays down that mental and physical effort can be harmful. The struggle for existence would not permit any nation holding such a doctrine to remain important for long. What is essential is to promulgate the exact opposite doctrine—that the correctly nourished human body is capable of miracles of effort, and that far from suffering as a consequence

of these efforts, it benefits from them both mentally and physically.

However, although in this work stress is absolutely excluded as a primary cause of peptic ulceration, it is conceded that it plays a part in one factor which aggravates the true primary cause. The primary cause will be advanced first, and the aggravating factor left till a later chapter, where it will be seen to consist of disregard of the instinct of appetite, through the eating of food that is not desired. In connexion with this disregard, stress will be shown to be important.

The incrimination of stress in this manner leads to no conflict with the law of adaptation. From a practical point of view, also, the distinction is vital. For if stress is a primary cause of peptic ulceration, the effects cannot be avoided if the stress continues, whereas if stress only plays a part in the disregard of the instinct of appetite, which is avoidable, it follows that the effects of stress are also avoidable, no matter how great the stress may be.

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CHAPTER IV

Protein-stripping in Carbohydrate Foods advanced as the Fundamental Cause of Peptic Ulcer

Part I

Protein a Natural Buffer of the Gastric Acid

If the evolutionary approach to the problem of the causation of peptic ulcer positively excludes both hydrochloric acid and stress as primary causes, this same approach leads directly to some recent alterations in the natural environment as the primary cause. Fortunately it is possible, through drawing on known facts in physiology and biochemistry, to decide what this alteration almost certainly comprises, and in the following pages it will be advanced that it consists of the unnatural stripping of protein from certain key carbohydrate foods (especially sugar cane and the sugar beet) during modern refining processes, the loss of protein leading to interference with the natural buffering of the gastric acid. The interference will be most serious where the refined foods are consumed by themselves, but may also be serious where they are consumed during only part of a meal, owing to layering in the gastric contents, to be described shortly. The physiological details of this argument will now be presented, and in later chapters it will be shown that the lines of incidence of peptic ulceration in every country accurately follow the lines of consumption of these refined carbohydrates, the disease being common where their consumption is high and disappearing altogether where their consumption is absent. The same will be shown to be true in Japanese and Russian prisoner-of-war camps in the last war, ulcer cases being very prevalent or completely absent, depending on the commonness or absence of these refined carbohydrates in the diet.

The argument begins with the known fact that the only food materials affected by the gastric hydrochloric acid and pepsin are disaccharide sugars and proteins. Fats are untouched (as the acid rapidly inactivates lipases). The hydrolysis affected in sugars does not result in any buffering of the gastric acid;[1] but

during the digestion of protein a considerable buffering of the acid takes place. This is one of the factors responsible for the difference between the 'free acidity' and the 'combined acidity' in the old test meals. It must be remembered, however, that this buffering never approaches the point of complete neutralization.

In conformity with these physiological facts, all the great dietetic treatments of peptic ulcer during the present century have been based on foods containing a high proportion of protein. One of the first of these high-protein diets was the Salisbury diet. This was introduced before the First World War and was based on some 3 lb. of chopped meat daily, eaten either raw or very lightly cooked. The next high-protein diet was the Lenharz diet, which had a considerable vogue in the period following this same war, and was based on eggs, again eaten raw or very lightly cooked. This was followed by the Sippy diet based on milk, which is still in use to-day. The only other class of food containing a proportion of protein comparable to the above is fish, and it is noteworthy that steamed fish has nearly always been prescribed early in any peptic ulcer diet.

The importance of protein in the buffering of the gastric acid, though it has largely been appreciated in the treatment of active peptic ulceration (but never enough, as the writer will try to show), has been very little appreciated in the prevention of the disease. A discussion later of peptic ulcer diet charts from the largest hospitals in England and Scotland will be seen to demonstrate this fact in striking manner. But much more important than this, the stripping of protein during the refining of carbohydrate foods, which takes place on a large scale in civilized countries to-day, with important repercussions on the buffering of the gastric acid, has not been appreciated at all. The extent of this stripping will be shown to range from 10 per cent of the protein in the case of white flour to virtually 100 per cent in the case of ordinary crystalline sugar. Some examples of the resulting loss of buffering power on the gastric acid are given in Appendix A.

It is abundantly clear, however, that the buffering qualities of the food eaten constitute only one-half of the equation governing the acidity of the gastric contents—the other half being constituted by the amount of acid secreted. For example, in theory a food that was a good buffer might cause so big a flow of gastric juice that the resulting net acidity was high, not low. In the author's opinion the correct way to pursue this subject is not by

experiments via test meals, but by cold logic via the law of adaptation, with careful epidemiological testing of the conclusions so reached.

As regards test meals, he firmly believes with E. N. Rowlands[2] that "most patients dislike intragastric tubes more than almost any other diagnostic procedure, and there is no doubt that the nausea and distress caused by the presence of a tube vitiate many of the studies of gastric function, because a patient's emotional state exerts a profound influence on secretion and motility", and as regards serial test meals, that though they constitute "an ingenious method of measuring the secretory response to an aqueous test substance given by mouth, only a physiologist could commit the gastronomic solecism of describing these substances as 'meals' "; and "it is very unlikely that the stomach empties or secretes in response to ordinary meals as it does to these tasteless solutes". (The word 'tasteless' here is used for insipid, i.e., failing to cause delight.)

This interference with the psychological aspect of a natural meal has particularly important repercussions on the gastric motility and therefore on the gastric emptying time. Thus Sir Charles Evans:[3] "The influence of the nervous system on stomach movements is undoubted, and emotional states or unpalatable food lead to suspension or reduction in the tone and movement of the stomach." It will be shown in Chapter XI that the gastric emptying-time also depends on the *hunger* present before a meal. Therefore, the emptying-time after a test meal that is not enjoyed, and for which the patient may not have been hungry, will be very different from that after a natural meal, where the opposite state of affairs obtains.

Furthermore, it will be seen later that the gastric emptying-time is also considerably influenced by the physical state of the food eaten, being very much faster, for example, if the state of the food is pultaceous, as so commonly occurs in test meals, than if it is coarse and fibrous, as would often be the case under natural conditions.

The gastric emptying-time is important because it directly affects the period during which the membranes of the stomach itself are exposed to the high levels of acidity prevailing during digestion, as will be elaborated later, and also because it directly affects the backward diffusion into the first part of the duodenum of the protective alkaline contents of the second part. If the

emptying-time is slower than natural, therefore, this may influence the production of a gastric ulcer, whereas if it is faster than natural, this may influence the production of a duodenal ulcer. It follows that the emptying-time is of the greatest importance and yet, for the reasons just given, is liable to be seriously distorted in any artificial test meal.

Finally, the effect of mastication on food, leading to its impregnation with saliva, which will shortly be shown to be important in buffering the gastric acid, is very largely lost in the case of test meals, owing to the frequent pultaceous state of the food, just described.

As an example of the difficulties in interpreting test meals, the teaching for many years has been that the highest levels of acidity are in the resting stomach. The work of M. Atkinson and K. S. Henley[4] now indicates the opposite. Thus, in a personal communication to the author (1961) the former states: "I think you are correct in assuming that if the stomach remains empty the acidity tends to fall, as during the night period, and it is certainly true that rises in gastric and duodenal acidity are greatest in the post-prandial period." Again, to quote from James's *Physiology of Gastric Digestion* (1957):[5] "The highest acidities of all are reached during the digestion of food, not when the stomach is empty." If the acidity of the gastric contents an hour after taking food is *p*H 2, the acidity of such juice as there is in the empty stomach is about *p*H 3.[6] Meanwhile, in the belief that the highest acidity occurs in the empty stomach, thousands of ulcer patients, year after year, have been advised—and still are advised—to avoid letting the stomach get empty, which has involved the constant eating of meals for which there is no desire, a most unnatural and baneful procedure, the consequences of which will be discussed in detail in Chapter XI. (It may be added that even if it were true that the highest levels of acidity occur when the stomach is empty, this would still never justify the advice to eat in opposition to the instinct of appetite. There are many ways in which such high acidity could be counterbalanced in nature, the most obvious one being that the mucous protection when the membranes are contracted in the empty stomach is clearly much greater than when they are extended in the full one.)

It will be seen from the above how hazardous is the interpretation of test-meal results, and why the amount of acid secreted, which, in conjunction with the buffering qualities of

the food eaten, governs the acidity of the gastric contents, will be pursued here not via these results (which, properly applying to the present argument, do not exist at present in any case) but via the law of adaptation.

This law indicates quite axiomatically that the interaction between the acid in the gastric juice and the protein in natural foods will result in a level of acidity which, though adequate to secure digestion, is safe to the secreting membranes concerned. This assumes a natural rate of emptying of the stomach, itself dependent, as will be elaborated later, partly on a natural physical texture in the foods consumed, and partly on a natural delight in the consumption of them.

Though under natural conditions the gastric acid interacts with the protein in the food in the harmonious fashion just described, nothing is easier to understand than that, if this protein is removed, or even reduced, injury to the membranes of the stomach or duodenum may arise. For example, the following three possibilities suggest themselves:—

1. There may result some overshooting of the mark, in secretion, so that the peaks of acidity that normally occur during digestion are higher than natural.

2. Such peaks, though not higher than natural, may persist for longer periods.

3. The peaks, though neither higher nor more persistent than natural, may be reached more quickly, so that the secretion of mucin, or other defensive step taken by the gastric and duodenal membranes, may not be able to keep pace.

To make no claims over the first two possibilities, it is incontestable, as regards the third, that any protein in the food must at first buffer the acid and therefore cushion the effects of the rise in acidity that takes place. Nor should such an effect be disregarded just because it may be small. An example is given in Appendix B of a condition in cattle where a much smaller, and infinitely shorter, interference with the natural environment, as regards the food, can have fatal consequences.

Nothing more than the above simple argument is necessary for the basis of this work, which is largely epidemiological in nature and will seek to show, as already stated, that the incidence of peptic ulcer in every country rises or falls with the high or low consumption of carbohydrate foods that have suffered removal of protein in the manner described.

Part II

Protein Calories in Natural and Processed Carbohydrate Foods

In order to assess from the present point of view the importance of the protein in carbohydrate foods, and the serious consequence of reducing it, a backward glance may first be given at the digestion of grass in herbivorous animals. It is a surprising fact that the hydrochloric acid in the stomachs of these animals is only a little less prominent than in the case of man and, for that matter, than in the case of carnivores.[7–10] For example, the gastric acidity an hour after taking food, which averages about *p*H 2 in man, averages about *p*H 2·5 in the sheep, and about *p*H 1·7 in the dog.

The reason for the prominence of hydrochloric acid in the stomach of the herbivore lies in the protein content of grass, and in this connexion it is less constructive to say that the protein averages 2½ per cent by weight than that it provides about 20 per cent of the available calories present.[11] The necessity for the high hydrochloric acid in the stomach of the herbivore is thus revealed. Nor is this explanation impaired in the case of ruminants by the temporary incorporation of some of this protein in the bodies of bacteria attacking the grass and fermenting it.

This raises a subject of great importance to the present work—the method of assessment of the protein content of foods. The assessment is made here on a calorie basis, not a weight basis, for the following reason. It is known that the qualities of the various digestive juices do not vary appreciably with the type of food eaten, each juice being almost constant in composition; hence the amounts of the various juices secreted must bear a close relationship to the calorific value of the meal taken, since the whole function of the juices is to digest the various proteins, fats, and carbohydrates of which the meal is composed. This applies equally to the gastric juice, which digests sugar as well as protein. Thus, the flow of gastric and other juices following the pleasurable taking of a 10 per cent solution of a food protein in water would clearly become considerably greater after taking a 10 per cent solution of the same protein in syrup. The buffering effect of the protein on the gastric acid would therefore be appreciably less in the second case, owing to the larger volume of acid secreted. This difference in buffering effect would not be revealed if the protein

content of the solution were given by weight, but is revealed at once if it is given by calories. The latter method of assessment, in short, takes cognizance of other components in the food as well as protein.

The different picture presented by the two methods of protein assessment has just been seen in the case of the grass eaten by cattle; it can equally be seen in the case of a carbohydrate food eaten by men. In a lettuce, for example, the protein content by weight is 1 per cent, but by calories is 40 per cent, and a food that appears a poor bufferer of the gastric acid by the first figure is revealed as a good one by the second.

As a matter of fact, unprocessed carbohydrate foods are remarkably rich in protein calories. This may be seen from the following table, where some of them are compared in this respect with animal foods. (These and later percentages, except where indicated, have been calculated from the tables in *The Chemical Composition of Foods*, by R. A. McCance and E. M. Widdowson.)[12]

Foods of Animal Origin (Bones excluded)	Percentage of Calories derived from Protein	Foods of Plant Origin	Percentage of Calories derived from Protein
Milk (cows)	21·1	Wheat (English 100 per cent whole wheat flour)	11·0
Milk (human)	12·0	Potatoes	10·5
Cheese (Cheddar)	24·5	Peas	37·4
Eggs	29·9	Cabbage	52·2
Rabbit*	60·4	Watercress	81·9
Pigeon*	47·3	Tomatoes	26·0
Herring	28·9	Mushrooms	100·0
Cod	89·8	Almonds	14·1
		Apples	3·0 (average)
		Bananas	5·9
		Oranges	9·3
		Lemons (juice)	17·0
		Blackberries	18·1
		Rhubarb	39·6

* Domestic animals and birds are usually much fatter than their wild counterparts, so that in foods derived from them the proportion of protein is usually considerably lower. For example, the protein calories in a raw mutton chop amount to only 10·3 per cent.

The assessment of protein content by calories is particularly important when assessing the effects of refining carbohydrate foods. For example, the protein content of sugar beet is usually 2 per cent by weight, whilst that of refined (white) sugar is 0 per cent. It might seem, therefore, that a reduction of protein of this order during the manufacture of sugar was of small consequence. But if the protein content be assessed by calories, then the figure for sugar beet becomes 12 per cent and for refined sugar 0 per cent—and the picture has greatly altered.

These refining processes in carbohydrate foods remove a considerable amount of protein, but hardly affect the carbohydrate itself. The resulting disturbance in the percentage of protein calories is brought out in the following table:—

Carbohydrate Foods	Processing Treatment	Percentage of Protein by Weight removed in this Treatment	Percentage of Protein Calories present before this Treatment	Percentage of Protein Calories present after this Treatment
English wheat, wholemeal flour (100 per cent extraction)	Milling and conversion to white flour (70 per cent extraction)	11·2	11·0	9·3
Rice (husked only)	Milling (70 per cent extraction) plus washing plus boiling	30·0 (average)	10·6	7·3
Potatoes	Peeling plus boiling	Variable, about 4–16 per cent depending on the degree of slicing	10·5	Variable; suggested figure 9·5
Manioc tubers	Peeling plus various water treatments	60·0	3·0	0·4 (tapioca)
Sugar beet	Extraction of refined (white) sugar	100·0	12·0 (average)	0·0

In the above table the sources of the figures for rice and manioc will be given later, but the figures for sugar beet have been calculated from data supplied by the British Sugar Corporation. For the figures for the potato see opposite.

As regards the reduction of protein effected in rice and the manioc tuber, this is of small importance in this country, but it will be shown later to be of great importance in certain non-Westernized countries, where these foods may form almost the only source of protein for the natives concerned, and where peptic ulcer becomes very common indeed whenever the processing procedures take place.

As regards the loss of protein during the peeling and boiling of potatoes, it is the double procedure that causes the loss—if the potatoes are unpeeled, practically no loss of protein occurs on boiling. *Per contra*, if the potatoes are not only peeled but also sliced up, the loss on boiling increases from about 4 per cent to about 16 per cent (if they are cut into 1-cm. cubes).[13] These figures refer only to loss of protein, but, in fact, during the peeling and boiling of potatoes an approximately equal loss of non-protein nitrogenous constituents takes place, most of which are likely to consist of amino-acids. It would be a mistake to suppose that these amino-acids have no buffering effect on the gastric acid. On the contrary, in the range of *p*H 1–3, which covers most phases of gastric digestion, amino-acids are more effective than protein in buffering the acid.[14, 15] For this reason the loss of protein during the peeling and boiling of potatoes, given above, underestimates the intragastric consequences of these steps. This is probably equally true of very similar steps taken with the manioc tuber, to be described later.

It is important at this early stage to deal with a possible developing counter-argument to that being advanced here, which might run as follows:—

> If the protein in carbohydrate foods is important in the buffering of the gastric acid, and if the protein calories in an unprocessed apple amount to only 3 per cent, then any processed food containing protein calories in excess of 3 per cent, such as chocolate, which contains 4 per cent, must be just as safe as an apple—which, according to the argument presented, being a natural food, must be very safe indeed.

This argument entails reference to two other important effects of refining processes in carbohydrate foods *vis-à-vis* the buffering of the gastric acid. One of these is the effect on mastication and the other the effect on the gastric emptying-time.

Protein is not the only natural buffer of the gastric acid. The saliva, which has a *p*H value of 6·6, as compared with the *p*H 0·9 of pure gastric juice, must also be reckoned with. In the refining

of carbohydrate foods the removal of fibre leads to a big reduction in the mastication of the food and therefore in its impregnation with saliva. This loss can lead to a natural food being a better buffer of the gastric acid than a processed one, even though the latter may have a higher percentage of protein calories. For example, the protein calories in natural foods can, most exceptionally, fall as low as the 3 per cent present in an apple, which is even lower than the 4 per cent in, say, plain chocolate. The total calories in a fair-sized, unpeeled apple of 4 oz. are, however, contained in a piece of chocolate not much bigger than a hazel nut, and the buffering effect of the apple, after being chewed, is clearly very different from that of the small fragment of chocolate.

The removal of fibre from carbohydrate foods also considerably increases the speed of passage of these foods through the stomach. The effect of the physical state of the food, in general, on this speed of passage is well recognized. As Schinz and his co-authors[16] put it, from a radiological point of view: "Easily digested pultaceous nutrients, as we know from experience, leave the stomach significantly faster." But the effect of the removal of fibre, in particular, on the speed is remarkable. This was established by Wilson, Dickson, and Singleton,[17] in a series of radiographic studies on human subjects. These workers used unprocessed bran in their experiments, and found that when this substance was eaten by itself it remained in the stomach an exceedingly long time, over 50 per cent of it still being present after $4\frac{1}{2}$ hours. Similarly, porridge with 12 per cent of bran remained in the stomach so long that 30 per cent of it was still present after $4\frac{1}{2}$ hours, whereas without the bran nearly all of it had left after 3 hours.

Not only does this unnaturally rapid emptying of the stomach vitiate the results of test meals, as noted on page 13, which nearly always consist of refined carbohydrates, but it is also very liable to play a part in the actual production of ulcer, especially duodenal ulcer, since the rapid emptying necessarily reduces the backward diffusion into the first part of the duodenum of the protective alkaline contents of the second part.

Hereafter, when discussing the refining processes in carbohydrate foods, attention will be directed only to the loss of protein that takes place, but the additional effects just set out, of reduced mastication and quicker gastric emptying time, should not be forgotten. These further effects make it impossible to assess the danger in these processes in terms of loss of protein alone.

For the removal of protein during the refining of carbohydrate foods the term 'protein-stripping' will be used, not because the refining process frequently involves stripping away the outer coat of the parent food, since, as in the case of sugar, it can sometimes involve much more than that, but because the term is shorter and more convenient than any other the author has been able to think of.

Part III

PROTEIN-DISPLACEMENT

In the refining of carbohydrates, the refining processes lead to increased consumption. It would seem that this is due to the fact that the unnatural concentration deceives the taste-buds in the tongue. This is well seen if the smooth, spongy nature of white bread be contrasted with the rough, stodgy nature of crude, wholemeal bread, but still more so if the 5 oz. of refined sugar at present consumed per average-aged adult per day in this country is contrasted with the $2\frac{1}{2}$ lb. of sugar beet or sugar cane from which it is derived. Who would consume that amount daily of the parent substance? In the case of sugar, the rise in consumption following its refinement and concentration can actually be traced from the 13 lb. per year in 1815 to the 104 lb. per year at the present time, as can be seen in *Fig.* 3 on p. 78.

How much of this extraordinary rise in sugar consumption is accounted for by an increase in consumption to the optimum level, and how much by a true over-consumption through the deception of the tongue, is irrelevant to the present chain of reasoning, where only the *magnitude* of the rise is important. For the extra consumption of such a protein-stripped food must be at the expense of other foods in the diet from which, under natural conditions, no protein would have been stripped at all.

In short, the refining of carbohydrate foods not only reduces the protein outright, but also leads to a marked increase in the consumption of these stripped foods, so that to protein-stripping there becomes added *protein-displacement*.

Part IV

Consequences of Protein-stripping and Protein-displacement

In Westernized countries like ours, where the total protein-consumption is high, neither of the above protein losses would probably have a serious effect on the neutralization of the gastric acid were it not for two other facts of great importance:—

1. The stripped foods are often consumed by themselves.
2. The various parts of a meal largely remain distinct in the stomach.

1. As regards the first fact, consider any young couple in a cinema. In the middle of the performance they are only too likely to consume some sweetmeat, which will probably consist of almost pure sugar, with practically no protein at all. The calorific value of this sweetmeat is high and will result in a considerable secretion of gastric juice. The consequences will be that the membranes of the stomach and duodenum are exposed to almost completely unbuffered acid, often for several hours.

Very similar events take place on other occasions, too, such as during 'elevenses', where some three hours after breakfast the almost empty stomach receives a cup of tea or coffee, often heavily sweetened with sugar but with very little milk and therefore containing hardly any protein. Another example is provided by drinks of alcohol taken some time before meals, especially at cocktail parties. In all these cases the stomach is liable to be subjected to almost completely unbuffered acid, often for a long time. *In civilized countries, therefore, there lies concealed, under the cloak of a respectable total protein protection, a protein protection that at certain times of the day is seriously deficient.*

2. As regards the second fact, it cannot be argued that, where the protein calories have been heavily reduced in parts of a meal, though the total protein calories of the meal remain high, the same danger is not present. It may be very much present. Consider a meal where a glass of sherry (containing no protein calories) precedes a beefsteak, and a tinned fruit salad (containing only 1½ per cent of protein calories) follows the steak. It cannot be argued that the gastric acid will be adequately neutralized in this meal, for the following considerations show that it will not.

In the first place, in any radiographic meal it is easy to see that every peristaltic wave carries some part of the meal straight into

the duodenum. The first waves often fill the duodenal cap. It is not a fact that the stomach contents are churned up till an even mixture results, and then the pyloric valve is released and the whole meal projected onwards. In the second place, it has long been known to physiologists that the succeeding portions of a meal are arranged in corresponding layers in the stomach. Thus, as long ago as 1893 Cannon demonstrated this in cats, and to quote from James's *Physiology of Gastric Digestion*: "Neilsen and

Fig. 1.—Tracing from radiographs taken at the times stated after a normal subject had eaten 200 g. of fried meat ball without contrast medium, 50 g. mixed with neobar, and then 100 g. without. Tracing from Neilsen and Christiansen (1932). (*Reproduced by kind permission.*)

Christiansen[18] made similar investigations on the human stomach. Their general method was to give their subjects a meal, successive parts of which either contained or did not contain a contrast medium. The food was either fried balls of minced meat, or oatmeal porridge. The subjects were students who attended lectures in the intervals of being radiographed. Their published photographs, a tracing from one of which is shown in *Fig.* 1, show clearly that the successive portions remain distinct or nearly so throughout their sojourn in the stomach. Usually the successive layers were disposed horizontally, but sometimes the lines of demarcation were oblique (as in *Fig.* 1) or almost vertical. As the stomach emptied, the lowermost part of the meal, usually that ingested first, left the pyloric antrum, and the contents of the fundus narrowed and tended to move towards the pylorus, the boundaries between the layers becoming distorted in the process, but remaining distinct."

A further important quotation from James's work is the following: "Gianturco[19] and Neilsen and Christiansen also investigated the behaviour of radio-opaque liquid drunk while the

stomach contained solid food. Their experiences, with cat and man respectively, were similar. The drink did not mix with the food, but by-passed it, and left the stomach within a few minutes."

Veterinary surgeons have discovered that the above applies also to animals. Dukes[7] refers to studies by Ellenberger, Scheunert, Kiok, and others, demonstrating that similar stratification occurs in the stomach of the horse and pig and persists for hours. Any water drunk similarly by-passes the food and does not disturb the stratification. It follows from these observations that if a person drinks to a certain extent during a meal, he will by no means necessarily remove the stratification.

This proved layering of the stomach contents is of great significance to the present work. For it reveals that, as regards the buffering of the gastric acid, the even distribution of protein in a meal may be just as important as the total amount of protein in the meal. To revert to the example given above, the glass of sherry taken before the steak, and the tinned fruit salad taken after it, each result in a flow of gastric acid that may not be buffered by the steak. The proportion of protein in the total meal may well exceed that in the grass eaten by the cattle, previously quoted, but the buffering of the acid may be much less perfect.

To sum up, it is obvious that there is a fundamental difference in the protection afforded to the gastric membranes by, say, 12 oz. of protein food divided equally between three meals and the same 12 oz. divided between two meals with almost none in the third meal, as often happens in civilized countries to-day; and if this fact is admitted it is possible to extend it, through the known occurrence of layering, just described, to cover parts of meals. In short, it is contended that what is essential to the preservation of the integrity of the gastric membranes is not the total amount of protein consumed per year, per month, per day, or even per meal, but the amount of protein continuously present during the eating of any food—and that amount is never too small in any food in its natural state, since man has been evolved to be able to eat these foods with impunity. Remove or lessen the natural protection of this protein during a meal or part of a meal, by processing the food, and the immunity of the stomach to the action of its own acid can no longer be assured.

It will be shown next, in the chapter on the racial incidence of peptic ulcer, that the incidence bears little relationship to the

total protein-consumption, being often absent in races, like those in certain parts of Africa, which consume little protein, and very prevalent in races like our own, which consume a great deal of protein, but that the incidence does bear a striking relationship to the consumption of foods that have undergone partial or complete protein-stripping. This evidence will be supported by collateral evidence from Japanese prisoner-of-war camps in the Far East, not so far appreciated, where it will be shown that peptic ulceration became a plague in certain camps where only highly-milled rice was eaten, but virtually disappeared where lightly-milled rice and other unrefined grain was substituted, though the total protein was still barely sufficient to support life. Equally striking evidence in connexion with a different type of carbohydrate will be produced from German sources, dealing with their Eastern front in the last war and the Russian prisoner-of-war camps.

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CHAPTER V

Racial Incidence of Peptic Ulcer

Incidence where no Protein is removed from any Carbohydrate Food

AFRICA

THERE will now be set out some examples of the differential incidence of peptic ulcer in various parts of the world, together with the pattern of protein consumption in these places, in order to see what support this furnishes to the argument so far developed from evolutionary and physiological considerations.

In this argument, it will be remembered, peptic ulceration is related not to a low total protein consumption, but to the consumption of carbohydrate foods from which the protein has been partially or completely removed, leading to the stomach becoming intermittently traumatized by relatively unbuffered acid. The distinction between this argument and one relating peptic ulceration to a low total protein consumption is a vital one. The latter argument has been used in the past to explain the frequency of peptic ulcer in certain parts of the world, such as India where manioc (tapioca) is consumed, in which the total protein consumption is exceedingly low, but has failed to explain the still greater frequency of peptic ulcer in Westernized countries, where the total protein consumption is exceedingly high. This difficulty has even led to the view, as will be seen in the next chapter, that two different forms of peptic ulcer exist—a Western form and a tropical form. The present argument, however, surmounts this difficulty without any such complicated view, and furthermore does so in accordance with natural, or if the term be preferred, evolutionary, principles, which inspire confidence. It will be shown, in fact, that the racial incidence of peptic ulcer follows lines not of total protein consumption (and emphatically not of 'stress', which is supposed to be greater in modern civilization), but always of interference with the protein content of carbohydrate foods—protein-stripping.

From the point of view of this argument it is clearly of decisive importance to examine the incidence of peptic ulcer in

communities where, though the consumption of animal, and therefore total, protein is very low, no protein is removed from any carbohydrate food. With the spread of civilization such communities are getting rapidly scarcer, but may, perhaps, best be studied in Africa, to which continent, therefore, attention is now directed. It may be stated that the reports on the incidence of this disease in Africans exhibit a remarkable degree of divergence, but an effort will be made to show that this very divergence lends support to the present argument.

At the outset it is essential to refer to three important considerations. First, in case it be contended that the negro possesses any racial, that is to say congenital, immunity from peptic ulceration, a glance may be cast at the incidence of the disease in the negroes in the United States, where their environment resembles, and is at times identical with, that of the white inhabitants. It may then be stated that when the environment is identical, as occurs, for example, in the armed forces, peptic ulceration occurs with equal frequency in the two races. So A. A. Kirschner[1] has concluded from observations made in a large U.S. military hospital. Many other references show that even when the environment is not thus standardized, any differences in the incidence are of small importance.

As regards the racial purity of the U.S. negroes, studies have shown that a proportion is still of pure stock,[2] but in any case the incidence of peptic ulceration in them differs so greatly from that in the negroes in Africa, now to be described, that any racial differences between the two groups of negroes are readily accommodated.

Secondly, it would be a great mistake to suppose that the clinical manifestations of peptic ulcer in negroes are to any important extent less obvious than in the white races. Not only is the evidence quite to the contrary from the United States,[1] but so it is also from Africa, where, as A. G. Shaper and A. W. Williams[3] have recently pointed out, the clinical history is often as clear and typical as any encountered in English hospitals. In the areas of India and Nigeria, where it will be shown that interference with the protein content of rice and manioc takes place and peptic ulcer is very common, the sufferers experience such severe pain that they often travel many miles to be operated on. Furthermore, these ulcers are specially liable to cause pyloric obstruction, the results of which cannot be hidden.

It is perfectly true that peptic ulcer may be clinically latent in coloured races, but so it may be in the white races. For example, in the United Kingdom to-day, though about 10 per cent of men and 4 per cent of women develop clinical evidence of the disease during their lifetime, the post-mortem evidence is nearer 20 per cent for each sex, and there is no indication that such clinical latency is any greater in one race than in another.

Thirdly, the opinions of well-qualified observers, formed over many years, are not appreciably the less significant because local circumstances preclude their being given statistically. L. S. P. Davidson[4] has made this very point over diseases in Africa. To await the statistical presentation of all such opinions would not only delay indefinitely taking advantage of valuable information, but might also prevent this being done at all, so rapid is the advance of civilization into the areas of Africa concerned. The present author would never admit that experienced local opinion was sensibly inferior to the average run of statistics, but even if it were, it could still be said, after Omar, observing the time factor: 'One glimpse of truth within the clinic caught, Better than in a census lost outright.'

With the above considerations in mind, it is now possible to examine communities in Africa which remove no protein from any carbohydrate food, examples being taken from Natal, Kenya, Ethiopia, the Gold Coast (Ghana), and Egypt. The incidence of peptic ulcer in these Africans can then be compared with that in Africans living on food showing various degrees of sophistication.

Part I

Africans living in a Tribal Manner

Natal and Kenya (Mombasa).—Of Africans who consume unrefined carbohydrates no better example exists than the Zulus of Natal, when living in a tribal manner away from the towns. The total protein consumption in these Africans is so low that kwashiorkor is common amongst them, but what is important to the present argument is that no protein is removed from any of the maize which forms their staple diet. The maize is, indeed, commonly crushed on a grinding stone almost identical with the quern used in ancient Egypt more than 2000 B.C. "From the

flour so produced some blow off the lighter chaff, yet even this is not usual, since it is said that the complete product makes a porridge that 'stays with you longer', i.e., the consumer does not feel hungry so soon. In better-to-do homes many natives have the 'Colonist' type of hand-mill, and the porridge is then normally made from a straight-run, complete meal."[5] These Zulus also eat very little sugar, as will be elaborated later.

Concerning the incidence of peptic ulcer in these Africans, the author is indebted to A. Barker, surgeon-in-charge of the Charles Johnson Memorial Hospital, Nqutu, for the following (1960):—

As regards your specific enquiry, I feel that first I should cover myself by saying that this is a country unit with relatively simple facilities for diagnosis (though we can and do screen if we consider this necessary) and a heavy case load. Thus we probably miss some cases of peptic ulcer which other and wiser and less busy medical men might pick up. I can only answer your question, then, on symptoms suggestive of ulceration, and here do most strongly confirm your idea that symptoms of peptic ulcer are very rare indeed among Zulus living in a more or less unmodified tribal manner. About 25–30,000 attendances are made yearly here, and the yearly admission rate to hospital has been in excess of 3000 patients for the past 5 years. In, say, 25,000 in-patients over the past 10 years we have only had 2 cases of peptic ulcer, while I have not, at least in the past 5 years, seen a single case, except one of the two mentioned above, suggestive of an ulcer.

For another reference on the rarity of peptic ulceration in Africans living almost entirely on unrefined maize, the author is indebted to D. G. Milton-Thompson (1960), who states that during his eight years in medical charge of the Kaloleni Hospital, Mombasa, Kenya, with 100 beds, which helps to care for a population numbering approximately 60,000, he has seen no case of peptic ulcer, including no perforation.

Similarly, the author is indebted to W. M. Politzer, of the South African Institute for Medical Research, for the following, with a somewhat broader sweep (1960):—

Peptic ulcer is seldom seen clinically in the Bantu living under primitive conditions. The cereals consumed by these people are unrefined. They make their porridge and bread from whole maize crushed by themselves. The bulk of their diet is maize. Sugar and sweets are great luxuries. One cup of sugar will last a family for weeks. One cannot imagine how poor these people are, and only the barest minimum which suffices to keep them alive is consumed. All this refers to the rural Masutu, the inhabitants of Basutoland.

(It will be remembered that Basutoland is adjacent to Natal Province.)

It may be interjected here that the high incidence of peptic ulceration noted previously in negroes in the United States was conspicuously absent when they lived largely on 'hominy', which was likewise prepared from unrefined maize. The rarity at that time of this disease in them compared with the whites was delineated by U. Maes and E. M. McFetridge[6] in 1936 and again by F. Boland[7] in 1942. Unfortunately, this distinction has been lost sight of to-day, but is of deep significance to the present work.

To revert to the Africans now under consideration, their consumption of sugar is so important, *vis-à-vis* the argument being advanced in these pages, that the author is indebted to W. R. Carr, Food Technologist of the Government Analyst's Laboratory, Salisbury, Southern Rhodesia, for the following additional note on this subject (1960):—

> The amount of refined sugar consumed by primitive Africans is, to all intents and purposes, nil. I think that a survey would show that purchases in remote areas would be less than 1 oz. per head per week. Even the Africans in urban areas consume only 1 lb. per week.

It must not be inferred from what has been written above that maize eaten by primitive Africans is everywhere unrefined. This is far from being the case; in many areas the maize is refined to a very considerable extent, even under tribal conditions. Full details of this have been published by W. R. Carr,[8] and this variation in practice in different parts of Africa, so important to the present work, has constantly to be borne in mind when examining the incidence of peptic ulcer in a given area.

Ethiopia.—Lest it be considered that there is anything particularly favourable about maize, as opposed to the unrefinement of the maize, in the prevention of peptic ulceration, attention may now be turned to an entirely different type of grain, closely related to millet, called teff. Teff forms the staple food supply in Ethiopia, and is of particular interest to the present work because the peasants in that country eat it unrefined.

The diet of these peasants has recently been exhaustively surveyed,[9] and it is possible to state categorically not only that unrefined teff supplies about two-thirds of their calories, but also that their consumption of sugar, the most refined of all

carbohydrates, is negligible, being $\frac{1}{7}$ oz. per person per day (as opposed to some 5 oz. in the United Kingdom), much of this sugar being consumed in the towns.

If, therefore, the argument presented in these pages has any substance, it is clear that peptic ulcer should be very rare amongst Ethiopian peasants. Through the kindness of B. O. Barry, Medical Director of the Princess Tsehai Memorial Hospital and Medical School, Addis Ababa, the author has been put in touch with M. Tausjo, radiologist in charge of the large Norwegian Lutheran Mission Hospital, and also Provincial Medical Officer of the Ethiopian Government, at Irgalem, in the heart of Ethiopia, to whom the author is indebted for the following (1961):—

I have received your letter, and shall be only too glad to give the information I have at hand. I have been working in the interior of Ethiopia for 12 years in a general hospital, with a yearly number of patients around 50,000. Let that be the basis for the answers.

1. There is very little peptic ulcer among the natives. Rough statistics show that only 2 out of 1000 patients (not of the population) have diagnosed peptic ulcer. And these few have always been people who are more or less Westernized. I cannot remember a single case of peptic ulcer among the country people who live in their own traditional style of living.

2. The people in the interior do not eat rice at all, as it is not cultivated in the country. The main food in the area is either teff or the false banana-tree leaves. The Amhara tribe uses teff, which is either ground by hand or by machine-driven mills, but in no case is there any degree of refinement. This is, for instance, proved by the fact that one may get teff more or less mixed with the sand from the soil where it has been threshed. . . .

The natives eating teff also regularly consume a large quantity of hot spices. It seems to me that these spices have nothing whatever to do with peptic ulcer. Some five years ago I was in Europe and heard the statement that in Ethiopia the people eat such hot, strongly-spiced food that there was naturally a lot of peptic ulceration there. I had to oppose this statement and say what the life here has taught me. My statement was accepted with astonishment, because it did not coincide with the prevailing opinion on the subject.

The fact that a very high consumption of red peppers by these natives has no tendency to cause peptic ulceration will be shown to have considerable importance when discussing the incidence of the disease in India and Nigeria.

Meanwhile in Addis Ababa and other large towns in Ethiopia peptic ulcer is common.[10] In these towns there are bakeries

producing white wheaten loaves and rolls, and sugar is eaten in considerable and ever-increasing quantities.[11, 12]

Gold Coast (Ghana).—The rarity of peptic ulceration amongst the natives of the Gold Coast in Africa has recently been described by E. Onori.[13] This observer begins his paper with the following: "When compared with Europe, where cases of peptic ulcer are prevalent, the almost total absence of this disease in the surgical wards of the Gold Coast Hospital is most surprising."

The food of these natives consists mainly of rice, millet, and maize, the grain in each case being pounded in a mortar and eaten in a very high degree of unrefinement.[14] In the case of maize, it is frequently eaten as whole grains, either raw or roasted.

Egypt.—The writer was led to inquire into the incidence of peptic ulcer in Egypt through a remark by Ogilvie[15] on the rarity of this disease in the Egyptian fellaheen, and was fortunate enough to secure the opinion on this point of S. Azmy, who is highly qualified in this country, and who has long been interested in gastro-intestinal complaints in his own. In personal communications (1960) Professor Azmy states that though peptic ulceration is becoming more frequent amongst the well-to-do classes in Egypt, "living on a diet similar to that of the same class in Europe", it is still a very rare disease amongst the poor villagers. The nutrition of these peasants is deficient in animal protein, and they are infested with intestinal parasites, so that "they suffer from avitaminosis, anæmia, etc., yet despite all these factors, peptic ulcer is very rare amongst this class of people".

If the diet of these Egyptians is examined, it is seen to consist largely of unrefined grain. The author is likewise indebted to Professor Azmy for details on this point:—

> Bread is the staple food, but the flour from which it is made differs according to the region. In the Nile Delta region the flour is mainly maize flour and is not refined. In the northern parts of the Delta rice is commonly consumed, which is half-refined, being only 100 per cent refined in big towns and cities. . . . In upper Egypt the bread is usually made from millet flour, which is unrefined. . . . The Bedouin in the desert use non-refined barley flour; they grind the barley with a hand-rolled millstone.

Part II

Africans leading a More Civilized Existence

Lest it be considered that the rarity of peptic ulceration in the above communities, pursuing a traditional and primitive mode of life, has anything to do with freedom from stress, which is supposed to be greater under civilized conditions, let it be stated at once that communities living in a traditional manner in Nigeria, India, and elsewhere, but eating rice or manioc from which approximately one-third of the protein is removed before consumption, suffer a heavy incidence of the disease. The crucial fact, as stated at the beginning of this chapter, is that the incidence does not in the least follow lines of stress, but emphatically does follow the lines of interference with the protein in carbohydrate foods. Before turning to these new communities, however, it is essential to devote a few words to the incidence of peptic ulcer in Africans who are no longer living in a tribal manner, but under relatively modern conditions, with access to sophisticated foods.

With regard to this latter incidence, it will be appreciated that, although no criticism is intended here, reports given by various observers on the subject will have little bearing on the present argument unless they are related to the food structure in the areas concerned, and this, alas, seldom seems to be done. What appears to happen instead is the following sequence. Into an African community, previously regarded by those with long clinical experience there as almost entirely free from peptic ulceration, there spreads a more sophisticated mode of existence—and this is taking place to-day very fast all over Africa. As A. W. Williams[16] puts it so well: "Changes in the eonomic and cultural patterns of African society are taking place more rapidly than ever before, and industrialization, the growth of cities and ease of communication are establishing a common pattern of life in areas which until recently provided striking contrasts in living conditions and ways of life. These contrasts which might throw light on the aetiology of disease are disappearing, and any delay in initiating research will result in the loss of valuable and unique opportunities." With the appearance, then, of modern conditions the food structure changes in the community, especially as regards the consumption of sugar and refined grain. Now peptic ulceration begins to

become noticeable, and soon even prominent, and at this stage doubts are felt about the previously reported rarity of the disease. It is considered that earlier observers may have been mistaken in their clinical judgement. What appears to the present author to be the serious error of re-assessing former clinical experience, instead of the new nutritional environment, appears to be made over and over again.

It will thus be understood that compared with reports on the rarity of peptic ulcer in African communities, those on its frequency convey little of value. In the former communities the food structure is usually primitive, highly stereotyped, and easily assessable, whereas in the latter communities it is usually sophisticated, variable, and assessable with difficulty. In fact, reports on the frequency of peptic ulceration in sophisticated African communities reveal little that cannot already be learnt from the United States. Nevertheless, the following reports must be carefully examined.

Writing of peptic ulcer in Africans in Rhodesia, B. J. Baldachin and P. E. S. Palmer[17] state (1959):—

> . . . It has become obvious to us within the past year or two that this condition is by no means as uncommon in African patients in Southern Rhodesia as has been formerly supposed. During the past six months 35 peptic ulcers have been diagnosed radiologically in this hospital. Of these, 28 were duodenal ulcers and 7 gastric. This is in marked contrast to the experience of one of us, who cannot recall having seen more than one case of peptic ulcer in an African in this centre during the years 1949–54.
>
> We have gained the impression that this sudden increase in the incidence of peptic ulcer may be more real than apparent. . . . Although diagnostic facilities have improved recently, and this undoubtedly accounts for some of the increase, we feel that it could not possibly account for the total observed increase in incidence. This opinion is strengthened by the fact that approximately 35 per cent of cases referred for barium meals during the past six months—for whatever reason—have had peptic ulcers demonstrated radiologically. Any doubtful cases have been excluded from the numbers quoted above. . . .
>
> It may well be that more modern standards of living are a potent aetiological factor.

I am greatly indebted to B. J. Baldachin for the following additional note (1960):—

> We have recently commenced a detailed investigation of epidemiological aspects of peptic ulcer in the African population, but it will probably be at least another two years before we shall be able to furnish

any figures. However, without reference to statistics, we have been struck by two obvious factors of some importance. Firstly, the number of peptic ulcer cases among the local African population appears to be increasing rapidly, especially during the past two years. Secondly, we have gained the impression that the majority of these patients come from the more educated strata, who are employed mainly as school-teachers and clerical workers, and who are adopting civilized standards and habits —including, presumably, some change of diet away from the traditional. . . .

With regard to the constituents of the diet itself, the staple food of the Africans in Southern Rhodesia is maize meal, the degree of refining of which varies. The more refined and whiter variety is certainly gaining in popularity. . . . White bread is freely consumed, as is highly refined sugar.

It follows, therefore, from the present argument that the increase in peptic ulcer in this area is not merely understandable, but indeed, if the argument be correct, inevitable.

Similarly, writing of the disease in Nyasaland, S. V. Humphries[18] states (1959):—

From my own experience I think that the incidence of peptic ulcer in Africans varies in different parts of the continent.

While I was a mine medical officer for native hospitals in the Transvaal for ten years I did not see a single case of clinically recognizable peptic ulcer or of gastric carcinoma, either in my own practice or at clinical meetings of the Transvaal Mine Medical Officers' Association, where cases of these conditions, had they been observed, would almost certainly have been shown. As a mine medical officer for African patients in Sierra Leone and the Gold Coast over a period of 18 months, I did not see a case of either disease. But here in Lilongwe I have in six months performed two partial gastrectomies—one for duodenal ulcer in a woman and one for carcinoma of the stomach (proved microscopically) —and four gastro-enterostomies: two for gastric ulcer and two for pyloric obstruction, of which one was probably carcinoma. The last five were all males.

I am deeply indebted to this observer for the following additional information:—

I have not observed the natives grinding their maize here. They send it to be crushed in the mill at Lilongwe.

And the further note, after examining this maize:—

The mealie meal (maize flour) after removal of husk and grinding is very white, and I can find no sign of any husk in it. . . . Bread is seldom eaten in the village. The bread which they eat in towns is white bread. I have not seen a brown loaf since coming to Lilongwe.

Similarly, again, abundant evidence has been set out[3] of the prevalence of peptic ulcer in Kampala, capital of Uganda, especially in the nursing staff at the Mulago Hospital and in undergraduates at the Makerere University College where[19] "... among educated young African men eating a European type diet the incidence of peptic ulcer is similar to that estimated in the United Kingdom by Doll, Jones and Buchstzch". When, however, the author asked at this medical school to be referred to a surgeon practising in rural Uganda, he was referred through the kindness of Mr. J. Cook, surgeon, to E. H. Williams of the Kuluva Hospital, from whom he was privileged to receive the following (1959):—

> It is a fact that I rarely see peptic ulcer in the African patients with whom I deal, and when I do see the condition it is usually amongst those who have adopted European ways of living and eating, and who are school teachers or whose occupations are those possible for educated people. . . . I would say that the incidence of peptic ulceration is definitely on the increase and seems to run parallel to the improvement in living standards.
>
> I have always been curious that peptic ulceration is so uncommon among these people, because superficially there are various factors which would seem to encourage it. For instance meals are infrequent and large. The food is prepared from various grains and is rough and starchy.
>
> The grains are as follows: Small finger millet and tall millet or Kaffir Corn. These are ground to flour between stones. Sugar is a recent innovation in the diet.

Finally, to revert to the Zulus of Natal, discussed at the beginning of this chapter, these begin to lose their freedom from peptic ulceration when they move into the towns and start eating refined maize and increased quantities of sugar. Through the kindness of G. D. Campbell at Durban the author is indebted to Professor D. S. Chapman for the opinion that, although peptic ulceration is still much rarer in the Africans in that city than it is in the Europeans, it is becoming increasingly evident in such Africans as medical students, brought up in higher-class families and eating sophisticated foods, who not infrequently develop intractable ulcers even at an early age. Peptic ulcer cases seen in the King Edward VIII Hospital, Durban, have recently been described,[20] and though these are very uncommon in this hospital, they are much commoner there than in the Charles Johnson Memorial Hospital at Nqutu, previously referred to.

This difference is still better seen in Kenya to the north, where the absence of peptic ulcer cases in the Kaloleni Hospital in Mombasa, previously referred to, caring for tribal Africans living chiefly on unrefined maize, may be contrasted with the prominence of these cases reported recently in the King George V Hospital in Nairobi,[21] caring for Africans living largely under modern conditions and having access to more sophisticated foods. In this latter reference the rising nature in the incidence is emphasized, and also the fact that the symptoms of the disease "do not differ appreciably from the set pattern of duodenal ulcer".

It may be added that the low incidence of peptic ulcer amongst the native populations of some African cities, like Durban and Johannesburg, which has been carefully documented,[20, 22] is in the writer's opinion due to the fact that substantial quantities of unrefined maize are still eaten there;[23] and to the fact that the consumption of refined sugar in these communities is still much less than in the United Kingdom; and also to the personal disposition of the members of some of these native tribes, *especially the Zulu tribe*, which is referred to under Personality in Chapter XI (p. 96). As regards the maize, though African natives, like the majority of mankind, tend to eat a refined product when they can get hold of it, the great mining and industrial houses in cities like Johannesburg, which provide meals for their labourers, usually issue unrefined maize, which accounts for the large amount of this substance still consumed in these areas.[24] As regards sugar, it has recently been shown that only 5 per cent of urban Zulu males, and 21 per cent of urban Zulu females, consume as much as 2 oz. of crystalline sugar daily.[25]

The incidence of peptic ulceration in sophisticated negro communities in Africa will not be pursued further. As far as the present argument is concerned, this incidence is of no greater significance than it is in such communities in the United States, and unless the incidence reported is carefully related to the food structure, which is seldom the case, nothing constructive to the present argument can be learned from it. It is always the incidence in the rapidly decreasing number of Africans still living in a *tribal* manner, with a known food structure, that is of such paramount value in the study of the disease.

Exactly the same argument applies to post-mortem studies on incidence of peptic ulcer in Africans. This includes even the series of post-mortems carried out by A. B. Raper,[26] who found an

average of 15 per cent of ulcers and scars in 647 African males, belonging to four tribal groups, who had, however, not been living in a tribal manner, but in and around Kampala, capital of Uganda, and who had mostly been treated in the Mulago Hospital. In this series every attention was paid to the pitfalls described by Ivy, Grossman, and Bachrach,[27] which led these authors to discard thousands of post-mortems from the past, in Europe, as unacceptable in the study of the disease. In the present post-mortems Raper made a deliberate search for ulcers and scars, and his results necessarily impair those of earlier studies by other investigators, showing practically no peptic ulcer in Africans, in which this specific search was not made. Though this investigation commands the immediate respect of all who examine it, yet in the quest of the causation of peptic ulcer pursued here it is of little or no assistance, for the following reasons:—

1. Nearly all the subjects of the study, having been living in or near Kampala for a long time, even when immigrants from other parts of Uganda, had been in contact with modern civilization, with access to sophisticated foods.

2. In keeping with this, the ulcers were of the type found in the Western nations, and not at all those of the type found in the areas of India and Africa where protein is stripped only from rice and other grains, and from manioc. It will be shown shortly that the ulcerative process in these cases is accompanied by the formation of characteristic masses of fibrous tissue.

3. A large number of the subjects were from the poor and destitute. These are just the types, as will be seen later in discussing India and Indonesia, who have the highest incidence of peptic ulcer, which on the present argument is because they are dependent on processed foods bought from the bazaars rather than on unprocessed foods prepared in their own homes, and also because they cannot afford to buy any appreciable amount of compensatory animal protein. This applies just as much to the maize in Africa as to the rice in India and Indonesia, and in the present case there must also be added the consumption of white flour and refined sugar. It is noteworthy that the least impoverished of the four groups had the lowest incidence of ulcer.

4. The range of incidence in the four groups studied was from 22 per cent to 4½ per cent and the present author would advance without hesitation that if a fifth group had been added, consisting of subjects who had been living in a tribal manner on natural

foods, prepared with their own hands, the lower limit of the range would have been 0 per cent.

The word 'ulcers' in Raper's study was used to cover both active ulcers and scars of old, healed ulcers. Half the 'ulcers' approximately were scars, and the question arises why the other half (active ulcers) were mostly latent. The answer would appear to be twofold. Firstly, most of the ulcers were small and mild. The author is indebted to Dr. Raper for the information, amongst much other, that "another point in favour of mildness was that deep penetration, involving adhesion to the pancreas, etc., was uncommon". Secondly, the subjects, being frequently poor and destitute, had less opportunity of reporting symptoms.

That ulcers in negroes cause no lack of symptoms has already been drawn attention to, and the reference given on the clarity of the clinical picture, by Shaper and Williams,[3] dealt in fact with natives in the Mulago Hospital, Kampala. It is certain that ulcers must be missed more frequently in the more primitive communities, but that would not justify doubting the clinical judgement of those working in such communities, who fully realize the possibility of missing some of the cases and yet are quite capable of assessing whether the disease is frequent or rare.

In concluding this chapter it will be seen that the diverse reports on the incidence of peptic ulcer in Africa, in which the experience of later observers is often in conflict with that of earlier ones, find reconciliation in the argument presented here. In the case of India, to which we now turn, it will be seen that the different incidence in different regions, which in this case is agreed to by all observers, is equally explained.

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CHAPTER VI

Racial Incidence of Peptic Ulcer—*continued*

Incidence where Protein is removed from Rice and Manioc

NIGERIA AND INDIA

So far the racial incidence of peptic ulcer has been studied in communities which strip no protein from any carbohydrate food. The incidence will now be studied in communities which first begin to strip this protein in the processing of rice and manioc. Before examining this incidence it will be essential to devote a few words to the processing treatments themselves.

Rice.—The milling of rice is too complicated a subject to be mentioned here other than in outline. Unlike most other grains, the rice grain is firmly adherent to its husk, the removal of which requires considerable physical force. This is achieved in a hulling machine. The brown outer coat, or pericarp, of the grain itself is then removed by milling, the removal being incomplete (in lightly milled rice) or complete (in highly milled rice). Sometimes the rice is steamed beforehand (parboiled) to make the subsequent milling easier, and sometimes it is polished afterwards, to make its appearance more attractive. It is not surprising, therefore, that no fixed figure can be given for the loss of protein that takes place in the refining of the rice. But it averages about 15 per cent and can reach 30 per cent.[1–3]

A large proportion of the world's rice, however, is still 'home-pounded' by the natives who grow it. This procedure usually corresponds to the lightest degree of milling described above, and little protein is lost. Nevertheless, if the natives want to do so, they can achieve virtually a complete separation of the pericarp,[4] as may happen in the case of maize, previously described.

There is a tendency all the time for more and more rice to be milled, and highly milled at that, the reasons being threefold:—

1. Milling saves an enormous amount of labour, and so the cost of the rice falls.
2. Highly milled rice keeps much better than lightly milled rice.
3. Highly milled rice is whiter, and its prestige value rises with the whiteness.

As an example of the spread of rice milling in nearly every country where this food is grown, it may be stated that the first rice mill in India was set up (in Madras) in the year 1900. The early mills were small ones, employing about five persons, the larger mills coming later. The spread has been rapid; excluding small establishments, that now exist in thousands, the number of large rice mills increased from about 500 in 1921 to over 1000 in 1937, and approached 1250 in 1944; and the increase continues.[5]

The milling of rice does not, unfortunately, complete the story of protein-stripping in this grain. Far from it. When natives cook the rice it is always first washed in water, to remove debris. This washing varies in its intensity in different communities, but if the outer coat of the grain has been removed by milling, then the washing invariably removes a further appreciable quantity of protein. An average figure for this loss would be about 10 per cent. If the water in which the rice is subsequently boiled is thrown away, which usually happens[6] (especially in southern India), a further loss of protein ensues, which raises the loss already incurred in washing to about 15 per cent.[7]

The protein content of rice, which is less than that of other grains, varies with the part of the world in which the rice is grown, but taking the figures of Platt, Kirk, and Williams, and Rosedale,[8] it averages about 9 per cent. The combination of milling, washing, and boiling reduces this figure by approximately one-third. At the same time these procedures hardly touch the carbohydrate present, so that the protein calories decline from about 10·6 per cent to about 7·3 per cent. As the poorer rice eaters frequently get no appreciable quantity of protein from any other source, it will shortly be seen that this decline has serious consequences.

(The actual loss of buffering power on the gastric acid, resulting from the loss of protein in the rice, is given in Appendix A.)

Manioc.—The plant *Manihot utilissima*, known in some parts of the globe as manioc and in others as cassava, is the plant from which tapioca is derived, and in one form or another provides the staff of life to many millions of human beings throughout the world.

The tubers of the manioc plant, which are the edible part, readily produce prussic acid in their outer coat as a protection against marauding insects, and mankind is therefore forced, when eating the tubers, not only to peel them and wash them, but also

to throw away any water in which they have been cooked or otherwise processed.

The original manioc tubers contain at least 3 per cent of protein calories,[3, 9] resembling in this respect apples in this country, with which they might well compare as a healthy food, but after the boiling—which cannot be avoided—the protein calories are reduced to about 2 per cent. This ignores any loss of amino-acids which, as stated in Chapter IV in connexion with similar procedures with the potato, also contribute to the buffering of the gastric acid.

So far the loss of approximately one-third of the protein is reminiscent of that which occurs in the full treatment of rice, but in the case of manioc the loss is likely to be carried much further. To some extent this takes place in the home, since the natives are liable to subject the tuber to considerably more than simple boiling. Even slicing much increases the loss of protein in boiling, as has already been seen in the case of potatoes, but natives may not only pass the freshly peeled manioc tubers through fine-meshed sieves, but in addition may squeeze the moisture out between heavy stones, before drying the pulp over the fire or in the sun to make a form of flour. These steps reduce the protein calories to approximately 1½ per cent, but even so, do not compare with the procedures taking place in the tapioca factories. In these factories it is estimated that the processing of 100 lb. of manioc tubers requires 100 lb. of water for washing, 450 lb. for rasping and sieving, and 1000 lb. for purifying.[10] As a result of this leaching on a grand scale the protein calories in the tapioca of commerce are reduced to under ½ per cent. It is fortunate for the natives that this tapioca, like the highest polished rice, is largely exported for consumption by other races (such as our own). Nevertheless, the steps taken by the natives themselves are serious enough, because the eaters of manioc are almost invariably amongst the poorest type of humanity and are seldom able to supplement their protein intake to any appreciable extent from other sources.

Before leaving the subject of protein-stripping in rice and manioc, attention must again be drawn to the fact that it is not only the loss of protein that is important in these refining procedures. Two other results must also be remembered—the big reduction in the amount of chewing necessary, with a corresponding fall in the impregnation of the food with saliva, itself a natural buffer of the gastric acid; and also the big reduction in

the gastric emptying-time, which may expose the duodenum to additional trauma from unbuffered acid. These two additional factors, as previously pointed out, make it impossible to assess the consequences of the processing procedures solely on a basis of protein contents.

It is against the above background of interference with the natural qualities of rice and manioc that the racial incidence of peptic ulcer will now be pursued.

Nigeria and India.—P. G. Konstam[11, 12] has shown that the southern parts of Nigeria and India, though geographically so far removed from each other, are, with regard to the foods eaten by their inhabitants, closely related, especially when compared in this respect with the northern parts. Thus, in southern Nigeria manioc preparations form the staple food, whereas in northern Nigeria the main foodstuffs are various forms of millet, eaten unrefined, as exhaustively recorded in both regions by B. M. Nicol.[13, 14] Similarly, in Madras in south-eastern India the basic food is highly milled rice and very little else, whilst in Travancore in south-western India manioc preparations again form the staple food. These areas may be contrasted with areas to the north, where the rice eaten has a much greater tendency to be home-pounded (which, as already explained, interferes with the protein content appreciably less), and especially with the Punjab, where unrefined wheat and unrefined maize[15] mostly replace rice, and where no manioc is eaten.

How closely the incidence of peptic ulcer follows the degree of protein-stripping taking place in these territories is best shown in the following quotation from the above papers by P. G. Konstam, to whom the author is greatly indebted, not only for information published but also for that supplied in many personal communications (1960):—

> Whereas peptic ulcer is ubiquitous in the countries of the western world, its occurrence in Nigeria is, in the main, confined to the southerly parts of the country. From personal travelling and correspondence with colleagues, it appears that in the southern areas . . . the disease is commonly seen, whereas in the northern . . . regions there is little evidence of it. Similarly in India, according to Dogra,[16 a, b] whereas there is an incidence of 143 per 100,000 population in Madras in the south-east (and probably even higher in Travancore in the south-west), it is only 9 per 100,000 in the Punjab in the north.

Mr. Konstam supports this latter reference by evidence collected personally, based on the ratio of ulcer operations to routine

herniotomies performed in key hospitals in these areas. The very low incidence in the Punjab has also been extensively commented on by others, such as McCarrison,[17] who emphasized *inter alia* the unrefined nature of the grain eaten there.

It should be added that in the ulcer belts both of India and Nigeria the incidence drops at once in localities where the consumption of protein-stripped foods is reduced or absent, such as in those where dhal and other protein-rich pulses are consumed, and in coastal districts where the consumption of fish alters the whole picture. It should also be added that in the ulcer belts the incidence is much lower amongst the richer members of the communities, who can supplement their diet with additional protein in this way.

Nigeria.—The commonness of peptic ulcer in southern Nigeria, where manioc is eaten, has been described by Konstam, as indicated above, and also by Ellis,[18] but the rarity of the disease in the north, where unrefined millets form the staple diet, has been less well documented. The author is therefore indebted to E. T. Mess, of the Wusasa Hospital, Zaria, Northern Nigeria, for the following (1961):—

> In reply to your letter our in-patient admissions last year were 486 men and 1960 women, whilst new out-patients totalled 2278 men and 6769 women. (The number for women includes maternity cases.)
>
> As regards your inquiry reference cases of peptic ulcer, the incidence of this disease here amongst Africans living in a tribal manner appears to be absolutely nil. I have in the course of twelve years seen two cases of pyloric stenosis of a fibrotic type among this class of people. There was, however, in those two cases no reliable history at all typical of recurrent ulceration.

It will be noted that this evidence is in close agreement with that from Ethiopia given earlier, where a very similar grain to millet is eaten (teff), and where the unrefinement of the grain is likewise absolute.

India.—In order to bring out the incidence of the disease in India in relation to the consumption of milled or home-pounded rice, the table on p. 46 has been compiled from tables in J. R. Dogra's "The Incidence of Peptic Ulcer in India", quoted above, and W. R. Aykroyd's[19] "The Rice Problem in India".

It will be seen how the marked predominance of peptic ulceration in Madras accompanies a similar predominance in the consumption of milled rice. These figures are deeply significant in their own right, portraying as they do a situation that existed

before the Second World War. Is that situation the same to-day? No comparable figures are available, but it seems certain that Madras still has a clear lead, both in the consumption of milled rice and in the incidence of peptic ulceration. How long that lead

State	Population 1931	Number of Hospital Cases of Peptic Ulcer 1939	Incidence per 100,000 of Population 1939	Percentage Rice-eaters eating Milled Rice as opposed to Home-pounded Rice 1940
				per cent
Bengal	51,087,338	16,976	33	25
Madras	46,740,028	57,397	143	70
Bihar	23,676,028	8851	37	10
Orissa	18,653,555	4132	29	20
Punjab	23,580,852	2131	9	*
Assam	9,247,857	926	10	15
United Province	49,614,833	4460	11	15

* Rice mostly replaced by unrefined wheat and maize.

will last is another matter. The spread of the rice mills, already referred to, must be considered. As the Government of India states in its publication on rice:—[20]

The extended use of the rice mill is bound up with other changes in the life of the people. The development of transport and improvement of roads enables the paddy-grower to bring his grain to the mill. The motor bus, rattling everywhere about the countryside, has loosened the bonds which attach the villager to his own plot of ground and traditional manner of life. Cheap electric power is obtainable over wide areas, and other sources of power—the steam and internal combustion engine—are familiar and obtainable. All these factors have played a part in the abandonment of the ancient practice of hand-pounding.

Certainly no reliance can be placed on the villagers of India consuming home-pounded rice to-day.

Very relevant to the subject of the removal of protein from the rice in India is the following, for which the author is indebted to S. F. Thomas, from Orissa (1961):—

I also am very interested in the problem of peptic ulcer, as we see a great deal here. I operate on about 100 cases a year and see many more who are sent away for medical treatment. I run a special gastric clinic once a week and see about ten cases a week. The point that interests me

is that our cases come mostly from the plains around us, and very few from the tribal people who live in the hills (at about 2300 ft.) in which our Hospital is situated. Because we have a reputation for surgery these cases come from anything up to 200 miles, but seem mostly to come from one area. We have recently received from the Indian Council of Medical Research funds to carry out a research project into the cause of this discrepancy. I am sure the cause is nutritional, but cannot as yet make any dogmatic statement about it. . . . One important fact is that in the hill area the people drink the rice water, but in the plains throw it away or give it to the animals. This water, I believe, contains much protein and is highly nutritious.

To revert to the geographical distribution of peptic ulcer in India, it is noteworthy that the disease, which is common in the south, where highly milled rice and manioc are consumed, and rare in the Punjab to the north, where unrefined wheat and maize form the staple diet, becomes common again in Kashmir still further to the north. Thus the disease accounts for 3·3 per cent of hospital admissions in Madras to the south-east,[21] 5·7 per cent in Travancore to the south-west,[22] 0·9 per cent in the Punjab,[23] but 2·5 per cent in Kashmir.[23] It is highly significant that the food in Kashmir changes back again to rice—mostly *milled* rice.[24] In addition the Kashmiris are great drinkers of tea, of which they take 20 to 30 cups a day.[25] This tea is sometimes sweetened with sugar, of which the population's average consumption is ½ oz. daily,[26] but normally it is flavoured with salt and fat; in any case it contains no protein and is often drunk by itself.

Peptic Ulcer amongst Millet-eaters.—Attention at this point may be directed to the eaters of millet in India, which consists of several varieties, such as *jowar*, *bajra*, and *ragi*. These millets, the consumption of which carries little social prestige, are eaten unrefined by the labourers and other poor people who depend on them, only being refined for children or by better-to-do persons. Clearly it is important to this work to be able to show, if possible, that peptic ulcer is as rare in such consumers in India as it is in those of similar grains in Nigeria and Ethiopia.

This is not an easy thing to achieve, chiefly because the consumption of millet in India is liable to be accompanied by the consumption of other foods, such as milled rice. There are no references on the subject, and the author is indebted for local experience to the following observers:—

1. D. A. Andersen,[27] who has made a prolonged investigation of the diet in millet-eaters in the Ahmednagar District, towards

central India, in connexion with a study on the formation of bladder stones (shortly to be published in this country), emphasizes the precautions necessary in interpreting statistics from India, but states that it may be taken as certain that the peptic ulcer problem in this district is much less serious than in southern India.

Actual figures from the Evangeline Booth Hospital, Ahmednagar, show that admissions for peptic ulcer number about 1 per cent,[28] as against 3·3 per cent in the Government Hospital, Madras, and 5·7 per cent in the Medical College Hospital, Trivandrum, Travancore, previously quoted—and, incidentally, 2·3 per cent for hospitals in England and Wales.[29] It is also the opinion of the medical staff that the peptic ulcer cases chiefly come from the rice-eaters in the district, though these numerically are very much in a minority. Thus E. Pedersen, at present in charge, writes (1962):—

> During the past eight years I have worked in four different, and from the nutritional standpoint, distinct parts of India, viz., Gujerat, the Punjab, Andhra Pradesh, and Maharashtra (Ahmednagar). Whilst I have no statistical data on hand to support the contention, I have the definite impression that peptic ulceration is much less common amongst communities using *unrefined* grains, even though they consume an abundance of chillies and spices.

2. C. B. Shrisunder, in charge of the Mission Hospital, Pachod, Aurangabad, also towards central India, states (1961):—

> I have been working in this part of India for three years, and in my practice I have found very few cases of peptic ulcer. Our out-patients number 6000 to 7000 a year, and of this number there are usually about 10 with peptic ulcer. The food of all of them is mainly unrefined millet, and very occasionally wheat and rice. But they are great smokers. . . .

3. Finally F. Tovey, surgeon at the Holdsworth Memorial Hospital, Mysore, where both millet and rice are eaten and where peptic ulcer cases account for 2·5 per cent of the in-patients, states the following (1961):—

> I had previously prepared a map of the areas from which our duodenal ulcer patients come and found that almost none come from an area south-west of Mysore where there is repeated famine, due to lack of rain. In this area much more millet is eaten than rice, because it is an easier crop to grow there.

It is not possible to pursue the matter further here, but it is clear that peptic ulceration in India is relatively rare amongst the

millet-eaters, and the author is confident that if some of these did not also consume jaggery (sugar) and milled rice, it would be as rare in them as in the eaters of similar unrefined grains in Ethiopia and Nigeria.

Relationship of Hot Curries.—At this stage it is essential to consider whether hot curries play any part in the causation of peptic ulcer, which they have frequently been considered to do in the past, not only in southern India but also in southern Nigeria—but nearly always by casual observers, not by those deeply involved in the problem. It is easy to understand the attribution, too. As Konstam[12] puts it: "Nearly one ounce of red chillies is taken daily by the Madrassis, and even more by the Andhras to the north. The fire of this substance has to be tasted to be believed."

The present author, however, considers that the consumption of hot spices can be excluded as playing any part whatsoever in the aetiology of peptic ulcer, for the following reasons:—

1. Since these chillies grow in the natural environment of those who eat them, and are eaten voluntarily and in an unprocessed state (i.e., they are not subjected to more than simple cooking to form a relish, or simple drying to form cayenne pepper), their incrimination would be incompatible with the law of adaptation. (It will be shown later that simple cooking does not invalidate this law.) This reason takes first place, not last place.

2. The lines of incidence of peptic ulcer do not follow the lines of high consumption of hot spices. For example, the consumption of red chillies, which has been closely studied, is 4 g. per head per day in Madras,[30] 7 g. in Ceylon,[31] and 5 g. in Ethiopia,[32] but whereas peptic ulcer is common in Madras (accounting for 3·3 per cent of hospital in-patients, as previously quoted) it is relatively rare in Ceylon (accounting for 0·2 per cent of hospital in-patients,[33] many of the cases in Colombo coming from southern India), and it has already been seen to be extremely rare in rural parts of Ethiopia. Furthermore, the lack of association is also obvious in India itself, where areas with the highest consumption of hot spices (e.g., the Deccan) frequently reveal very little peptic ulceration amongst the inhabitants.[12]

3. Although, as already stated, such an obvious possible cause has occurred to many observers, those best qualified to form an opinion have firmly rejected it. Thus, Somervell,[34] after working twenty years in the large mission hospital at Travancore, with the

experience of 4000 duodenal ulcer operations, of which he had personally performed over 2000, rejected this cause. He pointed out, as above, that even hotter curries are consumed in other parts of India, where peptic ulcer is relatively low, and also that the continued consumption of these hot spices does not impede the healing of peptic ulcers when they are being treated medically in hospital.

The truth appears to be, as advanced by Konstam, that a big consumption of hot spices usually indicates a tasteless and monotonous diet, which is only too likely to consist of either manioc or highly milled rice.

Relationship of Avitaminosis.—As regards a lack of vitamins being a possible cause of peptic ulcer, it will be clear that the present argument does not ascribe any aetiological significance to this. Not only is there no such lack in the Western nations, where peptic ulceration is highest, but even in the countries like India, where there often is a lack, there is no relationship between the two. Thus Konstam, who made a mission from Nigeria to India specifically to ascertain this point, states:—[11]

> As for vitamins they cannot, in my opinion, be held responsible for causing ulcer. Vitamin A is readily available as carotene in South Nigeria in red palm oil. Thiamine on the other hand, short in the Southern Nigerian diet, is amply supplied in South India. . . . Riboflavin is deficient in all areas irrespective of ulcer incidence.

Forms of Peptic Ulcer in Tropical Countries.—Konstam has pointed out that peptic ulcer in tropical countries tends to take two different forms. In the first form, well seen in the rural areas of southern India and southern Nigeria, the ulcer, which is nearly always duodenal, is very chronic in nature. Characteristic masses of fibrous tissue form, involving the first, and even the second, part of the duodenum. Reflecting this fibrosis, perforation and hæmorrhage are unusual. Clinical symptoms, however, are marked and include severe pain. From the fourth year onwards obstructive vomiting is liable to dominate the scene.[35] Surgical treatment on one or other count frequently becomes necessary. The relative freedom from perforation is well seen in the figures quoted by Konstam for the Government and General Hospital, Madras, where, in 1955, perforations were responsible for about $5\frac{1}{2}$ per cent of ulcer operations, whilst at the Medical College Hospital, Trivandrum, in Travancore, the figure was $7\frac{1}{2}$ per cent.[12]

In the second form the ulcer resembles the standard one seen in Westernized countries. This form is often seen in the large towns, such as Calcutta and Bombay. Here the dense sclerosis of the duodenum is inconspicuous, and perforation and hæmorrhage are much more frequent. Thus, in the Medical College Hospital of Calcutta,[12] in 1955, perforations were responsible for about 41 per cent of ulcer operations, and in a large hospital in Bombay for about 33 per cent. These are many times the percentages quoted for Madras and Travancore. This is probably a fairer comparison than one utilizing figures from Westernized nations where, for a given number of peptic ulcers, presumably many more routine operations are carried out than in India. Even so, in these nations the proportion of operations for perforation, averaging 21 per cent in England and Wales,[36] is much higher than in Madras and Travancore.

According to the argument presented here, the above facts can be explained very easily, without the necessity of postulating any fundamental differences between the two forms of ulceration, tentatively proposed by Konstam. For if peptic ulceration be due to the impact of relatively unbuffered acid on the membranes of the stomach and duodenum, it is clear that a difference would be expected between the ulceration produced by slightly unbuffered acid acting for long periods and that produced by highly unbuffered acid acting for short ones. The poor peasants in the south-eastern part of India are largely consuming polished rice, and those in the south-western part tapioca, and both groups very little sugar. In these the protein has been reduced, but is not absent, and the food varies little from day to day. On the present argument this would be exactly expected to produce chronic mild trauma of the membranes, and therefore a chronic sclerosing type of ulceration, with little risk of perforation or hæmorrhage.

Per contra, in the large towns of India there is a considerable consumption of Westernized foods, notably sugar. There is more protein consumed in many of the meals than in those of the peasants of the south, but on the other hand there are some meals where there is practically no protein consumed at all. For example, a young couple going to a cinema in one of the large cities of India will be exposed to just the same danger from eating unsupported sweetmeats as are their counterparts already described in the United Kingdom. In addition, the protein will often be unevenly

distributed in many of the meals eaten, as likewise previously described. Both circumstances would be expected to result in a greater number of acute ulcers, and consequently in a greater number of perforations and hæmorrhages.

To sum up, the present argument indicates unequivocally that the acuteness in form of the peptic ulceration occurring in a given community depends on the ratio of the consumption of foods from which all the protein has been stripped to that of foods from which only a part of the protein has been stripped—in a nutshell, the acuteness depends on the ratio of consumption of refined sugar, and to a certain extent, alcohol, to that of refined grain (and manioc).

The explanation for the predominance of the duodenal site in peptic ulceration will be discussed later with the similar predominance in Western nations.

A Note on the History of Peptic Ulcer in India.—The author is convinced that a prolonged search amongst local hospital records would show that the present ulcer problem in southern India began in Travancore during the last century, when the manioc tuber was first introduced there from South America, and in Madras during the present century, when in 1900 the first rice mills in India went up in this area. Unfortunately, it is exceedingly difficult to prove this, especially when working from the United Kingdom, partly because old hospital records are either deficient or difficult to get at; partly because post-mortems are, and always have been, infrequently performed in India on account of religious objections; and partly because surgeons were not ulcer-conscious even in our own country till the present century was well established, which affected the ulcer diagnosis-rate, and this applied also to India. In spite of all these factors the following points seem significant:—

1. In the years 1877 and 1878 a serious famine occurred in Madras. During this period A. Porter, Brigade Surgeon of H.M. Indian Army and Professor of Medicine at the Madras Medical College, performed autopsies on 226 men and 155 women.[37] These 381 autopsies were carried out with great care, especially of the gastro-intestinal tract, since three-quarters of all the cases died with some form of diarrhœa. The stomach and the intestines were opened up and the detailed descriptions were frequently accompanied by coloured illustrations. *Observing the special tendency of peptic ulceration in India to be accompanied by the*

formation of dense masses of fibrous tissue, it would appear unlikely that a new or old ulcer of any consequence would have been missed. Yet in all these 381 autopsies only one peptic ulcer was seen—a chronic gastric ulcer in an old woman. This compares with the present situation in Madras, where at the General Hospital over the last seven years 476 post-mortems have revealed 11 peptic ulcers (including scars), i.e., nearly nine times as many.[38]

2. Of 258 cases of 'dyspepsia' treated at the Government General Hospital, Madras, in 1895, described by Molesworth, the majority came from the west coast, only 54 coming from Madras itself.[39] This could mean that the manioc cases of peptic ulcer from Travancore were already appearing, but that the milled rice cases from Madras had not yet started up. In view of the diagnosis of 'dyspepsia', nothing more can be claimed here. Furthermore, other possible factors, such as the consumption of the sugary preparation known as jaggery, may have been operating in those days, just as it is now, nor must it be forgotten that many cases of peptic ulcer treated in Madras still come from Travancore to-day.

3. The author is deeply indebted to I. Orr for much valuable information on the subject of peptic ulceration in India, and in particular for permission to quote the following:—

> My late father-in-law's name was William Bentall, F.R.C.S.Ed. and he worked from 1900–1906 in the South Travancore Medical Mission, Neyoor. He visited me there some 25 years later and was certainly impressed with the vast number of ulcer cases we were dealing with. There is one factor one has to consider, however. . . . It was the work of Moynihan, who popularized the gastro-enterostomy operation, which started making surgeons ulcer-conscious. There might well have been large numbers of people suffering from duodenal ulcer in Travancore, which my father-in-law and his colleagues treated medically, and they had, of course, no means of X-Ray diagnosis in those days, nor did cases come to post-mortem, so that large numbers might have been missed. Nevertheless, taking all that into consideration, my father-in-law was quite convinced that the ulcer problem had become a very much more marked feature of medical life in south India since he had worked there.

T. H. Somervell[34] also refers to the rise in the incidence of the disease in Neyoor after the introduction of manioc.

To sum up, the lines of incidence of peptic ulceration in India, not only to-day but probably even in the past, follow closely the lines of protein-stripping in carbohydrate foods, which is in harmony with the present argument.

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CHAPTER VII

Racial Incidence of Peptic Ulcer—*continued*

INDONESIA, MALAYA, AND CHINA

FURTHER support for the argument presented in these pages comes from Indonesia to the south and Malaya to the north.

In 1930 W. Kouwenaar[1] published a series of post-mortem figures that has been considered a model of its kind,[2] and that has defied explanation right down to the present day. Briefly, he sought either to verify, or not, at autopsy a very notable difference in the clinical incidence of peptic ulcer between the Chinese and Javanese in Sumatra (now in Indonesia), which incidence was shown to be some twenty times greater in the former race.[3] Taking care in his paper to say how unreliable purely clinical impressions might be, Kouwenaar not only gave the post-mortem figures for ulcers and scars in some 3000 subjects, but also contrasted the incidence at various age decennia. Briefly, his figures showed 151 ulcers and scars in 1370 Chinese men, but only 8 in 1300 Javanese men, and also showed that the incidence of ulcer rose with the age in both races. He therefore verified by autopsy the difference that had already been established clinically.

Once Kouwenaar's figures were known, efforts were made to establish some difference in the anatomy or physiology of the stomachs in the two races. One team each started working on these two subjects from 1935 onwards, but found no difference in either.[4] Two other teams worked on the food habits, but were handicapped from the start by the multiformity of the menus in both races, and their work was eventually stopped by the war, having established nothing. Even very recently R. Roekmono, from the Department of Pathology at the University of Indonesia, in a paper on the incidence of gastric cancer in the two races (which also shows a big difference) presented at the Symposium of Geographical Pathology held in Tokyo in 1960, stated that the above difference between the Chinese and Javanese, first established by Kouwenaar, remains unsolved even to-day.

As the Chinese in Sumatra and Malaya were in those days young immigrants from China (indentured labourers), it seemed

clear to the present writer that they would be much more likely to be using a ready-to-hand, bazaar type of rice (which is the staple diet in the two races) than the local Javanese were doing, living in their own homes. A prolonged investigation has revealed, in fact, that this is exactly what they were doing.

The writer is indebted to E. B. Merry, Senior Nutrition Officer at the Institute for Medical Research, Kuala Lumpur, Malaya, for the following (1960):—

1. Up to 20 years ago you may assume that the Malays were, in general, home-pounding their rice. The small Japanese rice-mills were introduced during the war and in the years immediately after the war.
2. The Chinese have almost always taken highly milled rice—purchased ready-milled. I have never, personally, heard of Chinese taking home-pounded rice.

From the above it is reasonable to suppose that at the time the same difference existed to the south, in Sumatra, and indeed Professor T. Sutomo, of the Institute of Pathology at the University of Indonesia, in a personal communication (1960) states that although no survey of the differences in the manner of rice-consumption in Indonesia has ever been carried out, it has always been held in that country that the Chinese prefer polished rice from the mills, whereas the Indonesians themselves, especially the common people, usually consume home-pounded rice. As already explained, the fall in the protein content of the milled rice may not be compensated (as regards the uniform buffering of the gastric acid) by the consumption of increased protein in the form of lumps of meat or fish, eaten intermittently at certain meals or parts of meals, which the Chinese have often been better able to afford to do.

It may well be asked whether the Malays in Malaya did not also have very much less peptic ulcer than the Chinese did at that time. Fortunately there exists a reference on this very point. In 1937 L. E. Vine[5] stated: "It is a striking fact that it is not the Tamil who delights in chillies, nor yet the Malay fond of his curry . . . who suffers chiefly from the disease. It is the Chinese coolie. . . ." There is also a description of the ulcers, highly reminiscent of those characteristic of India and Nigeria, previously described, with gross fibrosis, leading to pyloric obstruction, but here complicated not infrequently by late perforation and hæmorrhage (which were the commonest reasons for the patients coming to hospital).

Mr. Vine, who practised as a surgeon in the Malayan Medical Service from 1930 to 1950, has kindly supplemented the above in personal communications (1960) to the present writer, alluding to the "quite extraordinary frequency of peptic ulcer in male Chinese coolies, in the thirties", and to the fact that the perforations and hæmorrhages were markedly different from the Westernized types, being in ulcers surrounded by dense masses of fibrous tissue, often causing pyloric obstruction.

The author is indebted to H. McGladding and J. A. P. Cameron for similar communications (1960). It may therefore be taken as certain that exactly the same racial difference in the incidence was present in Malaya, to the north, as Kouwenaar at that time was describing in Sumatra to the south.

Incidentally, as regards the Tamils in Malaya, mentioned above, Mr. Vine has the following to say: "They came from the Madras Presidency and North Ceylon. Their rice was undermilled, but being Hindus their protein intake was very low. They ate all their food very highly spiced indeed. I do not, off-hand, remember seeing a single case of perforated peptic ulcer amongst them."

It is not possible to leave this subject without referring to the incidence of beri-beri in the Chinese, on the one hand, and the natives of Malaya and Sumatra on the other, at the time of Kouwenaar's figures and earlier, as this forms a valuable cross-check on the different types of rice being consumed by these races. It may be stated that this check is not often practicable in the case of India, because of the frequent parboiling of the rice in that country before it is milled, which procedure tends to disperse the vitamins throughout the grain and so prevent the disease. But in Malaya and Sumatra the local inhabitants and the Chinese have never taken to parboiled rice and consequently the beri-beri check becomes available. As regards Sumatra, unfortunately, no clear-cut indications are now available, but as regards Malaya the evidence is overwhelming:[6] "Beri-beri in the early days of the development of Malaya was essentially a disease of the Chinese labourer. It was most severe during the period of the influx of the great numbers of Chinese adults, who came to work in the vast schemes of development that were under way. The disease was most often seen in the young male adult doing very strenuous physical labour and consuming large quantities of highly-milled rice."

It is interesting to note that beri-beri and peptic ulcer, though frequently associated in the same population, as would be expected

from the argument presented here, seldom seem to occur in the same patient. The simplest explanation for this would appear to lie in the vagus nerves, which control the secretion of the gastric juice, sharing in the depression in function of the peripheral nerves that is characteristic of this deficiency, so that low levels of gastric acidity result.

In concluding this subject, therefore, it is submitted that an explanation is now available for the perplexing difference in the incidence of peptic ulcer between the Chinese and Indonesian natives which was established by Kouwenaar thirty years ago, and that this explanation is in close accord with data from southern India and northern Nigeria, and, as will be seen later, from differently located Japanese prisoner-of-war camps in the last war.

An essential completing piece of information concerns the incidence of peptic ulcer in the Chinese in China, especially where they are living on home-pounded rice, but before dealing with this subject it may be of interest to quote de Langen and Lichtenstein's[7] interesting figures for gastric and duodenal ulcer in the Royal Netherlands Army and Navy in Indonesia, where during the period 1907 to 1929 about one death per year occurred from this cause in 40,000 native troops and about the same in 40,000 native sailors. In close keeping with these figures the sick-list reports of the Army showed an incidence of gastric and duodenal ulcer of 0·09 per 1000 in the natives (and 2·23 per 1000 in the Europeans, i.e., an incidence twenty-five times greater). I am indebted to Admiral T. A. de Haan, the present Director of Medical Services in the Royal Netherlands Navy (1960), for the information that in the native diet the white flour used in bread, etc., by the Europeans was nearly all replaced by rice, of which much was deliberately undermilled to avoid the risk of beri-beri.

And what about the situation to-day ? In Malaya nearly all the local inhabitants now eat milled rice, and for that matter, white sugar; and well-known bottled fruit drinks are on sale all over the country.[8] The distinctions noted above between the indigenous inhabitants and the Chinese are vanishing, aided by intermarriage. Fortunately, this change in no way effaces the evidence from the past presented here, nor the inference that may be drawn from it. In Indonesia, however, in spite of milled rice constantly displacing the home-pounded product since the war, just as in India and Malaya, the difference in rice-consumption between the Indonesians and Chinese persists to some extent, and in keeping with

this fact peptic ulcer (and gastric ulcer), as recently elaborated in the Tokyo Symposium, still occurs more frequently in the Chinese. For this information the writer is again indebted to the kindness of Professor Sutomo, whose deep insight into pathology and perfect knowledge of the English language have been invaluable in the preparation of this chapter.

Incidence of Peptic Ulcer amongst the Chinese in China. —The incidence of peptic ulcer amongst the Chinese in China cannot be established statistically. There are no figures available on this subject from the world's literature, nor has attempted contact with the medical authorities in Peking for any recent information been successful. Nevertheless, it is perfectly feasible to obtain a good general picture of the situation, owing to the presence in the United Kingdom of some who have practised in China for many years, and who are well aware of the commonness or rarity of peptic ulcer amongst the inhabitants of that country, as long as these terms are not expressed in actual figures.

It is first essential, from the point of view of the argument advanced in this work, to distinguish sharply between the Chinese living in the big towns, consuming highly milled rice and quite possibly having access to Westernized foods as well, and those living in rural China, still consuming rice pounded in the home in the manner of their ancestors.

As regards the first group, it is quite clear that peptic ulcer is common amongst them. The writer has himself seen some evidence of this fact during three years' work in Hong Kong, and those of far greater experience, such as Professor A. J. S. McFadzean of the University of Hong Kong, are in no doubt about the commonness of peptic ulcer (usually duodenal ulcer) in the Chinese in that city and the adjacent territories. What is now important to the present argument is to see if there is a marked difference in this respect in the Chinese living on unmilled rice in rural China. And it would seem unquestionably that there is.

The writer is indebted to J. H. Lechler, who worked in Szechwan province, 2000 miles west of Shanghai, from 1908 to 1938, for the information that gastric and duodenal ulcer were both very rare in these Chinese. The rice consumed was pounded by a primitive mechanism, activated by the feet. Dr. Lechler states that gastro-enterostomies were performed commonly enough in Chengtu, the capital, in the period before the last world war, and one of his assistants who joined him from there was very keen to

do this operation on the rural Chinese, "but there were no peptic ulcers on which he could operate".

The author is also indebted to E. N. Callum, who practised as a surgeon in a rural part of Fu-Kien province, South China, from 1930 to 1935. Mr. Callum, writing from the Epsom District Hospital in this country (1961), states that peptic ulcer was "excessively rare amongst these Chinese", who likewise consumed home-pounded rice.

Finally, the author is particularly grateful to S. D. Sturton, at present radiologist to the Hong Kong Sanatorium and Hospital, for the following from many valuable communications on China (and also on the Japanese prisoner-of-war camps to be discussed later): "I am of the same opinion as Haddow. In over 39 years of radiology (and some medicine) in Hong Kong and China, of which 30 were in Hangchow (1921–52), I have seldom diagnosed peptic ulcer in the class of Chinese who eat the less polished rice." P. Haddow, to whom the author is also indebted, indirectly, had 28 years' experience in Hangchow (1923–51), and is at present clinical pathologist at the above hospital.

In conclusion, therefore, the experience of these four observers is in striking contrast with that of the three observers in Malaya, previously quoted, where the incidence of peptic ulcer in the Chinese coolies, at that time eating highly milled rice, showed "a quite extraordinary frequency". It will be particularly noted that all these experiences are centred on the same period of time, before the last world war, i.e., the 1930's.

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CHAPTER VIII

Racial Incidence of Peptic Ulcer—*continued*

JAPAN

Of all countries, few would appear, on the argument advanced in these pages, so suitable for the development of peptic ulcer in the inhabitants as is Japan. In the first place the diet is predominantly one of rice, and the rice is highly milled (the extraction rate being about 70 per cent, as in the case of white flour in Westernized nations). Although some years ago there was a move to popularize under-milled rice, this was unsuccessful.[1] In the second place the relatively low standard of living in most of the Japanese precludes the inclusion of any large amounts of animal protein in the diet, so that the protection afforded by this, if eaten concurrently with the rice, is largely missing. Finally, there is the third fact that the amount of sugar consumed, including that in sweets, is considerable, as the high degree of industrialization in the country would lead one to expect. The amount of this substance consumed per head per day in Japan is about $1\frac{1}{2}$ oz.[2] (as against, for example, an average of about $\frac{1}{2}$ oz. per head per day in ten Indian provinces[3]).

These three facts should, according to the present argument, seriously interfere with the buffering of the gastric acid, and result in a high incidence of peptic ulceration in the inhabitants. It is, therefore, most interesting that the incidence of peptic ulceration in the inhabitants of Japan is the highest in the world. The very magnitude of the problem has led to a large amount of investigation by the Japanese, so that an abundance of statistics is available.[4]

It is not intended to pursue this subject here, except for setting down three points of importance to this work:—

1. The actual age-adjusted death-rate from peptic ulcer for males in Japan (1954) is 34·7 per 100,000, as against, for example, 14·1 per 100,000 in England and Wales.

2. Most of the peptic ulcers to which this figure applies are gastric ulcers. However, many of these ulcers are near the pylorus, and it will appear from what is shown later that, as regards the

present argument, the significance of a juxtapyloric ulcer is not appreciably different from that of a duodenal one. Also, as in England and Wales, the proportion of duodenal ulcers in the total is much higher in the living, the figure in Japan reaching 40 per cent.

3. The chronicity and tendency to sclerosis in the ulcers are not so marked in Japan as in some rice-eating countries, such as India, which is comprehensible on the present argument, in view of the approximation of the diet in some particulars to that in the Westernized nations, especially as regards the higher consumption of sugar, including that in sweets. Nevertheless, that these ulcers do not lack this characteristic is shown by the rarity of perforation (in '45 out of 3151 cases, in one series, only 8 of these perforations being in acute ulcers), and the commonness of pyloric stenosis (in 414 of the above 3151 cases).[4]

As regards the cross-check on the degree of milling in the rice eaten, afforded by the incidence of beri-beri, it may be stated that this disease is quite common in Japan. In spite of the ready availability of vitamin B_1 preparations in that country, a survey concluded by the Ministry of Welfare in 1959 indicated that 10·5 per cent of the population showed loss of knee-jerks and 6·2 per cent had pains in the calves, though it was not of course claimed that all these persons were suffering from the active disease.[5] The actual death-rate from beri-beri in 1958 was 0·6 per 100,000.

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CHAPTER IX

Evidence from Prisoner-of-War Camps in the Far East, 1942–45

INTERESTING evidence on the aetiology of peptic ulcer is provided by data from the Japanese prisoner-of-war camps, which, as far as the writer knows, has not been used in this connexion before.

In these camps the commonest element in the diet was rice, and usually highly milled rice at that. When little other food was provided the diet approximated, therefore, to that already described for certain parts of India and Indonesia and, if the present argument is correct, peptic ulcer should have become conspicuous. *Per contra*, if the rice or other grain was not highly milled—and sometimes it was not—then the diet approximated to one of unrefined cereal, with very little animal protein, as already described for certain parts of Africa and Asia, and peptic ulcer should have become rare, no matter how reduced in amount the protein in the rest of the diet or how unfortunate the general conditions. The facts are as follows.

1. Singapore and Thailand (Siam).—In the large prisoner-of-war camp at Changi, Singapore, the diet has been recorded in detail by R. C. Burgess[1] and D. A. Smith and M. F. A. Woodruff,[2] to the first of whom the author is grateful, not only for the helpful advice to follow up this clue, but also for information in connexion with it. He is indebted, too, to J. A. P. Cameron and E. K. Cruikshank, who likewise were inmates of this camp. From the point of view of the present work, two facts are important:—

a. The total protein of the diet was not grossly deficient, averaging about 60 g. per head, until March, 1945, when it fell to 50 g. and at times even to 40 g.

b. The rice which provided the bulk of the calories was nearly always highly milled, but, except for a period of some months at the commencement (1942), it was supplemented by an issue of rice polishings until the end of 1943, when this issue ceased.

The period of importance to the present work is the year 1944, when the issue of rice polishings ceased, though the total protein

remained unchanged for another fifteen months. (The reason why the total protein is mentioned is that, although the argument in this work attaches much more importance to the natural distribution of protein in a meal than to its total amount, it is clear that any protein consumed must be of value in the buffering of the gastric acid and should therefore be taken into account.)

As a cross-check on the issue of the rice polishings, *Fig.* 2 shows the incidence of beri-beri in the camp throughout the whole period. The rise in 1944 after the issue had ceased is well seen.

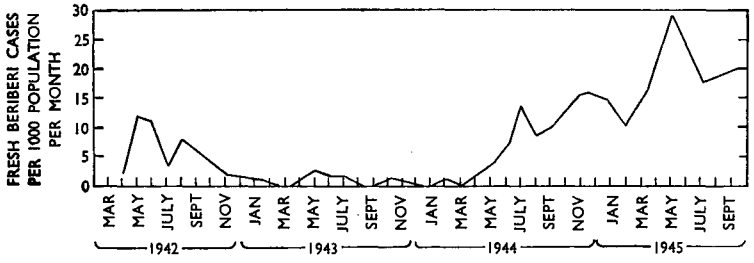

Fig. 2.—Incidence of beri-beri in the Changi prisoner-of-war camp. (*After Burgess.*)

In 1944, therefore, according to the present argument, the stage was set in the camp for a rise in the incidence of peptic ulceration, and the following quotation from the late Julian Taylor's contribution to the *History of the Second World War*[3] is consequently of great interest. After mentioning the deterioration in the general conditions in the camp in 1944, Professor Taylor writes: "Serious duodenal ulcers became a plague. The victims had perpetual and severe pain, leading them to beg for some sort of relief. They became emaciated and some suffered perforation and hæmorrhage. The ulcers were enormous. . . ."

As regards the period in 1942 when rice polishings were temporarily withdrawn, some cases of peptic ulcer occurred at this time amongst the Australians,[4] but they were relatively few compared with the later ones, which is in harmony with the much smaller incidence of beri-beri at this period.

Meanwhile, it is a remarkable fact that among the prisoners of war taken away from Singapore in 1943 to work in Thailand on the tragic Burma Railway project, where the captives lost over 40 per cent of their numbers before returning to Singapore in 1944, peptic ulcer was almost never seen. The writer is indebted to

Professor Taylor for the following information (which was given objectively and not in support of any dietetic cause whatsoever):—

> Never once did I come across a prisoner (including those who had had duodenal ulcer before going to Thailand) who complained of his duodenal ulcer while being really knocked about. . . . Then in 1944 when they all came back to Singapore . . . after a very few weeks they began to complain of symptoms like those of duodenal ulcer, including many who had the symptoms before going to Thailand, but who while away had had no ulcer symptoms at all. Now the duodenal ulcers became serious and we operated on a number, but not on one who did not beg for operation . . . usually sitting up in their 'beds' with the hand on the epigastrium, in pain all night and all day.

Some 25 gastrectomies were carried out under extremely difficult conditions, with only 2 deaths. The freedom from peptic ulcer in the prisoners of war when they were working on the railway project is supported in personal communications (1961) from other observers, including H. de Wardener, E. E. Dunlop, and A. L. Dunlop. Thus, in the tables describing the diseases occurring in the Chungkai 'hospital' in Burma, during the construction of this railway, peptic ulcer is not mentioned.[5] The tables given in this latter reference, by E. E. Dunlop, were prepared by A. L. Dunlop, to whom the author is indebted for the following:—

> It was into the general category 'all other diseases', I am afraid, that such infrequently occurring conditions as peptic ulcer were placed. As a physician I confirm the observations made by the surgeons to whom you refer. Patients suffering from peptic ulcer were very rarely seen.

If the argument presented in this book is correct, the disappearance of peptic ulceration from the clinical scene, especially observing the pitiable amounts of protein issued, suggests that little or no milling had been carried out on the rice consumed. On this subject the writer is indebted for the following to H. E. de Wardener. Writing from Charing Cross Hospital (1961), Professor de Wardener states:—

> The rice that we were supplied with on the railway was certainly not white. We always thought that it was the sweepings of the rice barns. It was broken and there was quite a lot of brown flecking on the grain. I may say that these appearances were those before it was cooked. By the time it was cooked this brown colour no longer seemed to be evident.

It will be noted that the colour after cooking might well have led to subsequent confusion on this point.

Mr. E. E. Dunlop was kind enough to write for information on this subject to L. E. C. Letts, Esq., who is engaged in commerce in Singapore and has a deep knowledge of rice, to whom the author is indirectly indebted for the following (1961):—

> All the rice I ate in Siam during the prisoner-of-war days was highly milled. The quality was not high. By this I mean a substantial amount of the grains were broken. . . . However, broken or whole grains, it was still highly milled. You will, however, recall that our rice ration was invariably supplemented by bran (commonly termed 'iron filings'). Because Siam has so much rice and so many rice mills within its boundaries, there is always a copious supply of bran available, which normally is only used for feeding pigs.

It is clear, therefore, that though some prisoners ate lightly milled rice, others ate highly milled rice but with polishings added. In either case the freedom from peptic ulceration on the present argument is explained.

No doubt still other batches of these prisoners ate highly milled rice unredeemed by the addition of polishings, but they could not have been very numerous, to judge by the cross-check afforded by the incidence of beri-beri, which was much less than in Singapore.[6] Also, the cases of beri-beri that did occur may have been relapses of previous attacks, induced by the dysentery which was so prevalent and which always carries such a threat.

At this point it must be stated that the addition of rice polishings to highly milled rice does not necessarily convert it into the equivalent of lightly milled or home-pounded rice. For one thing, the amount of polishings added may not equal the amount previously removed. This was certainly the case at the Changi camp and may well have been so on the Burma–Siam railway project. For another thing, the amount of protein lost in highly milled rice during washing and cooking will not be affected by the subsequent addition of polishings, and it has already been shown that this loss is important, though at least in the case of the Changi camp the protein so lost was deliberately recaptured. It could, therefore, be contended that the addition of polishings should not be given as much importance as is being done here. It must be remembered, however, that the fine distribution of a protein-rich material through the rice, influencing the buffering of the gastric acid, is not the only consequence of adding these polishings—the effect of the fibre in increasing mastication and prolonging the gastric emptying-time must also be reckoned with.

These two factors have already been discussed (Chapter IV, pp. 19, 20), and will be discussed again, but it can be advanced at this stage that the increased mastication, leading to greater impregnation of the food with alkaline saliva, may well be equalled in importance by the slower gastric emptying-time, leading to greater protection of the first part of the duodenum, through diffusion backwards of the alkaline contents of the second part. Anyone who has any doubts on the reality of these two factors should take into his mouth one teaspoonful of unprocessed bran, obtainable at any corn chandler. He will discover that the taste of this material is by no means unpleasant, which is to be expected since it is a natural food for mankind, but that the amount of chewing and salivation required to effect swallowing is remarkable. If further quantities are ingested, the consumption of such concentrated fibre may so affect the gastric emptying-time that some acid regurgitation into the œsophagus may easily become noticeable.

2. Hong Kong and Japan.—The scene now shifts to two other groups of prisoner-of-war camps—those in Hong Kong and those in Japan itself.

a. In Hong Kong the conditions in the prisoner-of-war camps have been described by H. L. Cleave[7] and A. J. N. Warrack.[8] It is to be noted that the rice eaten was highly milled. Beri-beri and peptic ulceration were both prominent in these camps, though the former was partially controlled by the giving of vitamin B_1 preparations, and the latter never assumed the dramatic proportions seen in Singapore in 1944.

The writer is indebted to A. J. N. Warrack and A. W. Dawson-Grove for further evidence on these points. Writing from the Group Pathology Laboratory, Sheffield, the former states (1961): "In Hong Kong prison camps the diet consisted mainly of highly polished rice. Beri-beri was common and I saw many cases of peripheral neuritis. . . . With regard to peptic ulcer, I certainly remember 'ulcer pains' being a tremendous problem." A. W. Dawson-Grove, writing from Hong Kong (1961), supports this statement.

In spite of peptic ulceration in rice-eaters being, as already pointed out, usually chronic in type, with much fibrosis and little tendency to perforate, perforations were still prominent in these prisoners. In one camp of 400–500 men there were 3 cases in one year.[9] This rate of perforation comes out at nearly 30 times

the rate recorded by Illingworth, Scott, and Jamieson[10] for the Glasgow area of Scotland just before the war, and although the latter referred to a total population and not just to adult men, the incidence of perforations in the Hong Kong camp was still by comparison very high, which is in keeping with the prevalence of the ulcer symptoms there, just described.

b. After two years a large proportion of the prisoners in Hong Kong were transferred to Japan itself, and it is here that developments of interest to the present work mainly took place.

In Japan an important change occurred in the diet of the prisoners. The food continued to be mainly cereal in nature, with lamentably small amounts of animal protein, so that famine œdema, which is always aggravated by lack of protein,[11] was present up to the level of the knees in most of the men for long periods, but the actual grain consumed was different. Instead of consisting predominantly of highly milled rice, it consisted of rice, lightly milled (i.e., with portions of the bran (pericarp) visible on the grain), plus barley and millet, both largely unmilled. This food was known amongst the prisoners as the 'Korean mixture'. It was imported, and some of the prisoners helped to unload it at the docks. It will be shown that these changes in the cereal food were attended by a dramatic disappearance of peptic ulceration from the clinical scene.

At this point it is important to note that during the period now under review (1944–5) Tokyo was almost entirely destroyed by air raids, the destruction far exceeding anything seen in London. The streets were flanked by mile after mile of burnt rubble, only a few concrete buildings being left standing. In these circumstances the escape of the prisoner-of-war camps was remarkable and was largely due to their being situated near the periphery of the capital. The importance of the air raids to this work lies in the fact that the great air raids on London and other cities in the United Kingdom were accompanied by a dramatic rise in the incidence of peptic ulcer perforations in the populations concerned.[10]

It will be advanced in a later chapter that under no circumstances would these periods of terror have resulted in peptic ulceration had they not led to snack meals only too liable to consist of just those refined carbohydrates incriminated in this work. However, not to anticipate this later chapter, but to revert to the situation in Tokyo, where the air-raid danger was much

greater, but where the prisoners were eating grain from which little or no protein had been stripped, the situation as regards the incidence of peptic ulcer was as follows.

i. *Tokyo.*—The Shinagawa Camp hospital in Tokyo, with an average of 180 patients, catered for all the prisoners of war in the Tokyo and Yokohama area, which H. M. Holland, Consul at the American Embassy, Tokyo (1961), has kindly informed the author numbered 6050 at the end of the war. In over a year's work in charge of the surgical side of this hospital H. L. Cleave never saw or heard of a patient with peptic ulcer, either as a case of perforation or as one needing surgery for pain.[9] I am grateful for the following to A. W. Dawson-Grove (1961), who worked on the medical side of this hospital for the same period: "In Japan peptic ulcer symptoms were practically non-existent. . . . The rice in Japan was definitely not highly milled. The barley and millet were extremely coarse and in many cases still had husks adherent to them. Beri-beri was completely absent, owing to the unmilled grain." And to J. N. Warrack (1961) for: "Yes, you may certainly quote me as saying that the peptic ulcer pains which were so big a problem in Hong Kong ceased in Japan." The author has verified by personal communication that a remark by Gottlieb[12] on the existence of peptic ulcer in this camp referred only to the time when it was being set up, and not to the long period covered by the above references, when the food was as stated.

It may be added that owing to the quantity and quality of the vegetables being much inferior to those in Hong Kong, the conditions considered in Singapore to be due to riboflavin deficiency also occurred in Japan, though for practical purposes new cases of beri-beri did not.

ii. *Kobe and Osaka.*—These camps were also subjected to intense air raids, some of them being partially destroyed. I am indebted to J. A. Page for the following:—

> Our diet in Kobe was mainly red rice as opposed to white polished rice. . . . That peptic ulcer was virtually unknown amongst the prisoners in Japan is not just an impression—it is a fact. I think my parish in Kobe and Osaka covered 7 or 8000 prisoners in something like 4 camps and must be fairly representative. I still have a list of 184 operations carried out between June '44 and June '45. These included one perforation in an old recurrent ulcer; otherwise there is no mention of peptic ulcer medically or surgically. But I also have a record of 75 operations I did in Kowloon (Hong Kong), and 3 of these were for perforated duodenal ulcer.

(These are not the same as those quoted previously, operated on by H. L. Cleave.) This communication also refers to the high incidence of famine œdema in the prisoners, but confirms that beri-beri itself 'faded out' after the prisoners reached Japan.

The writer is grateful to D. Longbottom for a similar communication confirming the complete absence of peptic ulceration in two camps in Kobe of 500 men each during the year Aug. 31, 1944, to Sept. 5, 1945.

iii. *Japan itself.*—Some reference may be made at this juncture to the incidence of peptic ulcer amongst the Japanese themselves during the war. Attention has been drawn in the previous chapter to the high incidence of this disease in Japan, and it may now be stated that this incidence greatly increased during the war, as the following will show:—[13]

> In males the age-adjusted death-rate for ulcer of the stomach was 23·94 per 100,000 population in 1937, and the figures showed little change down to 1941. In 1942, however, the rate rose to 28·72, in 1943 to 32·94, and in 1947, soon after the war, rose to the abnormal height of 47·59, showing an increase of 100 per cent over that of 1937. In other words, the death-rate among males for gastric ulcer doubled in these ten years. After 1948 the rate began to fall. The rate for the year 1955 was 20·09, indicating a somewhat lower figure than that for 1937.

This increase is readily explained on the argument presented here. The severe fall in the consumption of protein foods resulted in the Japanese living on a predominantly rice diet, much resembling, in fact, that in some of their own prisoner-of-war camps, except that there was more of it. This rice during the war was normally *highly milled* (70 per cent extraction rate).[14] During the war, also, much rice was imported from Burma and Siam, and this rice is always highly milled. Only at the end of the war was some unmilled rice consumed, bought on the black market.

In conformity with this information the death-rate recorded from beri-beri during the war averaged 10·2 per 100,000,[14] against 0·6 per 100,000 in 1958, but, as already explained, this cross-check in the case of Japan is less valuable than usual, owing to the high availability of vitamin preparations in that country.

(It must be repeated that the references to beri-beri in this work have been inserted solely to serve as a cross-check on statements about the type of grain being consumed. There is no suggestion that beri-beri plays any part whatever in the causation of peptic ulcer. In fact, to judge by the rarity of the association of

the two diseases in the same patient, the presence of beri-beri appears to offer some protection against the development of peptic ulcer, a reason for which has already been advanced. Furthermore, there is no question of any appreciable lack of vitamin B_1 in the food of the Westernized countries to-day, where the incidence of peptic ulceration is so high.)

3. Shanghai and Peking.—A note may be added on the Japanese prison camps in Shanghai and Peking. Here S. D. Sturton[15] does not recollect seeing a single case of peptic ulceration amongst some 400 prisoners interned for approximately three years, 1942–5, though the diet contained exceedingly little protein. The rice consumed was usually highly milled, but the prisoners were allowed to buy cracked wheat in bulk, which was consumed each day regularly. This cracked wheat was a crude wholemeal flour, the grains merely being crushed flat. The only cases of beri-beri occurred in those who did not eat this crushed wheat, who were very few.

4. Dutch East Indies.—The Japanese prison camps in this area were investigated by a Dutch medical team in 1945–6, sent out by the Netherlands Red Cross, a full report of whose findings is published.[16]

In this report the rarity of peptic ulceration is noted. "Another class of patients who reacted favourably on being interned was a great number of those suffering from peptic ulcer."

On inquiring further into this matter the author was privileged to receive the following details from S. Postmus (1961), from the Central Institute for Nutrition, Utrecht, Holland:—

> In the beginning of the camps we had many cases of gastric and duodenal ulcer . . . and most of the ulcer cases had been diagnosed, and often treated by our army colleagues. So we could be sure of the diagnosis. . . . More and more their number decreased, so that at the liberation less than 20 per cent remained.

What now concerns the present argument is the type of rice consumed by these prisoners. In the analysis of the vitamins and other constituents of the food taken, the report refers (p. 57) to the red (under-milled) rice issued by the Japanese, but for further particulars on this the author is again indebted to S. Postmus, who states that for the first three months various forms of rice were issued, including highly milled rice, but that thereafter the rice was always under-milled. J. B. Stolte, senior surviving member of the team, supports this:—

I can tell you without any doubt that in virtually all camps in the Dutch East Indies only *un*polished rice was distributed. As far as I know only in one or two camps that I did not visit was polished rice given for a time, it being very scarce during the Japanese occupation.

It is in keeping with this information that although famine œdema commonly occurred in these camps, beri-beri was almost never seen.

Conclusion.—In all the above Japanese prison camps it will be noted that, just as in the different races previously described, so also in these prisoners, the incidence of peptic ulcer bore little or no relationship to the amount of animal protein in the diet, which was low enough in every camp to facilitate the frequent occurrence in the prisoners of famine œdema to the level of the knees, but the incidence did bear a striking relationship to the degree of refinement in the cereal or other carbohydrate food being consumed. If the natural amount of protein in the cereal was undisturbed by refinement, no ulceration occurred, no matter how bad the conditions or how great the anxiety; but if the amount was interfered with in this way, so that the gastric acid was no longer buffered in the natural, uniform manner, then the disease began to appear very quickly. It will be particularly noted that the incidence bore no relationship whatever to the presence or absence of mental stress, or to the degree of starvation. This last is as would be expected, since studies[17, 18] in human starvation have shown that although the motility of the stomach is reduced in these circumstances, the secretion of hydrochloric acid is not. Furthermore, it is common knowledge that hunger strikes and other forms of fasting do not end up in peptic ulceration. In fact, it will be shown later that fasting has often formed the basis of the medical treatment of the disease.

It is not to be denied that in the conditions prevailing in the prison camps in the Far East there was a host of variables that could explain differences in the incidence of peptic ulcer. For example, the weather may have been hotter in some camps, colder in others; certain diseases may have been present in these camps, absent in those; here there may have been more of this food and there more of that; and so on. But if many camps are followed, these variables tend to cancel out, and what has been attempted is to show that the thread of the relationship between the incidence of peptic ulcer and the removal of protein from carbohydrate foods runs unbroken through all of them, just as it

has been attempted to show that it remains unbroken in many different races, in many different countries.

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CHAPTER X

Peptic Ulcer in Westernized Countries Explanation of the Difference in the Incidence of Gastric Ulcer and Duodenal Ulcer

THE high incidence of peptic ulcer in Great Britain, as an example of a Westernized country, has already been given (Chapter II), and it will now be convenient to take up differences in the incidence of gastric and duodenal ulcer.

These differences are well known. For example, the great rise in the incidence of peptic ulcer in Great Britain and other Westernized countries over the last century, to which reference has also already been made (Chapter III), has consisted much more of a rise in the incidence of duodenal ulcer than in that of gastric ulcer. Other differences exist, too, such as the different incidence of gastric and duodenal ulcer in the two sexes, and in certain social classes and occupations. Unless the argument presented here enables these and other differences to be explained, it must be reckoned to fail, however much light it may throw on the racial incidence, but if it can enable these differences to be explained, it will have accomplished something that so far has not been achieved. It is contended that the evolutionary approach does, in fact, carry these hurdles, and that with its help the differences can be explained.

The series of explanations begins with the known fact that the gastric hydrochloric acid is almost wholly secreted in the first portion of the stomach, the so-called fundus and body, where the oxyntic cells are situated. If we now remember the distinction between the level of acidity obtaining at the surface of the gastric membranes and the level of acidity in the mass of food being digested, we notice that, as the acid passes along the stomach and first part of the duodenum, the level of acidity, though it rises in the food being digested,[1] falls at the surface of the membranes. It rises in the food being digested because of the increasing penetration of the acid into it, but it falls at the surface of the membranes because in the fundus there is new acid just emerged from the ducts, at maximal strength, whereas at the pyloric end

and in the first part of the duodenum there is old acid, increasingly buffered by the food. In the second part of the duodenum the alkaline streams issuing from the pancreatic and biliary ducts complete the neutralization altogether.

It is clear, therefore, that each succeeding portion of the stomach and commencing duodenum must by evolution be adapted to the impact of acid of less and less strength. If, now, the buffering of the acid is interfered with by the removal of protein from the food (and it should be particularly noted that this may result in a level of acidity high only for the person concerned, the level being perhaps lower than average for other people), where are the consequences of this deficient buffering going to be most prominent? Not in the fundus and body, because this portion of the stomach is adapted to full-strength, unbuffered acid anyway, but increasingly in the next portions of the stomach and reaching a maximum in the first part of the duodenum. In this connexion it must be pointed out that the acid-secreting part of the stomach stops well short of the incisura, extending in fact little more than half-way along the lesser curvature.[2]

It is, therefore, logical according to the present argument that peptic ulceration in civilized countries to-day should consist mainly of duodenal ulceration, and that the rise in the incidence of peptic ulcer during the last century, which according to the present argument stems from the great increase in consumption of refined carbohydrates, should mainly involve a rise in the incidence of *duodenal* ulcer. It is also logical that over 90 per cent of gastric ulcers should occur in the more terminal, non-secreting, parts of the stomach, which include nearly half the lesser curvature.[2] However, the relatively small rise in the incidence of gastric ulcer during the present century will need further explanation and this will be advanced later.

It is tempting to extend the above reasoning to explain the greater tendency for gastric ulcer to affect the lesser curvature of the stomach. For as the gastric acid emerges from the ducts and begins to penetrate the food mass, it is bound to be influenced by the force of gravity and to collect to some extent along the lower border of the stomach. Therefore, this border of the stomach should by evolution be adapted to a higher level of acidity than is the upper border. Therefore, in the presence of an unnaturally high level of acidity, due to deficient buffering by the food, the

lesser-adapted lesser curvature will suffer the more, and if an ulcer is to occur in the stomach instead of the duodenum, it is, according to the present reasoning, on the lesser curvature that we should expect to find it.

It may be added here that a further reason why the above rise in the incidence of peptic ulcer should chiefly have involved a rise in the incidence of duodenal ulcer may lie in the removal of fibre attendant on the refining of carbohydrate foods. This removal, as previously set out, considerably increases the speed of passage of the gastric contents into the duodenum, with consequent reduction in the backward diffusion into the first part of that structure of the protective alkaline contents of the second part.

Finally, it will be recalled that the present argument affords a ready explanation for the chronicity of duodenal ulceration in the eaters of manioc and polished rice, and the frequency of acute ulcers, including perforations, in the eaters of refined sugar, since in the former case the carbohydrate food is stripped of only part of its protein, whereas in the latter case it is stripped of all of it.

To return to the rise in the consumption of white flour and refined sugar in this country during the last century and a half, there is a difference in this respect between refined (white) flour and refined sugar. White flour was firmly established in this country by the end of the eighteenth century,[3] and though the rise in its consumption unquestionably continued during the nineteenth century, the rise could never have approached that of refined sugar (in which substance, as already stated, the loss of protein is proportionately very much greater). The statistical material is much less complete in the case of white flour than in that of refined sugar, but the latter is summarized in *Fig.* 3.

It will be noticed that the consumption of 85 lb. of sugar per head per year was reached around the year 1895. At this level, to judge by the subsequent high incidence of peptic ulcer, the interference with the buffering of the gastric acid appears to become of decisive importance.

If it were asked why the incidence of peptic ulcer since the turn of the century appears to have increased more than the consumption of sugar, the answer would certainly lie, in the writer's opinion, in the increasing tendency since that date for the sugar to be consumed on its own, as in the taking of sweetmeats and sweet drinks between meals.

This latter factor appears to be almost as important as the high total sugar consumption itself. Many examples come to mind. Some of the most glaring are to be found amongst cinema audiences, amongst lorry drivers, and for that matter amongst the

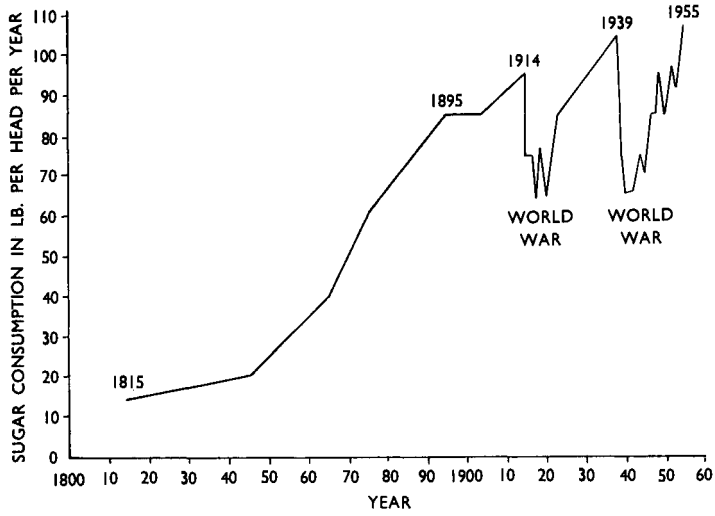

Fig. 3.—The rise in sugar consumption in the United Kingdom over the last century and a half. (*Drawn from information kindly supplied by Fairrie & Co. and the 'Board of Trade Journal'.*)

personnel in the Services who throng the canteens during stand-easy periods, but, in fact, the consumption of sweet things between meals is widespread amongst the whole population. In all cases a big dose of sugar, supported by little or no protein, is very liable to land in the stomach in the middle of the morning or afternoon, and this factor has become much more prominent since the turn of the century. A note in this respect, on ice-cream consumption, in which the increase has been little short of staggering, is given in Appendix C.

In connexion with the foregoing, it will be interesting to see if the increasing substitution of television for cinema-going makes any impression on the incidence of peptic ulcer in the near future. Any slight decline in the incidence that has been noticed recently might well derive from this cause, but with our present food habits will certainly not go very far if the argument in this book counts for anything.

The next group of explanations is centred on further physiological considerations, which involve the normal ranges of gastric acidity at different ages and in the two sexes. These ranges have been studied by Vanzart and others[4] in an analysis of 3746 records at the Mayo Clinic, and a summary of their findings may be appreciated from a study of the curves in *Fig.* 4, reproduced in a modified form from their paper.

It will be noted that the curve of gastric acidity, starting at a very low level in infancy, climbs steadily all through childhood,

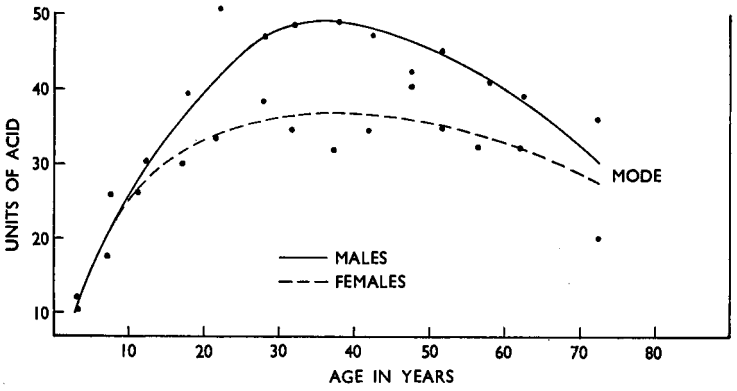

Fig. 4.—Calculated modes of free hydrochloric acid in the stomachs of males and females at different ages. (*Modified from 'Archives of Internal Medicine'.*)

and equally in the two sexes; that in the adult it reaches a considerably higher level in the male than in the female; and that subsequently there is a fall in old age, especially in the male, so that the levels in the two sexes again approximate to each other. The results of Pollard's[5] work are in close agreement, and that of others since.

In conjunction with these physiological facts it is possible on the present argument to explain further differences in the incidence of peptic ulcer. This argument, it will be borne in mind, absolutely excludes the gastric acid as being a primary cause of peptic ulcer, but singles it out as the agent through which the primary cause operates. It thus becomes clear that if the primary cause be present, i.e., interference with the protein in carbohydrate foods and hence with the buffering of the gastric acid, the cause can scarcely become operative at all in infancy, since there is almost no acid to be buffered; that the cause can seldom

become operative in childhood, since the curve of acidity climbs but slowly; that the cause in the adult will become operative more easily in men than in women, since the curve of acidity rises considerably higher in them; and that the cause in old age will again become operative less frequently, since the curve of acidity is now falling again. There is thus explained the rarity of peptic ulcer in infancy, its infrequent and equal sex distribution in childhood,[6] its predilection for men in adult age, and its decline in frequency, and more equal sex-distribution, in old age. To this may be added the rarity of peptic ulceration during pregnancy,[7] even in those who are normally subject to it, but the greater frequency during lactation, since it has been shown that the gastric acidity falls during pregnancy but rises during lactation.[8–10] Finally, there is also explained the greater incidence of peptic ulcer in those whose *natural level of gastric acidity is higher than average.*[11]

All the above references to peptic ulcer in the two sexes and at various ages involve duodenal ulcer rather than gastric ulcer, which will be readily understood from what was set out earlier in the chapter. It will now be desirable to go more closely into the question of these two forms of ulceration.

More than a generation ago Hurst[12] contended that duodenal ulcer tends to occur in persons of hypertonic build, with hypertonic ('steerhorn') stomachs, that empty rapidly, whereas gastric ulcer tends to occur in persons of hypotonic build with hypotonic ('fish-hook') stomachs, that empty slowly. That is, hypertonia and rapid emptying tends to localize the ulcer in the duodenum, while hypotonia and slow emptying tends to localize it in the stomach. This view has had much appeal ever since. Thus Ogilvie,[13] some thirty years later: "Gastric ulcer is usually associated with a long stomach of poor tone emptying slowly and with a level of acid secretion below the average, duodenal ulcer with a high stomach emptying rapidly and a high acid curve." Many other writers could similarly be quoted.

It is clear that the evolutionary argument advanced in these pages does not support the view that the duodenal localization of peptic ulcer depends on hypertonia and rapid emptying, but instead indicates that this site is the normal one for peptic ulcer and that the emptying-time in cases of duodenal ulceration will therefore usually also be normal. (This does not, however, exclude hypertonia and rapid emptying, if present, from being an aggravating factor, as will be seen from the next paragraph.)

It is interesting that some physiologists to-day strongly deny that duodenal ulcer is accompanied by rapid emptying.[14] However, the matter is complicated by the tendency of active duodenal ulceration to interfere with the normal motor functioning of the stomach and so to alter the natural emptying-time in the person concerned.

When it comes to gastric ulcer, however, the evolutionary argument solidly supports Hurst's original contention, though in a secondary sense. For an acidity of the gastric contents that is only a little higher than natural for a person (and possibly subnormal for most people) may, when acting for a long time, produce the effects of a much higher acidity acting for a short time. Any slowness in emptying, therefore, increases the danger of ulceration in the stomach and, by allowing the alkaline contents of the second part of the duodenum to diffuse more freely into the first part, shifts the danger away from the latter. It is interesting that at the present time there would be very little opposition from anyone to Hurst's original view where gastric ulcer is concerned. According to Schinz, Baensch, Friedl, and Uchlinger[15] 86·4 per cent of gastric ulcers occur in slow-emptying stomachs, whilst Kohler[16] considered the figure to be 90 per cent. Similarly, Dragstedt[17] and Daintree Johnson[18] have especially stressed the factor of stasis in the causation of ulcers in the body of the stomach.

It now becomes understandable why gastric ulcer is more prominent in women, in the elderly, and in those in poor health,[19] since cases of marked hypotonia and delayed emptying are more liable to be found in them. It must, however, be pointed out that if the primary cause, advanced here, were not operating, it would seem unlikely that slowness in the emptying of the stomach could produce a gastric ulcer on its own. That is why it was stated above that the argument only supports Hurst's view in a secondary sense.

The relationship between hypotonia and delayed emptying, on the one hand, and gastric ulcer, on the other, is exceptionally well seen in the case of concomitant ulcers of the stomach and duodenum. In these almost invariably the duodenal ulcer precedes the gastric ulcer, and not vice versa. The sequence appears to be duodenal ulcer—pyloric stenosis or spasm and delayed emptying —gastric ulcer—healing of duodenal ulcer. Thus, in a series of 119 patients with concomitant ulceration, investigated by Daintree Johnson,[20] "64 per cent unquestionably had gastric retention and most of the rest may have had it". And "at operation the commonest finding is an active gastric ulcer and a healed, scarred

duodenal ulcer". It therefore appears, exactly as described above, that the delayed emptying has increased the danger to the stomach and, by allowing the alkaline contents of the second part of the duodenum to diffuse more freely into the first part, has reduced the danger to the duodenum.

The factor of delayed emptying in the production of gastric ulcer is also well seen in the case of those acute gastric ulcers in young women, which were once so common but which declined so mysteriously at the turn of the century. There now seems no doubt that these originated from the tight corseting that occurred in those days, which likewise, possibly by pressure on the liver, was responsible for a typical anæmia (chlorosis), the occurrence of which was confined to the same epoch of history. Ivy, Grossman, and Bachrach[21] consider that the tight corseting produced the ulceration by direct pressure on the stomach wall, across the vertebral column, of which they consider demonstrable post-mortem grooving of the stomach was evidence, but the present writer finds himself in close harmony with the view of Daintree Johnson that the pressure acted indirectly, through interference with the passage of the stomach contents. It is clear that such an explanation at once brings these cases into line with other examples of gastric ulcer occurring in stomachs with delayed emptying.

Finally, on the present argument, the fact that the localization of a peptic ulcer to the stomach requires the presence of a delay in the emptying-time explains why gastric ulcer has not increased very much over the last century. Although the writer is not convinced that a delayed emptying-time could produce a gastric ulcer on its own, i.e., in the absence of the primary cause advanced here, he is equally convinced that the emptying-time is of crucial importance in this connexion. As the cases showing marked hypotonia set out above are not more common to-day than they were, say, a century ago, no great increase in gastric ulcer over that period is to be expected, even though the dietetic cause has become more pronounced. But according to the present view *some* increase must have take place, though it has been partly masked by the decline in the gastric ulcers in young women just referred to.

There remains to be discussed the incidence of gastric and duodenal ulcer in different social classes and different occupations, which has been set out very carefully by Doll, Avery Jones, and Buckatzsch.[22] This subject will be partly referred to in the next chapter, on the influence of stress, but two salient features must

be dealt with in the present one—the high incidence of gastric ulcer in the unskilled labouring classes, especially as compared with the upper professional classes, and the low incidence (chiefly of duodenal ulcer) in agricultural workers.

As regards the high incidence of gastric ulcer in the unskilled labouring classes, the writer is convinced that this is related to the condition of the teeth in these classes.

An association between an unhealthy state of the teeth and gastric ulcer has indeed been recognized for many years. Hurst[12] considered that this might be the result of a direct infection of the gastric mucosa from the pus produced in parodontal disease, but in the writer's opinion there is a much simpler and more convincing explanation, as follows.

The presence of defective teeth or imperfect dentures results in a noticeable reduction in the efficient mastication that accompanies the natural healthy bite, and the efficiency of mastication has a direct bearing on the physical texture of the food swallowed, which in its turn has a direct bearing on the gastric emptying-time. For coarse foods, whether due to a high content of vegetable fibre or to the fact that the food has been swallowed in chunks, suffer a slow rate of passage through the stomach. Schinz has already been quoted on this point in Chapter IV, but more precise evidence on the matter in the present context is available from the work of London,[23] who showed that lumps of meat, for example, are retained much longer in the stomach than is minced meat. From what has been written above, therefore, a slower gastric emptying-time in persons with defective teeth or imperfect dentures could well explain in them an increased incidence of *gastric* ulcer. Needless to say, the deficient bite also reduces the impregnation of the food with saliva, and also achieves such poor subdivision in the food that it encounters the gastric juice very imperfectly. The loss in buffering power on each of these counts necessarily increases the tendency to ulceration, but it is the *poor subdivision*, influencing the gastric emptying-time, that sites the ulcer in the stomach rather than in the duodenum.

With regard to the condition of the teeth and the presence of dentures in the unskilled labouring classes and upper professional classes, though no exact figures, unfortunately, seem to be available, there is not a dental surgeon in the country who is not aware of a very marked difference between the two classes in this respect. Thus, Professor J. L. Hardwick:[24] "I would certainly agree that

the unskilled labouring classes as a whole have very much less reparative dental treatment than the upper professional classes, and therefore have deficient teeth or complete dentures years earlier." There exists in this difference, therefore, a perfectly rational explanation for the different incidence of gastric ulcer in the two classes.

Furthermore, this explanation ties up in striking manner with the big rise in the incidence of gastric ulcer, as opposed to duodenal ulcer, that will be shown later (Chapter XII) to have taken place in all the enemy-occupied countries during the last war. Here a great coarsening of the food took place, and the result came to much the same thing as a fall in the efficiency of the bite. It is highly significant that these gastric ulcers arose largely in women and old people, who lacked either the muscular power or the teeth to masticate such food properly. Meanwhile in England, where no comparable coarsening of the diet took place, no such shift in the incidence of gastric and duodenal ulcer took place either.

Whether, however, a disparity between the physical texture of the food and the efficiency of the bite could lead to peptic ulceration in the absence of the primary cause advanced in this work is another matter. In the writer's opinion such a possibility is not in obvious conflict with the law of adaptation and must, with some hesitation, be allowed. It has to be remembered, however, that with the coarsening of the food on the Continent during the last war there was still consumed a certain amount of sugar and, as will be shown later, large quantities of saccharin, so that the matter cannot be regarded as settled on the score of the evidence there anyway.

As regards the lower incidence of peptic ulcer, especially duodenal ulcer, in agricultural workers, this reflects to some extent the lower incidence of peptic ulcer generally in the country as opposed to the towns. Morris and Titmuss[25] established that the mortality-rate of peptic ulcer for men was 75 per cent higher in London than in the rural areas of England and Wales, and the same thing has been shown to be true in Sweden and Italy. This lower incidence of peptic ulcer in the country, which reveals itself in the lower incidence in agricultural workers, is not out of harmony with the present argument. For it is clear that though the lower cost of distribution favours the consumption of processed foods in the cities, this is not quite so true in the country. Here the apple is a little less likely to be replaced by the sticky sweet.

Thus, it may not be entirely coincidental that Pulvertaft,[26] writing on the greater incidence of peptic ulcer in the towns than in the country, concludes his study with the following:—

> In the course of the study it was observed that the urban population consumed more sweet or sugary food than their rural counterparts. The quantity of sugar added to beverages has been used as a measurement. It was found that a significantly larger number of male duodenal ulcer patients, in both town and country, took sugar than the corresponding control groups. This is reported purely as an observation, as no causal relationship has been established.

Furthermore, a less rushed and more orderly mode of existence in the country leads to closer attention to the instinct of appetite, the great importance of which will be set out in the next chapter.

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CHAPTER XI

Disregard of the Appetite an Aggravating Factor in the Production of Peptic Ulcer

Influence of Stress a Special Example

Part I

Disregard of the Instinct of Appetite

It is now proposed to discuss what is considered to be an important aggravating factor in the production of peptic ulcer—the eating of food in the absence of any proper appetite for it. It must be very doubtful whether this unnatural act can ever of itself cause ulceration, but that it is frequently responsible for considerable aggravation of the primary cause, already advanced, appears certain.

The basis of the discussion is the fact that the size and frequency of the peristaltic waves, and therefore the rate of emptying in the stomach, are largely determined by the degree of hunger present.[1] Ivy and Farley,[2] confirming the work of earlier investigators, showed, indeed, that most of a series of human subjects averaged a fall of 40 minutes in the gastric emptying-time in a meal following a 24-hour fast.

In the resting, empty stomach there arise, in fact, peristaltic waves of such magnitude that they intrude into consciousness, producing definite sensations—the pangs of hunger. Physiologists, using intragastric balloons, have demonstrated these large hunger contractions in the healthy stomach, not only in man (Carlson, 1919, confirming earlier work by Cannon and Washburn),[3] but also in animals and even birds.[4] However, no one who has himself experienced the pangs will require to be convinced of their reality.

If, then, a meal is eaten in the presence of hunger, vigorous peristaltic action leads to a satisfactory rate of emptying in the stomach, but if a meal is eaten in the absence of hunger, or without any desire for that particular food, the peristaltic action becomes feeble and the passage of the food correspondingly sluggish. The relatively stagnant food, however, is liable to cause a continued

reflex secretion of juice, so that the acidity in the stomach may reach an unnaturally high level. Even if the stagnation does not result in this high level, it certainly will result in an unnaturally long exposure of the gastric membranes to the normal peaks of acidity that occur during digestion, noted earlier in this work.

This is not to say that if the stomach empties slowly, the rate of climb in the acidity is not reduced. It is well known and is, indeed, obvious that such slowing in the climb does occur, since the food necessarily acts as a diluent and buffer to the juice secreted, but this fact does not preclude an eventual high level being reached and persisting for a long time.

To the results of the above delay in the emptying-time must be added a tendency for any accumulation in the gastric contents to regurgitate into the œsophagus, the reflux producing a scalding sensation at the back of the mouth and throat.

To sum up, the unnatural act of eating without desire leads to a disjointed action in the stomach, in which the motor function is more impaired than the secretory one.

That eating without desire does cause this stasis in the stomach, with its unfavourable effects, is not only supported by the experimental results already quoted, but is also supported by personal experience. For there must be few observant people who do not sooner or later discover one of the surest facts that exist concerning digestion, or rather indigestion—that if they eat something they do not want they are very liable to get this 'acidity' at the back of the throat. Even a glass of milk, normally so easy to digest, will cause this symptom if taken when it is not wanted, whereas a pork chop, one of the most difficult of all foods to digest, will, when eaten with ravenous hunger, become like a ship sunk without trace, never to be heard of again. The author himself has never yet seen a case of acid dyspepsia ('peptic œsophagitis'), with its heartburn and scalding eructations, that was not immediately improved by keeping the patient hungrier for his food. The 'mystery' in this improvement, described in the *British Medical Journal* of Oct. 28, 1961, will not be apparent to anyone following the present argument. The complete cure, however, demands also the avoidance of concentrated carbohydrates, shortly to be set out.

It is clear that if the essential cause of peptic ulceration lies in insufficient buffering of the gastric acid, through interference with the protein content of certain foods, as advanced in this

work, then any stasis of the type just described must act as an aggravating factor, either through allowing the gastric acidity to reach a still higher level or to act for a longer period of time. This is especially true in the case of gastric ulcer, the production of which, it will be remembered, is always favoured by the occurrence of stasis.

In the author's opinion the reason why special food products of high buffering capacity, elaborated by biochemists and pharmaceutical firms, have failed in the prevention and treatment of peptic ulcer is almost entirely because such products have ignored the question of whether hunger has been present before the eating of them, and to some extent whether the prospect of their consumption has caused delight in the patient. Consequently, delay in the gastric emptying-time has produced the very opposite result to that intended. Such arbitrary food mixtures are in any case incompatible with the accurate following of the instinct of appetite, as will be seen in the general examination of this subject, now to be pursued.

As a preamble, it is necessary to emphasize the personal idiosyncrasy that exists in food consumption, and here it is important that whereas man towards the Arctic Circle is almost purely carnivorous, towards the Equator he may be almost purely herbivorous (vegetarian). The Eskimo, for example, lives very largely on meat and fish, whereas races near the Equator often subsist mainly on roots, like those of the yam, manioc plant, and sweet potato, and on cereals such as maize and rice. Even so powerful a higher ape as the gorilla is a pure plant-eater. It must be noted, too, that all meat-eating animals are accustomed to isolated big meals, following a kill, whereas plant-eating animals have to eat much more frequently, since their food is much less concentrated.

In conformity with the difference in man's food supply in these two widely separated regions of the globe, and his necessary evolutionary adaptation thereto, it is clear that there must occur some expression of this difference even between the inhabitants of northern and southern Europe. Consequently, inhabitants of Great Britain, descended as they are from invaders from one or other of these areas, must exhibit some of this difference, too. It would be expected, for example, that a person of fair complexion would show a ratio of choice between animal foods and plant foods appreciably different from that shown by a person of dark complexion. The former would be expected to have a distinct

liking for meat and not to have 'a sweet tooth'; and to prefer large, infrequent meals, whereas the latter would be expected to show the opposite tendencies. These personal idiosyncrasies in food consumption are of great importance. "One man's meat is another man's poison" is based on a biological truth.

Under civilized conditions the delicate, individual adjustment just described, between the body and the foods in its natural environment, is likely to be brutally disregarded, so that a person frequently eats food without any real desire for it. He does this, firstly, through arbitrary meals, and secondly, through arbitrary food mixtures.

Arbitrary meals are meals eaten without any great desire, or even without any desire at all, either because the person is not hungry, or because, if he is hungry, he does not fancy that particular food combination. Starting in the nursery, where children's meals are often dictated by their parents; progressing through boarding school and possibly university hostel, where routine meals are the order of the day; continuing in business or professional life, where in circumstances of anxiety or haste the midday lunch is often ill-desired, whilst after work fatigue may similarly affect the desire for the evening meal; and in meals with friends, where politeness takes precedence to taste, a person under modern conditions is very likely to partake of a long succession of these arbitrary meals. Finally, the best example of all is seen in the advice that has so frequently been given to ulcer sufferers, to keep the stomach from getting empty. This advice, which signally disregards the instinct of appetite and is, therefore, correspondingly at variance with the law of adaptation, has been based on the view that the gastric acidity is highest in the empty stomach—a view now known to be incorrect, as shown earlier in this work. Unless the natural eating-pattern in the person happens to be one of very frequent, small meals, such advice is seen to be grievously ill-founded. The matter will be pursued in the chapter on treatment (Chapter XIV).

In other cases arbitrary meals are not imposed on the individual in this way, but are taken voluntarily by him—though for an ulterior motive, unconnected with the appetite. Important examples include meals, and parts of meals, that are eaten to save them being wasted; meals eaten because it is considered they will do the person good; meals eaten because of loneliness, in order to obtain companionship; and meals eaten automatically

during discussions with colleagues and business associates, often under the influence of a considerable consumption of alcohol.

In all these arbitrary meals a person eats without any real desire, suffers considerable gastric stasis in consequence, and therefore, in the presence of the primary cause, renders himself more liable to develop peptic ulceration.

Turning now to *arbitrary food mixtures*, these often occur in connexion with fat-consumption, perhaps the best example being provided by fried foods. In these a food such as fish or potato is cooked in fat, and therefore becomes largely impregnated with it. As a result a person, in order to eat fish or potato he wants, may be forced to eat fat he does not want. Even if he is by nature a Jack Sprat, he is forced through an arbitrary food mixture to eat the same amount of fat as another person does.

It is probable that no other food eaten in excess of desire results in such prolongation of the gastric empyting-time as does fat. In extreme cases, as everyone knows for himself, this may end in nausea or even in vomiting. Furthermore, any such excess of fat must displace other food from the diet, which under natural conditions would be sure to contain some protein, whereas fat contains none. For both these reasons it is easy to understand why fried foods have a bad reputation with sufferers from dyspepsia and peptic ulcer. Such reasons are more logical than the one sometimes heard, that the fat-impregnation interferes with the free access to the food of the gastric juice, for in natural fatty meals efficient mastication produces just this impregnation. The dangers of excess fat-consumption from arbitrary meals and food mixtures have been described by the author elsewhere in connexion with coronary disease,[5] but they are clearly also a reality in connexion with peptic ulceration.

Many other arbitrary food mixtures to-day prevent a personal choice in the consumption of protein, fat, and carbohydrate. In plain chocolate, for instance, there has been added to the original cocoa large quantities of fat and sugar (which reduce the protein calories from 18·5 per cent to 4·2 per cent). It must often happen that any one person, when eating this chocolate, would not himself have chosen so high a quantity of fat for that amount of sugar, or vice versa; in which case he takes one of these foods in excess for the sake of the other—or he may take both in excess. Other examples of arbitrary food mixtures are ice-cream or a sweetmeat such as toffee (in which mixtures the 21·1 per cent of protein

calories in the original milk have been reduced by additions of fat and sugar to 8·6 per cent and 0·2 per cent respectively). Other examples, again, come from the kitchen, such as cake, where eggs, fats, and sugar are compounded with flour without any reference to the individual desire for these foods in the person who eats it.

Every one of these arbitrary mixtures is likely to lead to interference with the free play of the appetite, and therefore to affect the gastric emptying-time, with its influence on peptic ulceration.

If it were asked whether, since many natural foods themselves represent mixtures of nutrients, the tongue is not liable, even under the most perfect conditions, to lead to the consumption of certain of these nutrients in excess of desire, the answer would be *NO*. The reason for this lies mainly in the remarkable fact that, once man has left the specially suitable mixture of protein, fat, and carbohydrates in his mother's milk, he seldom meets any natural food where the mixture is not, for practical purposes, either one of protein and carbohydrate or one of protein and fat. Thus, in foods of plant origin, such as cereals, vegetables, and fruits, the mixture is nearly always one of protein and carbohydrate, any fat being present in negligible amount, whereas in foods of animal origin the mixture is nearly always one of protein and fat. Furthermore, the fat in animal foods, if present in any quantity, is often present in naked-eye masses, which greatly facilitates a selective consumption. Therefore, if a range of natural foods is available, the tongue seldom has difficulty in achieving optimal consumption of fat and carbohydrate, and as regards the variability of the accompanying protein, this is so valuable and usually so scarce a component that it is never likely to be taken in excess of the desire for it.

It is not denied that the tongue has to exercise greater powers of selection over the more complicated mixtures that sometimes occur in nature, but normally it enjoys much greater ease of choice than is possible with the arbitrary food mixtures of our present civilization.

To sum up, it has been shown that eating without desire, in the taking of arbitrary meals and arbitrary food mixtures, results in gastric stasis which, in the presence of the primary cause advanced in this work, of interference with the protein content in certain foods, can become an aggravating factor in the production of peptic ulcer, through increasing the exposure-time to insufficiently buffered acid. Whether eating without desire could

produce ulceration in the absence of this primary cause must remain very doubtful.

In Chapter XIV on treatment it will be seen that the whole subject of eating without desire, somewhat complicated in theory, becomes extraordinarily simple in practice, involving as it does merely the blind following of instinct—the instinct of appetite—as long as the instinct is exerted on foods in a simple, unprocessed state. For the moment, however, it will be necessary to see how stress enters the aetiological scene in peptic ulceration, which it does through this very portal of eating without desire.

Part II

Influence of Stress

In order to appreciate how stress can influence the production of peptic ulcer, it is first necessary to remember that this word is used to describe two quite separate states. One consists of sustained anxiety and the other of a continued exhibition of energy. The difference is that between feeling frightened and being in a rush. The two can be combined, since fear can engender a protective activity. They can also be quite separate; for example, in a situation where war is threatening, or the danger of some serious disease has appeared in himself or a close relative, a person may be in a continued state of great uneasiness and yet unable to exert himself over it; on the other hand, someone eager to obtain an advantage in his business or profession may be working very hard indeed, but without any alarm whatsoever. In the anxiety form of stress a person loses his appetite, or, as the saying is, goes off his food; in the striving form of stress he eats more than ever. The two forms will be considered separately.

Stress, in the Sense of Anxiety.—There could be no better introduction to the study of the effects of this form of stress on digestion than to observe the behaviour of a recently captured animal or bird. Neither of these whilst in an agitated state of mind will touch any food. It is as though Nature wishes to draw on reserves and keep the body clear of commitments during periods of danger, even those concerned with nutrition. Gradually, as the days pass and fear diminishes, the animal or bird will

begin to eat again. Herein lies the clue to the proper understanding of the subject. If a man labouring under anxiety similarly adjusted his food consumption to his reduced appetite, he would lose weight, but he would not develop a peptic ulcer. Fasting does not produce organic disease.

Under modern conditions, however, a man is unlikely during periods of anxiety to reduce his food consumption very much, however little appetite he has. One reason for this is that in his own house a carefully prepared meal imposes on him some obligation to eat it. Another reason is that he may become alarmed at not eating and will feel he must eat 'to keep up the strength'. This form of stress, in short, is likely to precipitate a series of typical arbitrary meals.

Stress, therefore, in the sense of anxiety, becomes, through the disregard of appetite in arbitrary meals, and the gastric stasis that results from it, an important aggravating factor in the causation of peptic ulcer, always assuming the presence of the primary cause. In the absence, however, of this disregard of the appetite, stress plays no part whatsoever.

It has been pointed out earlier in this work that the distinction between the above view of stress and the one normally held to-day is a vital one, since a person who understands it can endure immeasurable stress and yet never develop a peptic ulcer. Moreover, it must be remembered that anxiety is a natural protective state, subserving a definite *function*. The words of Laertes may be recalled in *Hamlet*:—

> And in the morn and liquid dew of youth
> Contagious blastments are most imminent.
> Be wary then; *best safety lies in fear*!

The author has italicized the important phase. Clearly it is a great advantage to anyone to know that as long as he follows natural principles in eating, he may experience as much fear as circumstances dictate without incurring any risk of peptic ulceration.

Stress, in the Sense of Striving.—Here the appetite is not lessened but increased, in keeping with the higher output of energy. Yet periods of intense striving are also associated with a greater tendency to develop peptic ulcer. Why is this?

The answer on the lines of the present argument is not difficult to perceive. For if peptic ulceration is due primarily to interference with the protein content of certain foods, so that the membranes are injured by insufficiently buffered acid, then the higher

the rate at which these injuries are inflicted, the greater must become the chance of ulceration. And since the more intense the striving, the higher becomes the rate of food consumption and food digestion, to supply the extra energy, the higher also must become the rate of injury of the membranes. The increased incidence of ulcer during periods of intense striving, always assuming the presence of the basic cause, is thus explained. And because in this case the eating has only altered quantitatively and not, as in the anxiety form of stress, qualitatively (through eating without desire), any resulting ulcer will probably be in the usual duodenal position.

At the same time there is explained the tendency in all ulcers to heal up, even in the presence of causative foods, if the patient be put to bed. For with the fall in energy output and attendant food consumption, the rate of injury to the membranes will likewise fall.

Finally, there is similarly explained the well-known tendency for peptic ulceration to occur more frequently during cold weather —the fall in temperature raises the consumption of (causative) foods and the injury increases. It is suggested that this is a more logical explanation than one based on the mucin contents of the gastric membranes being "known to change in the fall and spring"[6]—an explanation astonishing to those who appreciate the law of adaptation (and perhaps even to those who do not).

As previously stated, the two forms of stress may be combined in the same person, in which case a composite picture logically results, but the essential point is that no matter what the combination, only loss of weight can occur as long as the food has not been tampered with and the dictates of appetite have not been ignored.

Occupation and Personal Disposition (Personality) in relation to Stress and Peptic Ulceration.—It is perfectly clear that the two forms of stress just described will be increased if, in the anxiety form, a person is in a worrying occupation, especially, as happens with doctors, where the occupation interferes with the eating of meals; and if, in the striving form, a person is in an occupation that presents extra scope for advancement or profit, like a business executive's. It is also clear that the two forms of stress will be increased if, in the anxiety form, a person has a nervous disposition, or, in the striving form, an ambitious one. In theory, therefore, some occupations and

dispositions, through the mechanisms described, conduce to peptic ulceration. And, equally, others have the reverse effect.

In practice it has, indeed, been found that occupations do affect the incidence of ulcer. For example, doctors and business executives have a high incidence and agricultural workers[7] a low one, though in the latter case another factor has been seen to be possible, too. In the case of personal dispositions a comparable effect is less certain, since some hold that the influence of a nervous, shrinking type of personality, perhaps more often associated with gastric ulcer, or of a strong, aggressive type of personality, perhaps more often associated with duodenal ulcer, is a myth.[8]

That personal disposition at any rate is unimportant, compared with more fundamental factors, is seen in the incidence of peptic ulceration in Scotland and France. Here the personal dispositions are the opposite of each other, and it might be argued that the cool and phlegmatic Scots should be more immune than the sensitive and excitable French, but in fact statistics show that the death-rate from the disease in Scotland, which is the highest in Europe, is nearly double that in France, which is the lowest in Europe.[9] The difference in incidence would, therefore, appear to lie in something more fundamental than personal disposition, and in this respect is it purely fortuitous that the French have a higher regard for the pleasures of the table—that is to say, pay more attention to the appetite—than perhaps any other nation? A consumption of refined carbohydrates, which, however, in the case of sugar is less than two-thirds of the amount per head consumed in the United Kingdom,[10] and a marked prevalence of alcoholism, ensure that there is plenty of peptic ulceration in France, but at least there is absent the aggravation in the incidence due to disregard of the appetite. Many of the meals seen in this country would be thrown into the sink in France, as anyone knows who has lived in that country.

After much study the author has come across only one case where, to judge by the incidence in a large mass of people, personal disposition does appear to influence the incidence of peptic ulcer. He considers that in Africa the incidence of the disease in the urbanized Bantu, and especially *the urbanized Zulu*, is lower than would be expected from the consumption of refined carbohydrates already taking place in them, and that if this is so, it is probably related to personal disposition. According to N. McE. Lamont,[11]

with a profound experience, this disposition in the case of the Zulu shows such insouciance that "rather than make provision for the morrow, he will spend his last few shillings on a musical instrument". Nevertheless, the author is convinced that as these people experience the full impact of the consumption of refined carbohydrates, their incidence of peptic ulcer is going to rise, until an insensitive disposition stands them in little more stead in Africa than a tendency to the same disposition does their cousins across the Atlantic.

It appears, therefore, that merely as aggravating factors in the production of peptic ulcer, personality and occupation are relatively of little importance when compared with the main cause advanced in this book. Hence, though these factors make some people more vulnerable to the disease, so that particular attention should be paid by them to the appetite, still greater attention should be paid to the actual type of food eaten. And this applies to stress situations generally.

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CHAPTER XII

Peptic Ulcer and the War in Europe, 1939–45

BEFORE studying the effects of the last war on the incidence of peptic ulceration throughout Europe, from which much may be learned, it is constructive to cast a glance at the effects of the famine during the Bolshevik revolution on the incidence of the disease in Russia twenty years earlier.

The incidence of peptic ulcer during this famine is set out in one dominant paper by H. Hemperl,[1] which makes allusion to no less than 189 other German and Russian references bearing on the same subject. The reason for this activity in the literature lay in a veritable epidemic of peptic ulceration in Russia during the period in question. Freely quoted figures show a rise in ulcer cases from under 1 per cent of hospital admissions before the 1914 war to no less than 17 per cent in some areas during the famine of 1918–21. Since no dietetic treatment seemed possible at the time, a very large proportion of the cases were treated surgically, by gastro-enterostomy, until the unfavourable after-effects of the operation became known and made this form of treatment less popular.

Now, it is accepted that starvation of itself does not cause peptic ulceration. Quite apart from such a causation being incompatible with the law of adaptation, there is the known fact, as previously pointed out, that hunger-strikes and other forms of fasting are not attended by manifestations of the disease, nor have physiological studies in human starvation[2] been attended by it. There is also much epidemiological evidence to the same effect, *as long as refined carbohydrates are not being taken.* This was seen in certain of the Japanese prison camps already described, and there is similar evidence from the terrible famine in the Warsaw Ghetto in 1942, where no refined carbohydrates were consumed and where peptic ulcer was one of the rarest diseases encountered.[3] It is therefore intriguing to try to deduce what was the cause of the ulcer epidemic in the Russian famine, now under consideration.

In this respect it is highly significant that it was above all in the *peasants* that the ulcers occurred—and also in the people who

had fled from the towns into the countryside. This is brought out in many references in Hemperl's paper. Now if one thing is certain, it is that these peasants were not eating refined carbohydrates. What then were they eating that might have been the cause of the trouble? The answer may well lie in the following quotation (in translation) from this paper, showing the type of food being eaten by the peasant population in a typical area (Perm) at this time:—

> Seventy-five per cent of those questioned had eaten black bread, especially black bread with an admixture of oats (artiplex) or made from pure oats or barley flour. We do not wish, however, to speak of this section; we count them and they count themselves among the most favoured in the matter of nutrition. The other 25 per cent used over a more or less lengthy period of time the following substitutes: mare's tail, ground straw, peas, buckwheat, or rye-meal, bone-meal, stinging nettles, raspberry leaves, cabbage (brassica), pith from young lime-trees, sawdust or wood which is cut in the mills from young lime-trees.

It is submitted that if there were anything worse, *vis-à-vis* the buffering of the gastric acid, than a carbohydrate food which had suffered a partial or complete removal of its protein component, it would be a carbohydrate that was not a food at all—such as wood pulp, sawdust, straw, etc. Materials of this sort, quite apart from their resistance to any fine subdivision by mastication, have their protein component so intimately incorporated in masses of fibre that any interaction between the protein and the gastric juice would be negligible. Meanwhile, being of extremely coarse texture, these materials would, as already seen (Chapter IV, p. 20), suffer a very slow rate of passage through the stomach. Furthermore, it is known that though the gastric acidity does not alter appreciably during starvation,[2, 4] the emptying time in the weakened stomach becomes greatly prolonged.[2] For both reasons, therefore, the stay of these materials in the stomach would be a lengthy one, and during the whole of this time they would be liable to cause a reflex secretion of juice, just as a pebble in the mouth causes a flow of saliva. A resulting injury to the membranes from unbuffered acid would therefore be just as probable as in the case of refined carbohydrates—and perhaps even more probable. Clearly any ulceration in these cases would be more likely to occur in the stomach itself, as already seen in other conditions which prolong the gastric emptying-time. It is highly significant that most of the ulcers during the Russian famine were *gastric* ulcers. An aggregate of many reports showed

906 gastric ulcers to 240 duodenal ulcers, and in another series there were 859 perforated gastric ulcers to 264 perforated duodenal ulcers.

The reason why the townspeople were much less affected is presumably because they were much less able to get at these non-food carbohydrates. Where they were able to get at them, as in Tomsk and certain other towns in Siberia, the ulcer incidence rose sharply. The Russians themselves nearly always related the ulcers to the consumption of these ultra-coarse materials. One writer quoted a case in point—a woman of 38 years had eaten some bread made with straw; some hours later she was seized with symptoms of a perforation.

If it be argued that the materials described above caused peptic ulceration by direct trauma of the membranes of the stomach, in the presence of the gastric acid, and that they did not cause the ulceration, as advanced here, exclusively through the action of unbuffered acid, it will be remembered that the first explanation would not tie up with the virtual absence of the disease in communities consuming coarse, unrefined food-carbohydrates, as already described in various countries and in some Japanese prison camps (and also, as will shortly be shown, in the German army on the eastern front), whereas the second explanation ties up perfectly with this absence.

Turning now to the effects of the last war, 1939–45, on the incidence of peptic ulcer in Europe, it must be stated at once that, owing to the crisis-existence at the time, only imperfect records survive to-day in all the countries that were overrun by the enemy, and this makes it extremely difficult to obtain a reliable picture of what occurred. Nevertheless, enough information is available to see that the pattern of incidence has obvious affinities with that in Russia during the famine just described. (The big difference in this respect presented by the United Kingdom, of deep significance, will be noted later.)

Thus, reference after reference emphasizes the great coarsening of the food that took place in all the enemy-occupied countries. The various populations, driven to the starvation line, had to subsist largely on vegetables. For example, in Guernsey people were getting over 900 calories a day from 2 lb. of parsnips, 1 lb. of swedes, 1 lb. of turnips, 1 lb. of cabbage, and ½ lb. of onions or carrots.[5, 6] Meanwhile, in all countries the bread became of the crudest type and, owing to the scarcity of fuel, was often

incompletely baked, so that the centre of the loaf would not infrequently have been considered uneatable, by ordinary standards. Furthermore, some of the constituents of the bread were often not cereals at all; thus, the Belgians talked of "the beastly bread made from peelings and horse-beans" (*le sale pain d'épluchures et de féveroles*).[7]

Though death from starvation in occupied Europe was by no means rare, as many references testify, the author has not been able to unearth any evidence of the consumption of non-food carbohydrates on the Russian model. However, the consumption of the coarse food-carbohydrates just described could well have the same effects, *vis-à-vis* the buffering of the gastric acid, *in people who lacked the power to masticate them adequately*. Such people would be those of poor musculature or with deficient teeth, and would include, especially, many women and older persons. It is highly significant that a rise in the incidence of peptic ulceration took place in all these European countries, but it occurred chiefly in *women* and in *old people*, and most of the ulcers, as in the Russian famine, were *gastric ulcers*. This is borne out in nearly every reference, and as a consequence there resulted a big shift in the gastric ulcer–duodenal ulcer ratios.

Thus J. de Witte,[8] pointing out not only the big increase in the average age of ulcer-sufferers in Belgium during the war, and in the proportion of female sufferers, stated that the percentage of gastric ulcers rose from 23 per cent to 40 per cent in men and from 30 per cent to 72 per cent in women, and that there was not only a relative fall in the incidence of duodenal ulcer, but also a slight actual fall.

Similarly, A. Lambling, J. R. Gosset, J. Bertrand, and P. Vian,[9] pointing out the big increase in France in the average age of ulcer-sufferers, many of whom were over 60 years of age, and in the proportion of female sufferers, stated that the percentage of gastric ulcers rose from 17 per cent to 55 per cent in men, and from 25 per cent to 69 per cent in women. They referred to parallel figures from Hungary,[10] and even from Germany itself.[11]

Other evidence is available from Germany in strong support, and showing that the increase in incidence of gastric ulcer was at the expense of duodenal ulcer.[15] There is also similar evidence from Soviet Russia (1942–5).[12]

At this juncture it must be pointed out that a sugar ration was issued in all the occupied territories. For example, in Guernsey,

perhaps of all these territories the one most accessible to inquiry to-day, and in which, incidentally, there also occurred a big rise in the percentage of *gastric* ulcers, this ration averaged about two-thirds of an ounce per person per day for much of the war,[6] whilst in Belgium, even as late as January, 1945, the ration was double this amount.[7] Such sugar-consumption, though small by normal European standards, is of importance to the argument presented here. But much more important in the same connexion is the consumption of saccharin in all these territories. This occurred on a large scale. For example, in Guernsey the saccharin ration reached a total of 950 tablets per person for 1944, and considerable further quantities were bought on the black market,[6] whilst in Germany itself there was an average consumption of *at least* 3 tablets per person per day.[13] The importance of this saccharin consumption lies in there being no reason to suppose that the secretion of gastric juice, produced reflexly by stimulation of the taste buds in the tongue, would be any smaller with saccharin than it is with sugar. After all, in a famished person a cup of sweet tea would make the mouth water whether it contained the one substance or the other. It is hardly necessary to add that both substances are equally useless as bufferers of the gastric acid. In view of the issue of this sugar ration and saccharin ration it is quite understandable, on the present argument, why duodenal ulcer continued to be seen in the occupied territories, though never, apparently, to any great extent.

Meanwhile, as the war proceeded, and because, in the author's opinion, the body gradually adapted itself to these coarse foods (for example, by hypertrophy of the jaw muscles, *and almost certainly by hypertrophy of the stomach musculature, too*), a big fall took place in the ulcer-incidence in every country, as many references reveal.

Peptic Ulcer in the German Army on the Eastern Front.—Attention may now be directed to one of the most remarkable facts in the whole epidemiology of peptic ulceration—the near-disappearance of the disease in the German army before Stalingrad and on the eastern front generally. This development appears to be unknown in this country, though appreciated well enough in Germany itself.

Briefly, at the outbreak of war in 1939, peptic ulcer (chiefly duodenal ulcer) was so common in the German army that actual 'ulcer battalions' were formed, to enable the sufferers to have

special food. This high incidence will occasion no surprise to anyone who has followed the present argument, for on the one hand refined carbohydrates were still in plentiful supply, and on the other hand personal tastes over various foods were necessarily brushed aside when conscripts and reservists entered regimental life, so that many arbitrary meals were consumed. Later, when these refined carbohydrates became scarcer and the men got more accustomed to army food, the incidence fell considerably, though it never became very low.

It was, therefore, a source of astonishment to the German medical authorities that peptic ulcer in their troops on the eastern front became a rare disease, though the incidence rose all the way back, along the supply lines. There are many references on this subject, but they are all marshalled in two masterly papers by H. Glatzel,[14, 15] who was himself medical officer of a rocket regiment on this front. The present author is deeply indebted to this authority, not only on account of the papers themselves, but also for invaluable personal communications, and he can only hope that Professor Glatzel will forgive him for being unable to subscribe to certain psychological explanations advanced by him for what occurred.

And what occurred, as already stated, was a great fall in the incidence of the disease on this front. Thus, in rear hospitals ulcer cases constituted 3·2 per cent of admissions; in field hospitals nearer the front line they constituted 1·5 per cent; whereas in the front line itself they were almost never seen. Professor Glatzel quotes first-hand interviews in this respect with medical officers of other regiments. One of these medical officers during eighteen months in front of Stalingrad, amongst hundreds of thousands of troops, stated that he hardly saw a case, and that even old ulcer cases kept free from relapse. Other medical officers were of the same opinion. What was so striking to all of them was the difference in this respect between the front line and the lines in the rear.

Since every single factor on the eastern front seemed to predispose to ulcer development, such as (1) anxiety, (2) cold, (3) fatigue, (4) ultra-coarse foods, and (5) deficiency in animal protein, the German medical authorities were driven to explain the disappearance of the disease by the most complex psychological arguments. These arguments appear to be based on the supposed blissful absence, in those engaged in actual fighting, of

personal psychological problems, including the normal anxieties and difficulties in civilian life, and the striving for promotion in the military one. The problems presented by streams of machine-gun bullets, rockets, and shells, which to the ordinary man would seem a thousand times worse, were not, apparently, allowed to count, but in truth the present writer could not hope even remotely to do justice to these arguments in English (or, let it be quickly added, in any other language), and those interested should consult the original references. Such psychological arguments appear to be quite incompatible with the normal process of evolution, since they point to man being liable to develop peptic ulcer unless exposed to the greatest possible dangers; and they also appear fantastic on purely common-sense grounds, since a more perfect source of psychological trauma than the appalling conditions of climate and violence obtaining at Stalingrad, where nearly half a million Germans lost their lives, can hardly be imagined.

On the other hand, an explanation based on the food structure, in accordance with the argument presented in these pages, is immediately possible. For it is just here that a most remarkable change occurred.

The account of this change begins with the ever-lengthening supply line of the advancing German army, which at Stalingrad reached a point, even as the crow flies, 1250 miles from the homeland, a distance only 250 miles less than that of our own country from Moscow. At first the food supplies kept pace with demands, but as the Germans were not prepared for a winter campaign, the situation rapidly deteriorated. The roads soon became marshes, or got deeply covered with snow, and munitions were given priority over food, so that the troops were forced to eat whatever local produce they could lay their hands on. At first they were able to use stocks of grain left behind by the Russians, which were consumed in the crudest state, but later the only local food available was that left actually growing in the fields, and this consisted mainly of turnips and potatoes. These were eaten largely uncooked. "The turnips were taken out of the earth, and after scarcely being cleaned, were eaten raw; and the same was done with the potatoes, after being peeled.[16]

At this juncture it must be pointed out that the bread in the German army was already pretty coarse (the flour, according to Professor Glatzel, being of at least 80 per cent extraction), so that a bigger difference in the nutrition of these troops lay in the fall in

the consumption of noodles, made with white flour and consumed in very large amounts, and of sugar and sweets, the latter of which on this front were largely issued before special exertions. That a big total decline in the consumption of refined carbohydrates took place is certain. Thus Professor Glatzel, in answer to the author's further inquiries: "It is beyond discussion that the front line troops were supplied with less refined foods than those further back. As I have repeatedly informed you, the nutrition in these troops was much coarser and heavier, because of the difficulties in the supply line."

If the consumers of this ultra-coarse fare had been frail women or old people, who lacked either the muscular power, or the teeth, to masticate it properly, the present author feels certain that they would have experienced a considerable incidence in *gastric* ulcer, but the present consumers were in a very different category, being battle-hardened men, over 90 per cent of whom were under 50 and nearly 80 per cent under 40 years of age, and who were, therefore, well able to masticate what they ate. And the result was the dramatic fall in the ulcer incidence that has already been noted.

This fall came as just as big a surprise to old ulcer sufferers themselves as it did to their medical officers. Thus: "The patients often wondered at this strange behaviour in their complaint, which in times of peace they used to blame on dietetic indiscretions or the cold, and which now did not materialize, in spite of the consumption of frozen vegetables, sour bread and raw turnips."*[14]

Meanwhile, on the supply line towards Germany the food of the troops approximated more and more to the normal, and, as already stated, the incidence of ulcer rose correspondingly.

Peptic Ulcer in German Prisoners of War.—The climax of this whole incidence-story concerns the Germans captured by the Russians and placed in prisoner-of-war camps. This subject is likewise extensively pursued in Professor Glatzel's papers, and it would appear incontestable that the disease in these prisoners was one of the greatest rarity, as the following extracts reveal.[15]

* Die Kranken wundern sich oft genug selbst über dies merkwürdige Verhalten ihrer Beschwerden, die sie von Friedenszeiten her auf „Diätfehler" oder „Erkältung" zurückzuführen gewohnt waren und die nun bei gefrorenem Gemüse, naß-saurem Brot und tagelanger Rübenrohkost oder Erbsen-Speck-Suppe völlig fehlen.

Thus Zschau, whose report was based on careful clinical and autopsy studies during captivity, was amazed at the lack of peptic ulceration in the prisoners, and all the other physicians who were captured agreed with him.

Again, Paschlau, after examining in Germany 2000 repatriated prisoners in 1948–50, gave opinion that the coarse foods of Russian captivity, signally lacking in animal protein, had not only not resulted in peptic ulceration but actually appeared to have prevented it, since many old ulcer cases, which remained symptomless during four or five years of captivity, relapsed on return to Germany.

Finally, W. Burgmann, one of the greatest German authorities on this subject, to whom the author was referred by Professor Glatzel, has been kind enough to send the following personal communication (1962):—

I was a prisoner of war in Russia from 1944 to 1948. In several of the 22 camps in which I was interned I systematically searched for symptoms of peptic ulcer (X-ray examinations not being possible, of course). In other camps I asked the surgeons about ulcer symptoms, including bleeding and perforation. Yet though they were of the ulcer age, I suspected an ulcer only twice amongst many thousands of prisoners—and these two men were not the normal kind of prisoners, but belonged to the 'camp bourgeoisie' and received better food than we did. After their release many of those who had suffered from typical ulcer trouble before captivity, but had been free from it in the Russian camps, relapsed on their return to Germany.

In answer to your special questions: Our bread in the Russian camps did not consist of refined flour, but of unrefined wheat, rye, barley or maize, with frequently up to 20 per cent of peas, beans, or soya bean. We were fed the Russian way. Cabbage soup played a dominant part —for cabbage is the main vegetable with the Russians. Besides that, we had soups and mashes made from maize, millet, oats, and soya beans. Animal protein was almost completely missing, as we had no milk, cheese, eggs, or meat. The ration of fat was extremely small. Sugar, if any, 5 grammes a day [i.e., one-fifth of an ounce]; later 10–30 grammes [i.e., up to 1 ounce].

An explanation for this complete lack of a disease under such conditions, normally so widespread amongst German men, I could not find. I should be very grateful if you could let me know any of your own.

(It may be added for completeness' sake that there was an equal freedom from peptic ulcer in the inhabitants of the Warsaw Ghetto, already quoted,[3] in whom any food consumed was of the same type as that referred to above.)

To sum up these statements, it is clear that the freedom from peptic ulceration in these prisoners of war in Russia, on a diet consisting essentially of unrefined carbohydrates and practically nothing else, ties up perfectly with exactly the same freedom in the prisoners of war in Japan, at the other end of Asia, the only difference in the latter case being that the unrefined grain was mainly rice. A more perfect correlation, on the present argument, could not be asked for.

Peptic Ulcer in the United Kingdom during the 1939–45 War.—After the somewhat dramatic behaviour of the incidence of peptic ulcer in enemy-occupied Europe, the behaviour of the incidence in the United Kingdom during the same period seems a little tame; nevertheless it is sufficiently informative to merit some scrutiny.

This behaviour differs in two respects from that already described:—

1. The increase in the incidence in women and old people, and of gastric ulcer as opposed to duodenal ulcer, which was so evident on the Continent, did not materialize.

2. A conspicuous rise occurred in the number of acute perforations, in certain big cities, at the time of the earlier air raids in 1940 and 1941.

It will now be necessary to see how these differences may be explained by the argument presented in this book:—

1. The reason, on this argument, why the increase in the incidence in women and old people, and of gastric ulcer as opposed to duodenal ulcer, did not occur in this country is very clear indeed. There was no starvation anywhere in the United Kingdom, and apart from the flour, the food was no coarser than it was in peace-time. No one was driven to fill himself up with vegetables, and even the flour, which became of 85 per cent extraction rate, was not as coarse as that in any true wholemeal loaf sold in a baker's shop to-day. Nor was the fuel situation ever serious enough to interfere with the proper cooking of vegetables and other foods, or with the cooking of bread. It is, therefore, not surprising that the specific changes in the ulcer incidence seen on the Continent, and already explained in some detail, never appeared in the United Kingdom.

2. The rise in the number of acute perforations in certain cities at the time of the air raids is brought out in *Fig.* 5, which shows the course of perforations in Glasgow over a long period of

time.[17, 18] Similar rises took place in London, Liverpool, Bristol, and no doubt other cities during the period of air-raids.[19]

The view enjoying such wide currency to-day, that the human body is so imperfectly evolved that it cannot endure stress without the risk of developing a peptic ulcer, finds much support from the big rises in the number of perforations that occurred in London

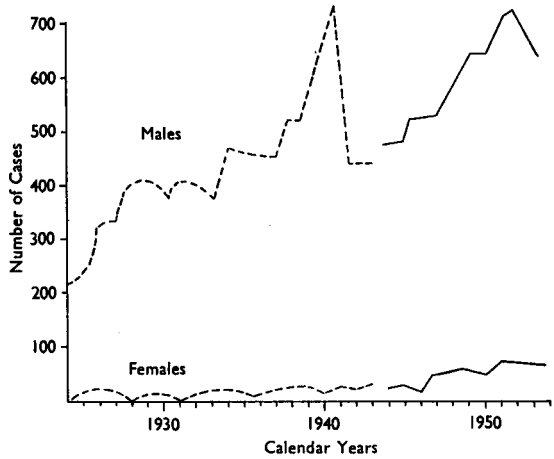

Fig. 5.—Number of perforated peptic ulcers occurring in the west of Scotland, 1924–53. (*Reproduced by kind permission of the 'British Medical Journal'.*)

and certain other cities at the time of these raids, but it is weakened by the fact that in the case of Glasgow the rise *preceded* the raids, and is still further weakened by the fact that, in spite of continued raids, the number of perforations later fell mysteriously, so that from 1942 onwards the number was below the pre-war figure and stayed so for the rest of the war, as may be seen on the chart. The stress explanation, however, is finally refuted by the fact that in the far more terrible air-raids on Tokyo, Osaka, and other Japanese cities, no perforations whatever occurred amongst the thousands of prisoners of war exposed to these raids, as described earlier in this work.

On the other hand, according to the present argument, which altogether excludes stress as playing any part of importance in this rise in the number of perforations, every feature in the rise can be related to the consumption of refined carbohydrates. Thus, during the raids people were routed from their firesides or their

beds and stuck for long periods in tube stations, air-raid shelters, and other places of refuge—and what food would be more likely to be consumed in such circumstances than ready-to-hand packages of biscuits, sweets, and other refined foods, together with cups of sweetened tea and coffee of negligible protein content? And furthermore, such foods would mostly be *consumed by themselves*, the importance of which, in the present argument, has already been stressed.

It has also been shown, on this argument, that acute perforations depend chiefly on the consumption of refined *sugar*, since this is robbed of all its protein, whereas refined grains (and manioc tubers) are robbed of only a part of their protein, and therefore the consumption of these latter is more likely to lead to a chronic sclerosing form of ulceration. Therefore, the prominence of *perforated* ulcers during these air-raids, accompanied as the raids were by the consumption of many sweetmeats, would be exactly what would be expected from this line of reasoning.

It is also easy to understand, on the same basis, why in the case of Glasgow the rise in the number of perforations *anticipated* the raids. For if it was not fright (stress), but the consumption of ready-to-hand processed carbohydrates, that was responsible for the perforations, then the *preparations* for the raids, including fire-watching and other defence duties, rendered so necessary by the examples of the raids on other towns (which preceded those on Glasgow), could well be understood as initiating the rise now under consideration.

The mysterious fall in the number of perforations later is likewise easily explained, for the 85 per cent extraction loaf became compulsory at the beginning of 1942, and the gradual fall in the availability of chocolate and sweets culminated in the institution of strict rationing in these by mid-1942. The disappearance of the compulsory 85 per cent extraction loaf, and of the rationing of chocolate and sweets, likewise explains the rapid rise again in the number of perforations after the war, which may also be seen on the chart.

A reference by G. Petren[20] that has been much quoted describes an equal rise in the number of perforations in neutral Sweden at this time. If this rise were a reality, it would be a considerable obstacle to the simple explanation of the rise that occurred in the United Kingdom, as set out above. The author has therefore been at some pains to investigate the rise in question,

and in this matter has been greatly helped by the kind action of Professor E. Ask-Upmark of the Royal University of Uppsala, who has procured for him comprehensive sets of figures for the perforations in leading Swedish hospitals over a long series of years. These figures, which are reproduced as an average in Appendix D, show quite clearly that no significance whatever can be attached to the rise in perforations that was described by Petren in Sweden at the time. Later figures show the rise to have been only a fluctuation, and Professor Ask-Upmark agrees about this.

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CHAPTER XIII

Heredity and Peptic Ulcer

IN order to appreciate the part played by heredity in the causation of peptic ulcer, it is desirable first to examine certain allied situations which are of help in this connexion.

As a start, consider the infantry assaults against enemy entrenchments in the First World War of 1914–18. In that war it was found, as would be expected, that during these assaults tall men were shot down by machine gunners considerably more often than short men were. Yet no one looking at a tall man killed in this way would have considered the death due to a structural defect in the body, since in natural circumstances the tall build might have been an advantage, not a disadvantage. No: the basic cause of death clearly lay in the new environmental factor of fast-travelling bullets, to which no adaptation could yet exist and to which this man's build made him especially vulnerable.

Consider, again, the case of hallux valgus. In this condition a hundred pairs of feet can go into shoes that are too pointed, and yet only ten of them, say, will develop the hallux valgus deformity. These latter feet are not built wrongly. Far from it: unconfined by shoes they might, in fact, be particularly efficient in certain natural circumstances, such as on long marches or during jumping from one piece of rocky ground to another. But the build of these feet, though perhaps particularly helpful in the foregoing circumstances, happens also to make them particularly vulnerable to the new environmental factor of pointed shoes—so new a factor that even to-day only a small fraction of humanity goes about in shoes at all, let alone in pointed ones. And it is this new factor in the environment, to which no adaptation can yet exist, and to which the build of these feet renders them especially vulnerable, that is clearly the basic cause of hallux valgus.

These considerations show the enormous distinction that exists between a disease due to a structural defect in the body and one due to an unnatural, that is to say new, factor in the environment, to which a person's build renders him especially vulnerable. The distinction is not only enormous in magnitude—it is also enormous

in importance. For it decides the whole approach to the treatment of a disease. If the basic cause lies in a structural defect in the body, the treatment can only consist of operative repair or the provision of some form of replacement therapy. But if the basic cause lies in an unnatural factor in the environment, to which a person's build renders him especially vulnerable, then the treatment is directed at once to the removal of that factor. The two treatments are clearly poles apart. In the case of peptic ulcer, for example, the contrast is not less than between an almost inevitable removal of the stomach, either anatomically or physiologically (vagotomy), and (on the present argument) a careful avoidance of certain processed foods.

There is seldom any trouble in deciding whether a given disease is due to a defect in the body or to an external factor in the environment. Quite apart from the date of onset, and many clinical characteristics (not one of which, in the case of peptic ulcer, suggests a congenital defect), there is the vital question of frequency of occurrence. It has already been seen in Chapter I that no congenital defect occurs to a greater extent than five times in 1000 persons. If, therefore, a disease occurs to a greater extent than this, and especially if it occurs to nearly twenty times this extent, as in the case of peptic ulcer, then clearly the basic cause does not lie in the body itself, but in the external environment.

Against this background the influence of heredity in the causation of peptic ulcer is seen to be of little or no practical importance, for all that is in question is not the inheritance of a bodily defect, but merely the inheritance of a particular build, that makes a person vulnerable to an external environmental cause. The important thing is clearly the external cause itself, and the best means of avoiding it, if that be possible. Thus, it does not alter the situation one iota if the tall man mentioned above had had a tall father, who was killed in the same way in an earlier war; or if the persons who develop hallux valgus sometimes have a parent with a similar build of feet, which, confined in the same type of shoes, have also developed hallux valgus; or, finally, if patients with peptic ulcer sometimes have a parent of similar build, including the stomach itself, governing the acid it secretes, who on the same type of food has also developed the disease. Clearly the only important thing in all these cases is the avoidance like the plague of the external cause itself, if that be at all possible.

For this reason it is waste of time to discuss hereditary influences in peptic ulceration here, or, in the author's opinion, anywhere else. What help is it to know that a person subject to this disease is liable to have a stomach that secretes much acid, let alone that he tends to belong to a certain blood group, or for that matter, to have ears with well-marked lobes? In fact, when it is seen that peptic ulceration is not due to a bodily defect, but to an external factor in the environment, to which a person's build renders him vulnerable, it not only becomes a waste of time to discuss the details in build responsible for this vulnerability—it also becomes a case of drawing a red herring across the trail, which distracts the attention from the one thing that does matter: the external factor itself.

To sum up, let it be granted that peptic ulcer patients do have certain hereditary characteristics, and that these may be seen in many parts of their bodies, ranging from their general build right down to their blood groups, and including the stomach itself. Yes, let this be granted, but let it also be admitted on the other side that these characteristics are merely those of personal build and in no sense constitute a bodily defect. Then the part played by heredity can be left severely alone and the attention directed to the unnatural cause in the environment, to which the build of these people makes them especially vulnerable, as fast and as completely as possible.

CHAPTER XIV

Application of the Present Argument to the Prevention and Arrest of Peptic Ulcer

Part I

PREVENTION

THE natural principles in the prevention and arrest of peptic ulceration, based on the argument presented in this book, are extremely simple in theory, and extremely simple in practice, too, if a person is living in his own home. Though it will be shown that they are neglected in the present medical and surgical approach to the disease, they are seldom in conflict with this approach, and therefore their value, which the author considers to be supremely great, is capable of being utilized at once. They apply equally to gastric ulcer and to duodenal ulcer.

Briefly, these principles may be generalized in one master rule, that natural instincts should be allowed to play on natural foods. This may be shortened to 'Natural instincts on natural foods'. The 'natural instincts' are essentially those involved in the choice of meal patterns and foods eaten, and will be referred to as the appetite. The 'natural foods' comprise all foods, whether raw or cooked, as long as they have not been subjected to refining processes.

It is, in general, just as simple as that, but neither half of the rule is of value in the absence of the other. Applying the appetite to refined foods achieves nothing; and eating natural foods without regard to the appetite achieves very little. The master rule must be applied *in both its components.*

The Appetite.—To particularize in the application of the above rule, a person should first of all be extremely careful never to eat anything unless he desires it, avoiding all arbitrary meals and arbitrary food mixtures, as described earlier. If his mind is on other matters, or if the food does not appeal to him, for heaven's sake let him leave it alone; he will make up for it fast enough a little later, when circumstances are favourable—practically no one ever *voluntarily* eats too little.

There is no need to be alarmed at the stomach being empty. It is forgotten that for a long period of time the treatment of peptic ulcer was frequently based on resting the stomach in just this way. First introduced by Leube[1] in 1875, this treatment, which included rest in bed, commenced with a fast of three to seven days' duration, and produced such satisfactory results that it was extensively practised for nearly fifty years. The treatment then swung to the opposite extreme, in which the stomach was never allowed to remain empty, if it could be avoided, because of the physiological belief that the resting gastric juice is the most acid. Never for one moment during these extremes of treatment, as far as the author has been able to discover, has the principle ever been laid down that it is not dangerous to eat if the appetite indicates it, but that it is dangerous to eat if the appetite does not indicate it.

In deciding by the appetite whether to eat at all, and in deciding what to eat, a person should make the decision away from the presence of food. If he tastes food first, for example, the instinct has not been applied correctly and the decision is much less reliable.

In following the appetite, a person will combine foods in any way he pleases, as long as they belong to the natural group described below. He will not follow the extraordinary advice, fortunately rarely heard to-day, not to eat carbohydrates and protein at the same meal, which, for example, would for ever divorce an egg from a slice of bread and butter, and so thwart Nature's desire for a balanced meal. Nor will he refrain from drinking with meals, if he wishes to drink, which is another remarkable restraint sometimes advised.

Similarly, a person will eat at his natural speed. If this speed is fast and his teeth are good, let the eating be fast and the natural rhythm be preserved. But note the crucial difference between eating fast in this way and eating in a hurry. If the speed is not a natural speed, but is imposed on the individual by external circumstances, the result will be disastrous. The inadequate mastication will lead to the food being insufficiently impregnated with saliva, and through not being finely subdivided, the food will not encounter the gastric juice adequately, and hence on both counts a fall in the buffering of the acid will occur.

Natural Foods.—These comprise all foods that are either still in the raw state or have merely been heated up from that state

by cooking (to which procedure it will shortly be shown that man is now well adapted). Over these natural foods a person should never allow anyone to dictate to him which ones he should eat or not eat, whether they be meat foods, vegetarian foods, or any other sort of food; the choice must be his and his alone. However, foods that have been altered from the raw state by more than cooking, as happens in refining processes, can no longer be regarded as natural, since the alterations produced in them are so recent that no adaptation worth the name can yet have taken place to them. These refining processes lead to increased concentration in food, which deceives the tongue, and the whole argument for a free choice falls to the ground. For example, a person wishing to eat something sweet in this country commonly buys a bar of chocolate, weighing a minimum of 2 oz. This contains as many calories as there are in 2 lb. of apples, which are a natural sweet food. After eating two or three apples out of, say, eight, the ordinary person would feel he had had enough. But how many would stop three-eighths of the way along the bar of chocolate? *The concentration has deceived the instinct.*

For practical purposes the unnatural foods mostly consist of the refined carbohydrates discussed throughout this work, and fall into two groups:—

1. Foods containing white flour (or other processed cereal, such as polished rice), and all the pastry, cakes, biscuits, macaroni, and other foods made from white flour.

2. Foods consisting of the ordinary white or brown sugar sold in grocers' shops, or containing it, as in chocolate, sweets, ices, and confectionery. These latter are usually taken by themselves and are particularly dangerous.

The ideal to be pursued with unnatural foods is their substitution by the natural parent ones. As regards the substitution for white flour products, this is perfectly practicable, because the natural parent food is freely available, but as regards the substitution for refined sugar, this is by no means so practicable, because the natural parent foods are not freely available; therefore, a special form of sugar consumption will be suggested here, to meet the situation.

1. Taking first the case of white flour, a true wholemeal flour should be substituted. This includes the substitution of a guaranteed wholemeal loaf for a white loaf. A true wholemeal flour can always be ordered quite easily, but a guaranteed wholemeal loaf

is by no means easy to come by, since few brown loaves are true wholemeal loaves. In some localities the only way to obtain decent bread may be to bake it in the home, a remarkably easy procedure if the method which avoids kneading is used.[2] In the case of breakfast foods, care should be taken to take those made from wholemeal flour.

It is very important that bread should not be eaten until it is adequately stale. Horder, Dodds, and Moran[3] have shown that new bread is not sufficiently chewed "and forms pasty lumps that resist, both *in vivo* and *in vitro*, penetration by gastric and other digestive juices". One of the most important of these other digestive juices is the saliva in the mouth itself, a natural buffer of the gastric acid. New bread, even if wholemeal, is dangerous stuff and should be avoided. (The staling process should take place with the bread wrapped in a cloth, not enclosed in a tin (which fosters mildew), and should be decided not by the days that have passed, but by the dryness that has resulted in the bread.)

It may be noted here that it is above all in Eastern countries, like India, Indonesia, and Japan, that scope exists for the application of the present approach to the prevention and arrest of peptic ulceration. In these countries, owing to the simplicity of the food structure in the poor (who provide most of the sufferers from the disease), the substitution of natural foods for unnatural ones can be accomplished almost at a stroke by the restoration of the bran to the rice. This restoration can be effected by one of two methods. The first and obvious one is to stop the milling of the rice. The objections to this are (*a*), that it reduces the keeping properties of the rice, as previously explained; and (*b*), that it forces the consumption of unmilled rice on everyone, which, though a most excellent thing, is sure to cause general opposition amongst the population, as the Japanese and Indian governments have already discovered in their efforts to stamp out beri-beri. The second method is to stir in a prescribed amount of rice bran to the rice just before it is eaten (so that any loss of protein during boiling can be eliminated). This amount would approach 50 per cent of the weight of the rice before being cooked, in the case of a 70 per cent extraction rice, and 25 per cent in the case of an 80 per cent extraction rice. The 'advantage' of this method is that the addition need only be carried out by actual sufferers. Whichever method were used, the change-over would

have to be achieved slowly, so that the adaptation kept ahead of flatulence and other symptoms—in short, the addition should on no account be attempted at a single stroke, but effected painlessly in quarter-amounts over several weeks. This is a vital point, or the patient might not co-operate. Also, his appetite for the mixture might at first be upset, with further unfavourable repercussions.

To the restoration of the bran to the rice should be added the careful avoidance of throwing away the water in which the rice has been boiled, which contains leached-out protein, as previously explained. It appears that this water is thrown away all too often, and the importance of avoiding it is seen in the communication that was quoted from Orissa, India (p. 46).

Since in the case of tapioca (manioc) consumption it is impossible to avoid the basic processing treatment, for the reason already set out, it appears that the only practicable step may lie in the addition of rice bran to this food also. No other step appears economically possible, since the eaters of manioc are the poorest of all the peoples of the world. As it seems to be a universal rule that no one will protect his health unless he is actually being hurt, presumably this step also would be carried out only by actual sufferers.

2. Turning now to the case of refined sugar, a natural substitution for this is really most difficult. The parent sugar-cane and sugar-beet are not available; raw fruit, which is so desirable, is apt to be expensive; and there are special reasons, given in Appendix E, why such material as honey is just as dangerous as sugar itself. The author has found by long experience that part of the solution lies in *a supported* sugar consumption, which will now be explained in detail.

Many persons find that they like to eat something sweet *all the way along a meal.* Many famous food combinations depend on this fact, such as roast duck or roast pork with sweet apple sauce; roast lamb, jugged hare, and even roast turkey with sweet red-currant jelly or cranberry jelly; roast lamb with sweet mint sauce; and so on. Similarly, many people, when eating a meal of fish or egg, like to take a cup of sweet tea or coffee at the same time, and a final example is the eating of marmalade and other preserves with bread and butter. This tendency to take something sweet all the way along a meal is probably universal (in people who like any sweet things at all), if only it is encouraged

to reveal itself, and it represents an effort by the body to ensure a balanced meal.

In the writer's opinion this tendency should be utilized to the hilt in the present problem, so that refined sugar is taken only when supported by a collection of simple, unprocessed foods, just as in the above combinations, *and, what is of cardinal importance, only in the smallest amounts compatible with pleasure in the meal.* This will remove the menace of consumption of unsupported sugar, whether taken in isolation, as in the case of sweetmeats and sweet drinks, where the danger to the stomach is very great, or as a sweet course at the end of a meal, where the danger is still considerable. Exactly the same precaution is necessary in the taking of alcoholic drinks (which, especially in the case of beer and other malted liquors, often contain considerable quantities of sugar as well); the protein present in these is negligible and it is obvious that they cannot safely be taken in an unsupported manner. The same is true of soups containing *meat extracts*, and of hot drinks made with these substances; and also of coffee. All these substances produce a large flow of gastric acid, but neutralize none of it.

Concomitantly with the above there should be achieved as big a substitution for refined sugar by raw and dried fruit (such as raisins and currants) as the purse permits. It is no use preaching a gospel of elimination—it is too unpleasant to have any chance of being followed. The gospel to preach is one of substitution, which has a good chance of being followed, if the person is not too poorly off. For substitution leads to little loss of pleasure and may lead to a gain. Thus, a junket or a milky rice pudding, which is sweetened not by sugar but by a banana or two eaten at the same time, is improved in taste, not depreciated. It follows that though advice to eliminate chocolate and sweets has little chance of success, it may be otherwise if their substitution is advised by grapes, apples, and other fruit in season, which can often be bought quite cheaply in the shops. Unlike sweetmeats, these raw fruits can, of course, always be taken quite safely by themselves; dried fruits and certain foreign fruits, however, require some care in this respect (*see* Appendix E).

To the above substitutions should be added that of unpeeled and unsliced boiled potatoes for potatoes normally both peeled and sliced. This will prevent the loss of some 10 per cent of the protein calories present, as explained earlier, to say nothing of amino-acids.

Additional Notes

Cooking.—It is important at this point to raise the question of any dangerous alteration in food from its natural state by cooking, and here the law of adaptation is again of great value.

At the beginning of this work it was stressed that in evolutionary adaptation the time factor is of supreme importance, and that the danger of any new feature in the environment should be assessed on this basis, i.e., by how long it has been there. In this connexion it does not seem to be sufficiently realized that, although ancient man occupies only the Quaternary period of animal existence, yet he extends over a temporal span of nearly a million years, and Le Gros Clark[4] has shown that for about half this vast epoch he has been sufficiently evolved to be able to light a fire and heat food over it. This epoch is between one and two hundred times the length of recorded history, which it reduces to a thing of but yesterday. Compare with this the short history of the refining of flour and sugar, which first appeared only a few thousand years ago and became properly established only a few hundred years ago.

The law of adaptation, therefore, indicates very little danger in cooking, as compared with that in refining processes. Nevertheless, certain precautions are very desirable. Over-cooked meat, for example, presents to the gastric juice a mass of highly coagulated and even charred protein, so that combination with it becomes difficult. The contrast in this respect provided by underdone, soft, red meat, that can be chewed to a semi-liquid, is very obvious. The advantage of stewed foods from this point of view is also self-evident.

On the other hand, the disadvantages of fried foods have already been pointed out, though these become minimal for people whose natural liking for fat is high, and the author would not castigate such foods out of hand, as long as they do not exhibit overcooking (which frequently happens, e.g., in chipped potatoes). It is known, incidentally, that if the same fat is not used over and over again very little chemical change is produced in it during ordinary frying;[5] the author would, therefore, recommend a natural fat in frying, especially fresh beef dripping, used once only. It may be added that some of the weird imported fats seen in the country to-day are *not* natural to people of our nationality,[6] and some of the synthetic ones are not natural to those of any nationality.

Arbitrary Food Mixtures.—The reference just made to cooking in fat, which results in an arbitrary food mixture, brings up the whole question of these mixtures.

Such mixtures often consist of sweetmeats, like ice-cream, chocolate, and many other sweets. These contain large quantities of refined sugar, and are seriously deficient in fibre, which influences mastication and impregnation with saliva on the one hand, and the gastric emptying-time on the other. They should for these reasons be carefully avoided, especially as they are likely to be consumed by themselves. Other arbitrary mixtures comprise puddings, including Yorkshire pudding, cakes, and many proprietary foods purporting to build the person up. Such mixtures should all be replaced by simple, natural foods, combined by the person to suit his own taste. For example, cake, which contains white flour, sugar, fat, and eggs, should be replaced by a meal of wholemeal bread, butter, eggs, and, if insisted on, some tea sweetened with sugar, drunk concurrently. By this means the person will be able to decide for himself what he needs in the way of these food materials, instead of having the matter settled for him by somebody else, and usually with refined, and even adulterated, foods at that. Arbitrary food mixtures can all be easily substituted for, and the whole lot should be avoided. (A currant bread is mentioned in Appendix E.) The substitution also saves a great deal of trouble in cooking. In fact, the whole of the present approach is one of simplification and saving of trouble.

Period of Cautious Adjustment Essential.—In a younger person with sound teeth the transition from refined to unrefined carbohydrates is attended by minimal disturbances and can be achieved at once, but it must not be imagined that this would be true in an older person, still less in an old one. Great care is required in some people.

The need for this will be apparent if a parallel case is examined. Parodontal disease, or pyorrhœa, as it is often called, is a typical disease of modern civilization, caused by the soft, pappy nature of refined carbohydrates. As E. W. Fish[7] puts it so well, such a diet does not provide sufficient friction to keep the gum margins hard and keratinized, any more than clerical work gives a person the hands of a bricklayer. If coarse wholemeal bread replaced the present soft white bread, and fruits such as the apple replaced the sticky sweets consumed everywhere to-day, there would be no parodontal disease or wholesale loss of teeth from this cause.

However, this is not to say that a person with parodontal disease established could effect the transition from refined to unrefined carbohydrates over-night. If it were not for the protective pain experienced, such a sudden change would in places shift the gums right off the teeth. No: the transition can only be achieved slowly. For example, in the case of the apple just mentioned, this will have to be peeled and sliced up for some time before a whole unpeeled apple can be tackled.

It is exactly the same as regards the digestion. Even the change from white bread to true wholemeal bread causes a commotion for a week or two. Over the necessity of making the transition from refined to unrefined carbohydrates there should never be a moment's hesitation, but the transition itself must be made very cautiously, the attention being focused on the bite. The teeth must be carefully examined in older people, and if necessary the bite improved by dental treatment. Until this improvement is achieved, there should be no hesitation in crushing up natural foods before eating them, and crusts of bread and skins of hard fruit may have to be omitted altogether.

Here an essential point must be remembered. These particularly coarse elements in natural foods are to be guarded against by those with a deficient bite, not because they inflict mechanical injury on the membranes of the stomach, any more than they do on those of the mouth, but because the poor mastication prevents their adequate impregnation with saliva, and also results in such poor subdivision that afterwards there is imperfect combination between them and the gastric juice, with consequent loss of buffering power on each count. This difference in reasoning engenders an equal difference in attitude towards these coarse elements. Those elements that are not meant under natural conditions to be removed, either by the hands, such as the peel of an orange, or by the tongue, such as the core and pips of an apple, are meant to be eaten. And they should be eaten. They protect the teeth from caries and parodontal disease, and because many of them, such as the skin of the apple and potato, are rich in protein, they protect the stomach membranes from acid injury. In the prevention of peptic ulceration these coarse elements are therefore *to be sought after*, and not avoided, as is done in a wholesale manner to-day in the prevention of the disease. It is only in those with deficient teeth that they should be treated with circumspection for the time being.

To sum up, over the necessity of making the transition from refined to unrefined carbohydrates, in the prevention of peptic ulceration, there should never be the least doubt, but a sensible person will achieve this transition with great caution and not rush at it like a half-wit.

Feasibility of following the Present Approach.—It will be seen from what has been written that if a person is living in his own home, and therefore able to arrange his meals as he wants them, it is as easy as it is pleasant to follow the natural dietetic approach outlined above. There is nothing complicated or difficult about this approach; all the steps involved are ones of simplification, especially in the kitchen. Furthermore, although 'to live medically is to live miserably', there is nothing miserable about living naturally; quite the contrary. It is clear that the approach costs a little more money, and that is its only disadvantage. Most people would think good health worth the extra cost.

However, if anyone is not living in his own home, the approach, very easy in theory, becomes very difficult in practice. In such circumstances it can only be followed if the health is highly valued, so that the necessary amount of trouble is taken to maintain it. This may involve steps ranging from the preparation of sandwiches to the deferment of meals until later. The present book, unfortunately, cannot help in this respect. It can only endeavour to present the truth, and must leave to the individual person the practical application of that truth. One thing is certain: if the will be present, the way can always be found.

The Orthodox Diet in Ulcer Prevention.—It is at this stage highly constructive to examine the orthodox dietetic approach to the prevention of peptic ulcer in this country. The author has before him the relevant diet charts from many of the largest hospitals in England and Scotland, which reveal a remarkable degree of unanimity and give a good idea of the advice being tendered at the present time. Assuming the patient is up and about again, in his own home, the following points may be noted in the charts supplied:—

1. The appetite is never considered in any of them. A minimum of six meals a day is recommended, with something taken during the night if the patient wakes up. One sentence recurs constantly: "Have small meals frequently, so that the acid in the stomach has 'something to work on'. Take something every 2 to 3 hours."

2. There is no hint of any warning against the taking of foods that have lost much, or all, of their original protein buffering power. An exception is the advice to avoid soups and drinks containing meat extracts, though this appears to be based on the acid-stimulating properties of these extracts rather than on their lost buffering power. Sugar, sieved jams, honey, treacle, chocolate, and sweets are not only allowed, but are actually recommended in many of the charts, often as snacks by themselves between meals. Similarly, white bread and other foods containing white flour are recommended axiomatically.

3. Behind all these charts, in fact, is clearly the guiding idea of coarse elements in natural foods inflicting mechanical injury on the stomach membranes, instead of, as in the present argument, these elements inflicting injury only in the presence of a deficient bite, and then never a mechanical injury but an acid injury. Instead, therefore, of recommending these coarse protein-rich elements in natural foods, with reservations based on the bite, these instructions recommend their wholesale avoidance. Wholemeal bread, shredded wheat (a wholemeal breakfast cereal), salads, many vegetables, raw fruit, nuts, and other similar foods, are all to be avoided—the very foods that in the present argument form the spearhead of the preventive attack. A perfect example is provided by oatmeal. Even in Scotland it is recommended that this be avoided unless it is a fine variety, or the bran is strained out, whereas in the present argument oatmeal is only allowed if it is a coarse variety, or, in the case of a fine variety, if, say, a tablespoonful of bran is added to each plateful.

4. The taking of arbitrary meals has already been shown to be characteristic of these instructions, but so also is the taking of arbitrary food mixtures—especially proprietary ones, the mention of which abounds in them.

In short, excluding at this stage any reference to the dietetic treatment of active ulceration, it may be said that if it were desired to draw up a set of preventive instructions directly contrary to natural, that is to say, evolutionary principles, these instructions could hardly be bettered. And what do they achieve? As far as can be discovered, absolutely nothing. This is not only the author's opinion, but also a great many other people's. Well-known clinical studies,[8–10] with some patients on a typical ulcer diet and others on an ordinary diet, have not shown any difference in results, which from the argument presented in this

book is exactly what would be expected. The matter may be summed up in the words of G. D. Hadley:[11] "During remissions ulcer patients can and should eat practically anything they like . . . a melancholy cuisine of steamed fish, sieved vegetables, and tapioca pudding should be imposed on nobody, unless there are good grounds for supposing that it will avert relapses. These grounds are totally lacking, and present dietary régimes of this kind are obsolete and should be discarded."

And according to natural principles, that is exactly what should be done with them, so that the decks can be cleared for the institution of a preventive approach that is logical instead of completely illogical.

Part II

Arrest of Active Peptic Ulceration

Treatment of an active peptic ulcer does not lie within the scope of this work to anything like the extent that preventive treatment does. One reason for this is that the orthodox diet on these occasions will nearly always already be based on the consumption of milk and other high-protein foods, and the only important contribution that the present approach can make is the principle that pain due to free acid in the stomach should be controlled, not by the taking of unwanted food, but by the taking of a suitable antacid drug, which will of course be prescribed by the medical attendant.

Another reason why the treatment of active ulceration does not lie greatly within the scope of this work is that once a scar of any magnitude has resulted in the stomach or duodenum, the whole situation becomes radically changed as regards prevention. The blood-supply to the overlying tissue is now impaired and the recuperative power of the tissue is impaired with it. The prevention of further ulceration may therefore be quite impossible in actual practice, and consequently surgical treatment may become obligatory. All that can be said is that early attention to preventive principles may save many a stomach that would otherwise be lost; further than that it is clearly not possible to go here.

It will be noticed that no reference has been made to smoking in relation to peptic ulceration. This is because it has been shown that smoking is not an important factor in the causation of the

disease.[12] However, as it has also been shown that smoking has some influence on the rate of healing in established ulcers, clearly the less there is of this practice the better. The author himself, however, would always regard this particular element in the prevention and treatment as of little importance compared with the fundamental ones already set out.

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CHAPTER XV

Relationship of Peptic Ulcer to Other Diseases

EVERY year 30,000 human stomachs are removed in operating theatres in the United Kingdom.[1] The effect of this operation on the main symptoms of peptic ulcer, such as pain, hæmorrhage, and perforation, is remarkably successful, and no one deploring that such a step should ever become necessary (as needless to say it frequently does become) could fail at the same time to have the profoundest admiration for the skill and design shown in these operations, perfected with such great endeavour over so many years.

Nevertheless, the operation of gastrectomy carries certain recognized liabilities. These mostly stem from the loss of energy which inevitably follows the withdrawal of the gastric attack on the valuable protein component in the food. The patient, in fact, resembles to some extent a bird that has lost a handful of feathers from one wing. The manifestations of this loss of food material range from a fall in the sexual power to the contracting of pulmonary tuberculosis. There is no indication for discussing these liabilities here, but the author wishes to refer to another liability of the operation, which is never mentioned: the non-removal of the cause of the original ulcer. The patient may well have moved into calmer waters as regards his stomachic symptoms, but the original cause of the trouble continues silently to operate, and in consequence other diseases may supervene in the future.

For the author has no doubt whatever that peptic ulceration is only one expression of a much more extensive pathological condition, which may be termed the 'saccharine disease'. This disease stems solely from the refining of carbohydrates, which refining causes its effects partly through loss of protein, partly through loss of fibre, and partly through overconsumption of sugar, either directly or via the digestion of starch. The term 'saccharine disease' is suggested because the greatest degree of refining takes place in connexion with *sugar*, but clearly the most accurate term would be 'refined-carbohydrate disease'.*

The saccharine disease is considered here to include the following main conditions, but needless to say they will very seldom all

* Needless to say, the word *saccharine* has nothing to do here with the chemical, *saccharin*.

occur in the same patient. Which manifestation or manifestations occur in any given person depends on the particular build in each of his systems, which governs the vulnerability of each system to this new environmental factor. The conditions are listed in groups, according to the causative mechanism:—

Peptic ulcer Acne rosacea	From loss of protein
Dental decay and parodontal disease Varicose veins and hæmorrhoids	From loss of fibre
Obesity Diabetes Appendicitis, cholecystitis, and other *Bact. coli* conditions	From over-consumption of sugar

The saccharine disease includes a good many other conditions, too, especially amongst skin diseases, but as they are less important, they will not be discussed here.

It might be objected that it would be a coincidence if all these conditions were due to one and the same cause, and that the above list shows a loss of perspective. Such an objection does not bear a moment's scrutiny. Are not the manifestations of any disease in medicine always described in terms of systems? Is it a coincidence that typhoid fever, for example, can manifest itself in nearly every system in the body? Or that syphilis can do the same? How many systemic manifestations are described in Addison's anæmia and in diabetes? How many diseases, in fact, are ever confined to one system? Far from it being a coincidence that the saccharine disease can manifest itself in many different systems, it would be a coincidence if it were not able to do so.

There is no intention to discuss here the individual conditions given in the above list, except in the most cursory manner. *Peptic ulcer*, in this connexion, need not be further discussed. As regards *acne rosacea*, the author is convinced that this condition arises mainly from the effect of either relatively stagnant gastric acid, or relatively unbuffered gastric acid, or both, on the reflex arc which influences the dilatation of the vessels of the face; he has never seen a case of rosacea yet that did not respond to the exact dietetic approach recommended here for peptic ulcer.

As regards *dental caries* and *parodontal disease*, the cause of these is accepted everywhere as lying chiefly in the loss of fibre that takes place during the refining of carbohydrates, which results on

the one hand in fermentable accumulations of food around the teeth, and on the other in a softening of the gum margins, leading to pocket formation. The author has tried to show in his book on varicose veins[2] that this loss of fibre, through the stasis it causes in the colon, resulting in pressure extramurally on the iliac veins and intramurally on the rectal veins, is also responsible for the production of *varicose veins* and *hæmorrhoids*.

In a proposed further evolutionary study on the saccharine disease in general, and diabetes in particular, the present author hopes to show that neither in man nor beast does consumption of unrefined carbohydrates ever result in *obesity*, the cause of which lies essentially in the refining of this class of foodstuff. Refining processes lead to concentration, and it has already been shown that this concentration results in a deception of the instinct of appetite, and hence in over-consumption. With unrefined, unconcentrated carbohydrates over-consumption does not occur and obesity does not occur either. In the author's opinion present-day views that obesity is due to the instinct of appetite being at fault, or to insufficient exercise being taken, are fundamentally incorrect. A note on this subject is given in Appendix F. It is hoped to show that a similar line of reasoning applies in the case of *diabetes*. The decline in the incidence of this disease during the sugar-rationing of the last war, and its virtual absence in primitive peoples who consume little or no sugar, support this contention. A note on this subject is given in Appendix G. It is likewise contended that *appendicitis* and *cholecystitis*, which show similar characteristics in incidence to the foregoing, as referred to in Appendix H, arise solely from alterations in the intestinal bacterial picture, produced by the unnaturally large amounts of sugar arising in the gut, through the consumption of these same refined carbohydrates.

From the foregoing arguments some clinical association between these various conditions would be expected. This is, indeed, frequently to be seen, the simplest example in the present disease being the constipation that notoriously accompanies duodenal ulcer, but the matter is a complicated one, since ill health can impair the gastric secretion and so reduce the incidence of concomitant peptic ulceration; and in diabetes a neuritic impairment of the gastric secretion is also possible, as was suggested earlier in the case of beri-beri.

This digression must cease. Suffice it to say that there exists evidence that peptic ulceration is part of a much more extensive disease than one confined only to the stomach and duodenum—

a disease termed here the 'saccharine disease'. It follows that any treatment of one of the manifestations of this disease, if it does not remove the cause, leaves the patient exposed to the supervention of one or more of the other manifestations.

A good example of this argument is seen in the injection treatment of early hæmorrhoids, which, so easy in its accomplishment and so successful in its local effect, may ultimately involve the patient in disaster. For if these early hæmorrhoids were left alone, their presence could lead to the patient being forced to make a simple but crucial change in his diet. The substitution of unrefined for refined carbohydrates would not only correct the causative stasis in the rectum and so ensure the quiescence and eventual disappearance of the hæmorrhoids themselves, but would also have important repercussions in other parts of the body. Manifestations of the saccharine disease in the teeth and gums would be brought to a halt, so that the patient perhaps retained the use of his teeth to an advanced age. Obesity would be prevented. And so on right down the list of these manifestations, and with certain added implications as regards cancer of the rectum, which is also probably related to stasis in this part of the colon. But remove these hæmorrhoids by injection, leaving the cause to continue operating, and the supervention of one or more of the other manifestations is only too probable. In more advanced cases of hæmorrhoids, surgical measures may of course become obligatory, as with peptic ulcer, but that is not the present point.

The above argument would be considerably weakened if, with the injection treatment of hæmorrhoids, there were instituted the essential change in the diet that ensured the removal of the cause, though the taking away from the patient of a valuable danger signal might still remain an objection to this form of treatment at this stage. But in actual practice how often is the situation carefully explained to the patient and any preventive treatment (apart from the shocking use of aperients) ever really instituted? It would seem only too rarely.

And this example reveals, in the final analysis, the most serious liability of all that may be attendant on the operation of gastrectomy, and the importance of the recognition of the saccharine disease itself.

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CHAPTER XVI

Summary and Conclusion

THIS work on the causation of peptic ulcer is based partly on a chain of reasoning, which utilizes the Darwinian theory of evolution and a certain number of well-recognized physiological and biochemical facts in digestion, and partly on an accumulation of evidence on the incidence of the disease, in many countries, in peace and war, which supports this reasoning. This approach indicates the loss of protein that takes place during the refining of carbohydrate foods as the basic cause of peptic ulceration, and excludes stress as playing any important part in the causation.

The approach also leads to almost exactly the opposite dietetic advice over the prevention and arrest of the disease to that being proffered at the present time, and since the results of the latter advice are so bad, there is some hope that those of the former may be correspondingly good. The results of the treatment when imposed by force in prisoner-of-war camps in the last war also give reason for hope in this respect. The approach is above all a safe approach, since it advises no steps over eating habits or foods that are not being taken by the rest of creation.

To amplify these paragraphs under appropriate headings:—

The Argument.—As regards the chain of reasoning, or argument, this is of stark simplicity and makes no demands on the credulity of anyone. It may be crudely generalized as follows:—

1. Peptic ulceration is contingent on the presence of the hydrochloric acid in the gastric juice, and does not occur in the absence of it.

2. The only component in the food that buffers this acid is the component which the acid is designed to digest—the protein one.

3. Consequently any removal of this protein component would be exactly calculated to cause an abnormal and dangerous activity in the acid itself, such as might result in peptic ulceration.

4. Removal of the protein component in food does in fact take place during the refining of carbohydrates, partially in the case of cereals and certain tubers, and totally in the case of sugar-beet and sugar-cane.

5. Due to these 'protein-stripped' foods being frequently consumed by themselves, the danger of peptic ulceration attendant on their consumption is greatly increased. Also, because layering in the gastric contents largely retains the parts of a meal in the order in which they were eaten, the danger from these foods, even when they are not taken by themselves but in one part of a meal only, is still considerable.

6. It follows that not only does a high protein consumption in one meal not compensate for a low or absent protein consumption in another, but that this is even true of different parts of the same meal. Therefore, the total protein in the diet is no true guide to the danger that may be present in this respect.

7. The taking of food in the absence of desire for it, frequently seen in civilized countries to-day, leads to a well-recognized delay in the emptying of the stomach, which prolongs the time during which the stomach may be exposed to insufficiently buffered acid, as described above, with consequent increase in the danger from it. The only way in which stress plays any part in the production of peptic ulceration is through interference with the desire for food, on the assumption that the fall in desire is not then matched by an equal fall in consumption.

The Evidence.—As regards the evidence collected, this reveals a remarkably close correlation in many countries, both in civilized and uncivilized communities, and both in peace and war, between the incidence of peptic ulceration on the one hand and the removal of protein in the refining of carbohydrate foods on the other. It is submitted that it would be an extraordinary coincidence if the single basic cause, indicated so strongly by the biological facts, should be supported by this very complicated pattern of incidence, if it were not, indeed, the true cause. Concerning the evidence itself, the author is confident that, as in the older type of house, built of stone, although a sizeable fragment here and there may well become dislodged, the mass of the structure will still hold it strongly together.

Results of the Present Approach in the Treatment of Peptic Ulceration.—The author has long established to his own satisfaction that provided the disease is not too advanced, or the personal situation one where the indicated régime cannot be followed, the results of the present approach in the prevention and arrest of dyspepsia in general, and of peptic ulceration in particular, are *excellent*, assuming the intelligent co-operation of

the patient. However, the author would prefer that his own experience with this approach should be completely discounted, and that instead the attention should be directed to the results of the actual application of these principles by *force majeure* in certain of the prison camps in the last war. It is exceedingly important that where the diet in these camps consisted largely of refined carbohydrate, peptic ulcer became a plague, but that where the diet consisted of unrefined carbohydrate, not only did new cases of peptic ulcer not appear, but established cases cleared up. The sections on the Japanese and Russian prisoner-of-war camps bring this point out very clearly.

It has been shown many times (e.g., in connexion with the teeth) that the average man in this country, though prepared to take endless trouble over the servicing and maintenance of his motor-car, will do practically nothing over any comparable attention to his own body. The images of the Buddha show this god apparently lost in contemplation of his abdomen. And, indeed, the miracle of the digestion of food, that constantly takes place therein, is well worthy of some contemplation. Yet the average man will spend less than five minutes in his whole life thinking about this part of his anatomy, or its functions, unless he is suffering pain in it. It follows that the present approach will almost certainly be of help only to those already afflicted with dyspeptic troubles, but to these it is hoped it will be very helpful indeed.

It is tragic that there exists such enormous apathy amongst the general public over the preservation of health. If this were not the case, the important implications of the conception of a saccharine disease, as put forward in this work, in the prevention of many dangerous conditions, could be utilized with great advantage.

Above all, the Safety of the Present Approach.—The dietetic steps indicated in the present approach are pre-eminently *safe* steps, comprising as they do the careful attention to the instinct of appetite and the careful avoidance of an artificially refined class of foodstuff—in short the restitution of natural behaviour in a natural environment. These steps do not seek any clever escape from this restitution. In this connexion there cannot be mistrusted too strongly any medical advice based on physiological arguments which are in conflict with natural, that is to say, evolutionary, indications, such as the advice to eat when

there is no desire to eat. The physiological arguments underlying advice of this sort will be rejected by all thinking persons, and the advice itself regarded as very dangerous. To those sponsoring such physiological conceptions of the working of the human body well might Nature herself be imagined as addressing the words of Emerson:—

> They know not well the subtle ways
> I keep, and pass, and turn again.

There is, indeed, encountered in England to-day far too much of this "science falsely so called", described long ago,[1] in which basic natural instincts are brutally disregarded and alterations to natural foods freely condoned. In an even earlier period Horace warned people that "though they could drive out Nature with a pitchfork, yet she would ever hurry back, to triumph in stealth over their foolish contempt".[2] Does every generation have to be crucified on this cross? Will the lesson never be learnt? If this book has any merit at all, it is that it does not countenance trying to be clever at Nature's expense.

REFERENCES

1. 1 Timothy vi. 20.
2. "Naturam expellas furca, tamen usque recurret, et mala perrumpet furtim fastidia victrix" (Horace, *Epistles*, Book 1, X. 24).

APPENDIX A

I am obliged to Mr. F. I. Tovey for permission to reproduce here his recent work at the University of Liverpool on the buffering powers of certain processed and unprocessed grains on the gastric juice. The procedure carried out was as follows:—

1. Millet, white flour, wholemeal flour, rice bran, and wheat bran were steamed for 1 hour.
2. Rice (polished) was cooked for 20 minutes with a minimal quantity of water, which disappeared into the rice.
3. The above were dried completely for 36 hours in a hot air oven and 2 g. of each were weighed out.
4. Distilled water, 10 ml., was added to each and left for 18 hours to soak through.
5. Filtered gastric juice, 9 ml., was added to each and the *p*H readings recorded.
6. The specimens were then kept at 37° C. for 1 hour and the *p*H was again recorded.
7. As a control, 9 ml. of gastric juice was added to 10 ml. of distilled water and the *p*H recorded before and after incubation.

(The *p*H of the gastric juice undiluted was 1·76.)

The following are the results of the investigation:—

	*p*H *Readings*	
	Immediate	*After 1 hr. at 37° C.*
Millet	2·85	3·63
Rice (polished)	2·45	2·67
White flour	2·46	3·52
Brown flour	3·18	4·32
Rice bran	4·18	5·60
Wheat bran	3·82	5·35
Control	2·37	2·12

APPENDIX B

Those who consider that small alterations in the natural environment can be ignored should study carefully a disaster like that of acute bloat in cattle. In this condition a great accumulation of uneructated gas occurs in the fourth stomach or rumen of ruminants, especially cattle. It is a common and serious problem in animal husbandry, and carries a high mortality (15–100 per cent).[1] The cause of acute bloat lies in an insufficient amount of fibre in the diet, usually due to allowing animals to graze on grass having too high a proportion of clover, which contains relatively little fibre. This lack of fibre leads to insufficient stimulation of the stomach musculature, and so accumulating gas is not eructated. The rumen becomes, therefore, distended to a dangerous degree, and the animal frequently dies.

Grass and clover are both natural foods for cattle, but in a natural environment grass would never have such a high proportion of clover; this is achieved in farming to-day by ploughing up the land and sowing a suitable seed mixture. It must always be a source of amazement—and of instruction—that so large and powerful an animal as an ox can rapidly be killed by such a trifling alteration in its natural environment. When it is remembered that to-day mankind commonly alters its natural environment by many times this extent, in removing some 30 per cent of its substance from his grain and 90 per cent from his sugar-beet and sugar-cane, it must engender profound scepticism in any thinking person that he can be a party to these practices and get away with them. In particular, the greatest reserve should be exercised in dismissing any known effects of such practices on stomach function just because the effects appear to be small.

REFERENCE

1. MENZIES, D. W. (1952), *Bloat and the Bloat Survey*. British Veterinary Association Publication No. 23. London: British Veterinary Association.

APPENDIX C

Ice-cream.—Ice-cream is at once a refined carbohydrate food and an arbitrary food mixture. In this product the 21·1 per cent of protein calories in milk have been reduced to 8·6 per cent by the addition of sugar. This sugar, being refined sugar, lacks the counterbalancing fibre which, in nature, would influence mastication and impregnation with saliva on the one hand, and the gastric emptying-time on the other. Furthermore, 25 per cent of all the ices sold in this country are water-ices, in which protein has disappeared altogether. It is clear, therefore, that the consumption of these products is to be deprecated as strongly as that of raw fruit, the natural alternative to them, is to be extolled.

The following figures show the enormous rise in the consumption of ices that has taken place in the United Kingdom during the present century. The figures, which are approximate and show the consumption in thousands of gallons, have been derived from Board of Trade reports and personal communications:—

1920	1930	1938	1951	1960
Under 1000	8000	36,000	40,000	50,000

APPENDIX D

Average number of peptic ulcer perforations per 1000 surgical admissions to the University Hospital, Uppsala, the University Hospital of Lund, and Sahlgren's Hospital, Gothenburg, Sweden, during the years specified:—

1939	4·0	1947	3·8
1940	5·4	1948	5·1
1941	3·5	1949	4·1
1942	5·2	1950	5·1
1943	4·0	1951	2·8
1944	2·6	1952	4·4
1945	5·0	1953	3·9
1946	4·2	1954	4·7

From figures supplied to the author in 1961 through the kindness of Professor Erik Ask-Upmark, of the Royal University of Uppsala, Sweden.

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APPENDIX E

Honey; Dates.—At first sight, honey, and certain fruits such as dates, would appear to be natural foods. A deeper look at these foods, however, considerably modifies this view. Until very recent evolutionary times honey, in fact, has very seldom been available at all. It is true that ever since the discovery of fire mankind has in theory been capable of smoking out the nests of wild bees, and there is the archæological evidence that he occasionally did this. A little reflection, however, will show that such a feat must have been an extremely difficult one to bring off, and in any case would have yielded so little reward as seldom to have been worth the effort. To the consumption of honey, which contains practically no other food material than sugar, man is therefore little more adapted than he is to that of sugar itself, as sold in grocers' shops, and anyone arguing that honey is a natural food, which he can eat *ad libitum*, is in for some nasty surprises. Solomon may not always have been wise, but he certainly showed his wisdom in advising people to keep off much honey (Proverbs xxv. 27). In short, honey should be regarded for practical purposes as being as dangerous as sugar, and should therefore be consumed as sparingly. This applies in all the manifestations of the saccharine disease and not only in the case of peptic ulcer.

As regards dates, these certainly are a natural food—but not for the white races! In the date the sugar present is enormously concentrated. Thus, if the percentage of sugar present in the apple is about 10 per cent and in the banana 20 per cent, in the date it is 64 per cent. The argument used in connexion with chocolate on page 115 of the text applies to some extent to the date, too. It would, of course, be an entirely different matter if the white races were adapted to the consumption of this subtropical fruit, but they are not. If, therefore, an inhabitant of this country, imagining that dates are a natural food for him, eats half a box or more of them by themselves, he may, like many other people, experience a little later a scalding acidity at the back of the throat that is long remembered. The same applies to certain dried fruits if consumed by themselves, such as raisins, which have a comparable sugar content to that of dates, and in fact the

banana, with a content of 20 per cent, seems to indicate the comfortable limit in this respect for most white people. Needless to say, the circumstances become very much altered if these concentrated fruits are diluted by other natural foods, as are currants in a wholemeal currant loaf. Then they become highly desirable, since they lead to the avoidance of materials containing refined sugar, which is much more concentrated still.

This subject has merely been appended here as an example of the application of evolutionary principles in clinical and dietetic practice.

APPENDIX F

Obesity.—The view that in this condition the appetite is at fault can easily be assailed. There is first of all the general evidence. The holders of this view are really contending that the human body is often wrongly built, i.e., imperfectly evolved, in this respect. In that case they are caught on the hook of the incidence figures. Once again it may be stated that no known congenital abnormality occurs more often than five times in one thousand births. How often does obesity occur? Besides, if this condition were a congenital abnormality, the age of onset would be all wrong.

Then there is the specific evidence. A glance at any wild creature in its natural environment shows that no matter how plentiful its food supply, it never eats too much of it. Even a poulterer's shop reveals that no wild rabbit ever ate too much grass, no woodpigeon ever ate too much wheat, and no herring ever ate too much plankton. No wild creature, in fact, is ever overweight. The forces of evolution have ensured that in nature organisms react to an abundant food supply never by developing a disease, such as obesity, but by raising the rate at which they propagate themselves. This is true for the whole of creation, from a lactic acid bacillus in a bottle of milk right up to man himself in circumstances of plenty.

For as regards man, it is to be noted that primitive communities, living under natural conditions, show no evidence of obesity whatever. Yet these communities are probably existing on an almost purely carbohydrate diet, in the form of unrefined grain and various tubers. It is only when these carbohydrates become refined and therefore concentrated, so that the instinct of appetite is deceived, that the condition starts up. These points will be elaborated in the future study, mentioned in the text.

The view that obesity is due to insufficient exercise is just as vulnerable. In the first place, the lack of exercise held responsible is nearly always a *voluntary* lack, not an imposed lack. People are blamed for using lifts in office buildings and for not walking when they leave their offices. Yet this is what their natural inclinations often tell them to do. Some don't *want* to climb stairs, and they don't *want* to go walking, either; they would rather sit down when

they get home and put their feet up. Therefore, those who ascribe obesity to these instincts being wrong are just as much in error as when they ascribe it to the instinct of appetite being wrong, and are open to the same critical attack. Throughout the whole animal kingdom, in fact, no living creature, unless forced to do so in order to get food, ever takes any more exercise than it wants to take. Nature obviously likes to conserve the heart—and certainly never inflicts on any organism the penalty of obesity for 'laziness'.

Even in cases of *imposed* lack of exercise obesity does not occur as long as the food is not tampered with. A visit to any zoo will show this quickly enough. Here will be seen the two opposite poles of creation—a large animal like a tiger, accustomed to hunt its prey over many square miles of jungle, and now confined to a space measured in cubic feet; and a small bird, like a finch, accustomed to fly about many acres of countryside and now confined to a space measured in cubic inches. In each case the natural exercise has been enormously reduced. Yet, just because each of these creatures continues to take its food in its natural form, in the one case raw meat and bones, and in the other case unaltered seeds, the weight remains the same and obesity does not occur.

An exception to this appears to exist in the fattening up of animals and birds for market. But a closer examination will nearly always reveal in these circumstances either that food concentrates have been given, in which case the whole argument instantly alters, or that the animal or bird is of a kind that has been evolved by selective breeding over many centuries to become much fatter than its wild counterpart. For example, in the Peking (Aylesbury) duck the bird's muscles have been so replaced by fat that it is never able to fly off the ground. Finally, what is under discussion in humanity is not an imposed lack of exercise of this sort, but a voluntary one. No one in this country is prevented from taking exercise if he wants to. The point is that he often doesn't want to, and this discussion then applies.

It is therefore contended that the *sole* cause of obesity lies in the consumption of refined carbohydrates. A large appetite is not a cause, and a dislike of exercise is not a cause. It is perfectly true that restraint of appetite or enforcement of exercise will reduce obesity, but as long as the true cause continues to operate—the consumption of refined carbohydrates—the use of either is an example of two wrongs not making a right. To be sure, these two factors are valuable in the removal of surplus weight already

in existence, but in the basic matter of prevention the mind should be riveted on the essential cause and not confused by irrelevancies.

Equally, it should be thoroughly realized that unrefined carbohydrates, such as wholemeal bread, potatoes eaten in their skins, raw fruit, etc., are *no more fattening than any other natural food*, including protein foods and fatty foods. Some starvation for established excess of weight is clearly indicated, but a distortion of the personal choice of the various classes of foodstuffs is emphatically *not*. Moreover, those who, for example, counsel the substitution of fats for carbohydrates, thereby upsetting the natural ratios of these in the diet of a person, are courting other and possibly greater dangers of interference with evolutionary principles, not the least of which may lie in the domain of coronary disease.

APPENDIX G

Diabetes.—Of all the conditions included in this work under the term 'saccharine disease', none, in the author's opinion, is more obviously related to the consumption of refined carbohydrates, especially sugar, than is diabetes itself. Amongst many pieces of evidence supporting this view are the two mentioned in the text—that the incidence of diabetes declined in all the belligerent countries during the sugar-rationing in the last war; and also that, as the author will elaborate in the future study referred to, the disease is virtually absent in primitive communities which consume no refined sugar. However, before these two pieces of evidence can be used effectively, the following objections to them must be met.

As regards the decline in the incidence of diabetes during the war, the food shortage at that time caused a big fall in the consumption of proteins and fats and a corresponding rise in the consumption of carbohydrates. It is true that these latter were eaten relatively unrefined, mainly in the form of grain and potatoes and including little sugar, but since all the starch in carbohydrates is itself digested to, and absorbed as, sugar (glucose), it is clear that during the war the ultimate absorption of sugar in the belligerent peoples was higher than ever. How, therefore, can diabetes be related to the consumption of refined carbohydrates, especially sugar ? It would seem on these grounds more reasonable to relate the disease to the consumption of protein or fat, the fall in the consumption of either then being in harmony with the fall in the incidence of diabetes, too. Such a relationship has, indeed, been attempted, especially as regards the consumption of fat (e.g., by Himsworth[1]).

As regards the virtual absence of diabetes in primitive communities, these communities are usually ones that live almost entirely on carbohydrates, such as various grains and tubers. It is true that the carbohydrates are eaten substantially unrefined, as in the form of whole maize or millet, or home-pounded rice, but again, since all the starch contained in these is digested to, and absorbed as, sugar, it is clear that the ultimate sugar absorption in such communities, which do not eat refined carbohydrates, is higher than in civilized peoples, who do.

The best way to expose the fallacy in these arguments is to consider another condition altogether—dental caries. It is accepted everywhere, as already stated in the text, that this condition is related to the consumption of refined carbohydrates, especially sugar. During the last world war, for example, a big fall in the incidence of caries occurred in all the belligerent countries, *pari passu* with the fall in the consumption of these refined products; also, in communities all over the world caries is slight or absent until these products appear amongst them, a recent striking example of this being provided in the inhabitants of Tristan da Cunha.

This overwhelming epidemiological evidence quite rightly ignores the fact that during the war the total carbohydrate consumption in the belligerent countries went up, and that amongst communities which get little or no caries the total carbohydrate consumption is nearly always much higher than amongst civilized peoples who get a great deal of it. For what matters in the production of caries is not the quantity of carbohydrates consumed, but the form in which they are consumed—to be more accurate, whether they have been refined or not. A whole cartload of carbohydrates in the form of raw apples or sugar-beet, for example, would do the teeth nothing but good, whereas a few cases of refined, sweet biscuits, leaving a sticky residue round the teeth, could initiate the fermentative processes responsible for decay and therefore do the teeth great harm.

Exactly the same argument, though involving a different pathogenic mechanism, is applicable to the causation of diabetes. Here also what is concerned, as the author has pointed out elsewhere,[2] is not the quantity of carbohydrates consumed, but the form in which they are consumed, i.e., whether they have been refined or not. The simplest way to relate the disease to this refinement is to assume that what strains the pancreas is what strains any other piece of apparatus—not so much the total amount of work it is called upon to do, but the rate at which it is called upon to do it. In the case of eating potatoes, for example, the conversion of the starch into sugar, and the absorption of this sugar into the bloodstream, is a slow and gentle process, very different from the abrupt and violent one that follows the eating of a piece of Brighton Rock, for example, or other mass of concentrated sugar.

It is thus easy to understand that in the belligerent peoples during the war, as in primitive communities to-day, the stresses

imposed on the pancreas, in spite of a large consumption of unrefined carbohydrates, were much less than in Westernized peoples at the present time. These, taking some 5 oz. of refined sugar per head per day, can consume in a matter of minutes material that, in its natural form, would normally be eaten over several hours.

It is seen, therefore, that the two objections noted above can be met.

It may be pointed out, in connexion with this chain of reasoning, that in the discussion on page 15, of the various ways in which reduction in the protein buffer could result in acid-injury to the membranes of the stomach and duodenum, there was included the possibility of the reduction allowing the rise in acidity to occur too *quickly*. Such a mechanism would be in close harmony with the one just delineated in the case of diabetes.

Finally, to make obesity the cause of diabetes shows, in the author's opinion, a serious confusion in reasoning. It is not that obesity causes diabetes, but that the consumption of refined carbohydrates is the cause of both. The two conditions are therefore often associated, but, as with other manifestations of the saccharine disease, may also occur singly. In any case, the basic dietetic prevention and treatment of the one will always apply also to the other, since in each case this consists in the removal of the cause —the consumption of refined carbohydrates.

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APPENDIX H

Appendicitis, Cholecystitis, and other Bacterium coli Conditions.—The rarity of these conditions amongst primitive communities, consuming no refined carbohydrates, again offers strong support to the view that all of them are manifestations of the saccharine disease. This differential incidence will be elaborated in detail in the work planned for publication later, but, indeed, there exists weighty evidence on this point already. For example, as regards appendicitis: "The disease is common in highly civilized countries and urban communities, but rare in remote rural districts and among primitive peoples. During the nine years that McCarrison practised amongst the hill tribes of the Himalayas he never saw a case of appendicitis. Natives who live on a diet abundant in cellulose are immune from the disease, but when they adopt the diet of civilization they lose that immunity."[1] The rarity of cholecystitis and gall-stones amongst primitive communities has been equally well established. This is especially true for Africa,[2] and also to some extent for Northern India.[3]

In the author's opinion these conditions are due, not to the intestinal stasis that follows the removal of cellulose in the refining of carbohydrates, but to the over-consumption that follows such removal. This surplus of food to the body's needs is not wholly digested and absorbed, and forms a food supply for overwhelming numbers of bacteria in the intestine. It is very striking that, in the treatment of *Bact. coli* pyelitis, the administration of aperients, presumably through the stirring-up of the intestinal contents allowing a greater number of bacilli to gain access to the blood-stream, is very apt to make things considerably worse, whereas changing the carbohydrates consumed back to their natural form, thereby much reducing their total consumption, starves the bacilli out and nearly always produces excellent results. There is thus considerable clinical support for the view that all these *Bact. coli* conditions are manifestations of the saccharine disease.

The relationship between appendicitis and the consumption of refined sugar has been pointed out before. Thus, as long ago as 1938, A. Brown,[4] in Southern Rhodesia, advanced such a

relationship on epidemiological, clinical, and historical grounds. As regards the last, the main rise in the incidence of the disease in the United Kingdom occurred between 1880 and 1900, during which time the consumption of sugar rose from 60 lb. per head of population per year to nearly 100 lb.

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