DYSPEPSIA IN MEDICAL PRACTICE

By

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INTRODUCTION.

There is, perhaps, no branch of Medicine a knowledge of which is of more importance to the general practitioner than that of diseases of the gastro-intestinal tract. One is not long in practice before the prevalence of gastric pain and discomfort is brought home to one. The symptoms are frequently of so indefinite a character that it is a temptation to adopt an empirical form of treatment rather than investigate the functioning of the gastric secretion by the test meal and stomach tube. For some years I was content to try the effect of gastric sedatives empirically, and, if the symptoms persisted, to advise an X-ray examination. Quite often this latter procedure yielded a negative result, whereas a test meal carried out at the beginning may have definitely indicated a rational line of therapeutic and dietetic treatment.

In this paper I propose to discuss the various forms of dysfunction of the stomach dealing particularly with so-called Functional types of Dyspepsia, outlining the method I adopt in arriving at a diagnosis and the treatment I have found most suitable. As a student and later as a resident I rarely came across cases of functional dyspepsia largely because these cases were not of the type for hospital admission, and in ordinary text-books of Medicine little information was forthcoming. As Alvarez[1] says, "More than half of the persons who go to a physician for advice in regard to chronic indigestion have symp-

toms which appear to be largely "functional" in nature. That the field is a neglected one in medical schools should be evident to anyone who has attended seminars, lectures, or clinics." This is an opinion with which I agree.

Before proceeding further it may be as well to define the word "Dyspepsia" as by some writers it has been limited in its meaning to refer to functional gastric diseases only. I prefer, however, with Hutchison,(2) to regard it as meaning discomfort of any kind arising during the process of digestion as the result either of organic disease of the stomach or of a primary disorder of its functions.

The type of practice where my cases are taken from is industrial, and this, of necessity, has had some bearing on the manner of carrying out the investigations and also on methods of treatment. Economic considerations in many cases have precluded private X-ray examinations but where these have been deemed necessary sufficient data have been obtained through hospital facilities. Another general point, not without interest I think, is that since the great increase in unemployment of recent years there appears to have been a relative increase in cases of functional dyspepsia whereas no such increase has been noted in regard to organic gastric disease: this will be referred to later.

It is of course of very great importance to be able to discriminate between organic and functional disturbance and sometimes the difficulties in reaching an accurate diagnosis are

so great, even after utilising all modern resources, that the problem even at the end cannot be settled with certainty. Further, it is now well recognised that the gastric functions may vary within wide limits not only in different individuals, but also in the same individual in different circumstances. The ingenious experimental work of Pavlov[3] and Cannon[4] bears testimony to this. Hence to place undue importance on test meal findings would be inadvisable, but when taken in conjunction with a careful history they are of considerable value.

While the more modern Fractional Test of the gastric contents has greatly increased the interest of gastric analysis, in general practice the older Ewald test-breakfast yields useful results and has the considerable advantage of saving much time. In the routine examination of gastric cases, therefore, I have relied on the latter method, reserving the Fractional method for cases where a fuller investigation seemed advisable.

While realising the necessity of making a thorough examination before making a diagnosis of a functional disorder I feel Hurst[5] is too severe in his assertion that indigestion which persists for more than a few days is generally due to organic disease, the only common exception being the dyspepsia due to fatigue. Actually the analysis of gastric contents has proved to be of little direct value in the study of "nervous dyspepsia", because one finds all varieties of results in cases which present similar clinical symptoms and frequently those persons who complain most have normal concentrations of acid,

while some of the apparently normal students who have volunteered to have their stomachs examined by the tube are found to have no hydrochloric acid at all, as in Bennett and Ryle's[6] well-known series of one hundred normal cases four of which were found to have constitutional achylia gastrica. But in spite of this I feel that unless such a test has been carried out we are not in a position to say how the gastric glands are functioning. Besides, in the diagnosis of early Addison's ("pernicious") anaemia and gastric carcinoma gastric analysis may be most helpful. As Hurst[7] says the presence of free hydrochloric acid in any fraction of a test-meal is a decisive point against the diagnosis of Addison's anaemia.

Before concluding these general remarks, I venture to state that while the subject of Dyspepsia primarily concerns the stomach, there is one particular condition of the intestines of which I feel some special notice should be taken, viz., Constipation, as I constantly find in practice that this symptom is often almost an obsession with the patient, and in any case its treatment has an important bearing on the general treatment of dyspeptic conditions whether organic or otherwise. I propose, therefore, later to discuss this common symptom in relation to dyspepsia, and particularly its treatment apart from drugs.

The classification of the different forms of dyspepsia I have found it most convenient to adopt is as follows:-

I. FUNCTIONAL DYSPEPSIAS:

(a) Motor neuroses.

(b) Secretory neuroses.

(c) Sensory neuroses.

(d) Dyspepsia of infancy and childhood due to faulty diet.

II. ORGANIC DYSPEPSIAS:

(A) Gastric in origin, (gastritis, ulcer, and carcinoma).

(B) Extra-gastric in origin, (reflex dyspepsia, associated dyspepsia, and simulated dyspepsia).

Before considering these conditions in detail I think it would be advisable to state the routine procedure I adopt with my gastric cases, and this I propose to do in the next section.

PROCEDURE ADOPTED BY THE WRITER FOR INVESTIGATING PATIENTS WITH DYSPEPTIC SYMPTOMS.

Probably there is no field of practice in which it is more important for the physician to take a complete inventory, so to speak, of the patient, physically, mentally, and emotionally, than in gastro-enterology. Indeed, there are few branches of medical science in which symptoms are so frequently encountered which are in reality the result of disorders far removed from the abdominal cavity. The examination of the patient, therefore, I arbitrarily divide into three main parts, viz., (1) General Examination, (2) Physicial Examination, (3) Special Examination, which involves examination by special methods, analysis of gastric contents, examination of faeces, X-rays, and examination of blood and urine.

In order to avoid missing points in the detailed investigation involved in the above classification, it has always seemed to me desirable to have a systematic routine suitable for all cases. For this purpose I have had a chart printed, a copy of which is enclosed. It is a modification of one suggested by Morgan.[8] It enables a record sufficiently complete to be of real utility to be kept on one folded sheet. The two blank pages I use for notes made at subsequent examinations. Any special papers such as hospital reports are inserted between the pages if it is desired to keep them, but usually I incorporate such details on the chart itself. The charts are filed alpha-

NAME ADDRESS

DATE AGE OCCUPATION

FAMILY HISTORY

PAST HISTORY

HABITS HAS LOST, GAINED, STATIONARY WEIGHT LBS. IN MONTHS

PRESENT ILLNESS DURATION

MOST TROUBLESOME SYMPTOMS

APPETITE THIRST TASTE PYROSIS

DEGLUTITION FEEL BETTER AFTER FOOD BLOATED FEELING OF FULLNESS SENSE OF WEIGHT

REGURGITATION IS IT ACID BITTER BELCH RUMINATION HEARTBURN

PAINS—GASTRIC

NAUSEA VOMITING JAUNDICED ABLE TO REST ON EITHER SIDE SLEEP

BOWELS

NUMBER OF STOOLS DAILY CHARACTER BLOOD MUCUS

FLATULENCE BORBORYGMI PILES

PAINS—INTESTINAL

HEADACHE DIZZINESS BREATH

WEIGHT HEIGHT GUMS AND TEETH

TONGUE

SKIN

CONJUNCTIVA SCLERA CAPILLARY CIRCULATION VESSELS PULSE

REFLEXES

LUNGS

HEART

LIVER

GALLBLADDER

KIDNEYS

ABDOMEN

SPLEEN

GASTRIC SECRETIONS DATE AMOUNT FOOD PARTICLES

MUCUS BLOOD TISSUE

BILE COLOUR ODOUR

FREE HCL. COMBINED TOTAL ACIDITY

OCCULT BLOOD LACTIC ACID

B.P.

BLOOD EXAM. HAEMOGLOBIN RED WHITE URINE

FAECES

WASSERMANN

X-RAY

RECTAL EXAM.

REMARKS

DIAGNOSIS

TREATMENT

RESULT

betically so that ready reference is assured.

It may now be convenient to discuss the three main divisions of the examination seriatim:

1. GENERAL EXAMINATION:

History. It would be difficult, I think, to over-estimate the importance of obtaining a complete history from the patient. Indeed, in certain cases it may be quite possible to arrive at a tentative diagnosis which later proves correct, simply as a result of obtaining a full history from an intelligent patient. I always endeavour to avoid putting leading questions, and, unless the patient is unduly loquacious, I prefer to let him discuss his case with me in the first place, as completely as possible, without any interference on my part. This is, of course, at times a tedious procedure, but usually it is well worth it. Later, especially where some functional derangement is suspected, it is usually necessary to elicit information regarding the patient's daily habits and to enquire tactfully into his mental and emotional life.

Pain. This is a relative term, as what to one patient may be mere discomfort is to another a more severe symptom, hence I am reluctant to rely too much on this as a definite factor in the history without careful interrogation. The onset with regard to meals, frequency of occurrence, localization, duration, and, (if present), radiation are all points of importance. The question of the possible reflex origin of the pain must be considered

in the light of the later examination.

Vomiting. When the word "sickness" occurs in the patient's history of his symptoms, I find that this term is often used simply to connote nausea and not actual vomiting. Hence to avoid ambiguity I enquire whether food was actually brought up. As a symptom vomiting is important, and if present to a considerable extent it is usually more suggestive of organic disease - of intra- or extra-gastric origin - than of functional gastric disorder. But as Maclean[9] says, the most important thing to remember regarding vomiting is that it is but a further stage of nausea, and in many patients the presence of nausea is of as much significance as if vomiting had actually occurred. In the early diagnosis of acute appendicitis in particular I have proved this statement to be true many times.

It is, of course, advisable to inquire into the nature of any vomited material, the time of day when most frequently present, and whether any pain which may have been present has been relieved by the vomit. All these factors I find of considerable importance from an etiological and diagnostic point of view.

Wasting. To be of use this symptom should be definite - so many pounds lost in a certain time, and then, if there is no history of over-strict dieting to account for the loss of weight, any marked emaciation would tend to suggest the presence of organic disease.

Appetite. I find that sometimes when a patient tells me he is "off his food" on further interrogation it transpires that the taking of food produces pain, hence he is reluctant to take any, though in reality he is anxious to eat, showing a condition of pseudo-anorexia. Then again there is the patient who has a frequent desire to eat as he knows from experience it will ease the pain or discomfort from which he is suffering. In short, any abnormality as regards the appetite is noted as an aid to differential diagnosis.

Thirst. The presence or absence of thirst to an excessive degree is noted, as while it may not be of special significance as regards stomach disease it may be an indicative factor of some metabolic disorder which is indirectly causing gastric symptoms.

Taste. If this sensation is absent or diminished it may have an effect on the gastric secretion, since it is, of course, a recognized physiological fact that the rôle of the taste-buds situated at the base of the tongue is to stimulate the salivary and gastric glands. I realize that a full examination of the sense of taste is usually carried out where disease of the nervous system involving the sensory distribution to the tongue is suspected. In gastro-intestinal dysfunction, however, it is my practice merely to enquire of the patient whether he has noticed any alteration in his powers of tasting different foods.

Deglutition. If there is any suggestion of dysphagia this is noted, as it would effect a disturbance of digestion in

its initial phase. If there is difficulty in swallowing, the question whether it is associated with imperfect gustatory function causing a diminution of salivary secretion and its lubricating action or whether there is some nasal, pharyngeal or oesophageal abnormality, should be investigated when the physical examination is undertaken.

Abnormal sensations after meals. A sense of bloating or fulness in the epigastrium immediately or shortly after meals is a very common symptom in cases which have proved to be of functional rather than of organic gastric disorder. On the other hand, a sensation of sinking or emptiness in the epigastrium is fairly common and should be noted as I find in these cases gastroptosis is often a factor, though I quite agree with Hurst[5] that this condition may be present without producing symptoms.

Eructation. If mild this is of no particular significance. If severe it may be most distressing, and I find it of very common occurrence in cases where the dyspepsia is of "nervous" origin. In my cases it is most prevalent in females, and in one of the worst instances it commenced about fifteen minutes after meals and continued for twenty minutes to half an hour. Fermentation could not have occurred in such a short time, and the patient did not appear to be subject to aerophagy, though this may be a factor in some cases. Maclean[10] has suggested the condition may be due to a combination of gas secretion in the stomach with bad gas absorption.

Pyrosis. Heartburn is a common symptom and when present in excessive degree I used to hold the opinion that it was due to hyperacidity. The carrying out of the analysis of numerous test-meals, however, does not bear out this opinion, as I have often found the amount of free hydrochloric acid was not above normal in several cases where pyrosis was a definite symptom. It simply means the chyme is regurgitated from the stomach into the oesophagus and this produces a burning sensation in the gullet and epigastrium.

Regurgitation. If there is a history of part of a meal being returned into the mouth, without nausea, and then ejected, regurgitation is present. This is a particularly common symptom in the dyspepsia of infants and will be considered later. Rumination, where the food is swallowed again, usually after being re-masticated, is a much less common indication of gastric dysfunction.

Headache. Where this is complained of associated with vomiting I always endeavour to ascertain whether the headache preceded the vomiting, as in that case migraine or cerebral disease may be the cause of the gastric symptoms. I am not, of course, referring to the typical migraine where the hemicrania is a prominent symptom, but to those cases in which the headache is of minor intensity but where vomiting is a prominent symptom. These cases do present difficulty, but the periodicity of the attacks is a guide leading one to suspect an extra-gastric cause

of the disturbance. Some years ago I had a severe case of this type in a female, Mrs. H., aged 40. It was only after getting a history of previous attacks which cleared up after some hours, that I decided to adopt palliative measures. About two years later she developed a keratitis which was found to be specific in origin. The Wassermann reaction was strongly positive and she has been on anti-specific treatment since. In the light of this one might at first think the gastric attacks were in the nature of gastric crises, but although five years have elapsed she has not shown any other signs of tabes dorsalis. On the other hand, her vomiting attacks have been less in frequency and severity since she has been on anti-syphilitic treatment. The case appears to have been one of atypical migraine on a specific basis.

Vertigo. This may be present from a slight giddiness to a definitely staggering gait, and it may be the main symptom associated with gastric disturbance. In this type of case in particular the taking of the blood pressure may be very helpful in suggesting a possible diagnosis. In April 1932 I had a case of this type. The patient, a man, J.H., aged 55, a machinist, suddenly took ill at his work with vomiting and vertigo. The latter symptom was so pronounced that he could not walk home. Headache was also a feature. I saw him about an hour after the onset of the illness. The vomited material did not present any special features and there was no epigastric pain or tenderness. Having satisfied myself there was no sign of a gross cerebral lesion I

left him for a few hours. He slowly improved with rest in bed and occasional sips of water, the diet being gradually supplemented. Urine examination was negative. Blood pressure 240/120. Ophthalmoscopic examination showed arteriosclerotic changes in the retinal vessels. Wassermann reaction negative. Later he attended at the Hospital Dispensary where he again had a thorough examination, nothing being found beyond the hyperpiesia to account for his gastro-cerebral syndrome.

Bowel Movements. It is so common to find constipation complained of in the general interrogation of the patient, that as a symptom it is of little diagnostic significance. Indeed a slight degree of constipation does not appear to me to be of much importance. The idea of auto-intoxication is one of the oldest in Medicine, and one that has always had a strong hold on the public imagination, because it all seems so rational and obviously true. But where constipation is present the faeces are comparatively solid and dry, and so lacking in nutriment that most of the bacteria die. Little absorption can be expected from solid masses of faecal material as compared with what would occur from more liquid excreta which can come into more intimate contact with the intestinal mucosa. Hence I incline to the view of Alvarez[11] that the individual is safest from auto-intoxication when constipated. But, as with most things, moderation must be aimed at, and certain cases are so intractable that something must be done for them - this will be considered later under treatment.

Diarrhoea, on the other hand, is less common as a symptom and so is of more significance when present. The relation of the movements to the intake of food is noted and also any peculiarities in the appearance of the faeces which the patient may have noticed.

2. PHYSICAL EXAMINATION:

General Appearance. The height and weight are ascertained and their relationship to the physiological standard considered. Any peculiarity as regards appearance is observed, particular note being taken of the skin, such as undue pallor, blotchiness, or tendency to cachexia. Sometimes even a cursory inspection of the face gives an indication of probable gastric dysfunction, - the presence of acne rosacea being a case in point.

Oral Cavity. The tongue is always examined as a matter of routine, though the significance of the lingual surface being heavily coated is not usually regarded nowadays in the same way as it was years ago. It is not necessarily a sign of gastric disease or defective alimentary elimination when the patient's tongue is furred. I find it usual for such a patient to have a definitely preconceived idea that his "stomach" must be out of order because of this very obvious sign. But as Bennett[12] says, a fundamental error lies behind such a theory, in that the tongue and mouth differ radically from the rest of the alimentary

canal, as regards development, histological anatomy, and nerve supply. They are ectodermal in origin, like the skin of the face, and the tongue, like the skin of the face, sheds its superficial epithelium. I have often seen a very coated tongue in a patient with no symptoms of a gastric nature. Per contra, the tongue may be of normal appearance in the presence of more or less severe gastro-intestinal disease. By regarding a coated tongue as being probably due to dietetic error - insufficient roughage in the diet - treatment along this line may be helpful in removing the epithelial débris.

The teeth, gums, and pharynx are examined at the same time as the tongue is inspected. In the industrial district in which I practise the presence of pyorrhoea alveolaris is very commonly present in patients who consult me, apart altogether from gastro-intestinal symptoms. Indeed I have long been struck by the relative frequency of this disease and the apparent absence of pathological consequences. As an etiological factor in arthritis I am not concerned here, but when one considers the quantity of pus which must be swallowed by these patients, without producing dyspeptic symptoms, it seems to require some explanation. It is rational to suppose that the free hydrochloric acid in the normal gastric secretion speedily destroys the virulence of any organisms present. But where I find achlorhydria present associated with pyorrhoea it is my practice to advise immediate dental treatment, as the danger of infecting the intestines, coupled with the greater likelihood of toxins being absorbed, is ever present.

Respiratory System. Following upon a brief examination of the nose and nasopharynx the chest is examined by inspection, palpation, percussion, and auscultation. I place great importance on a careful chest examination, chiefly with a view to ascertaining the presence or otherwise of pulmonary tuberculosis. This is, of course, a well-known cause of apparent gastro-intestinal disease and I propose later to discuss this in connection with certain cases I have had.

Cardiovascular System. Here again, a careful examination may reveal the cause, or a contributing factor towards the cause, of the gastric symptoms. The heart is examined by the classical method as a matter of routine. I have already mentioned (p. 12) the importance of taking the systolic and diastolic blood pressure. It is my practice to use a Tycos sphygmomanometer, determining the pressures by the auscultatory method. Blood counts and the estimation of the percentage of haemoglobin are reserved for those cases which present an anaemic picture associated with dyspeptic symptoms.

The Abdomen. The abdomen is inspected, in the first place, with the patient in the upright position, as useful information may be obtained by this means. Notice is paid to the size of the subcostal angle, as when this is sharp the abdomen is correspondingly narrow, and I agree with Hutchison[13] that abdomens of this type are potential factors in the production of "functional" types of dyspepsia. Any undue swelling of the

abdomen is noted and correlated with the result of examination by palpation which is next carried out, the patient lying recumbent on a couch. I find it usually inadvisable to commence palpation over the site where the patient complains of pain, as by causing any increase of discomfort the patient is naturally "on guard" and some degree of muscular rigidity may be encountered which serves to obscure the findings. This voluntary rigidity is sometimes a manifestation of fear of pain, and once the confidence of the patient has been secured it is possible gradually to increase the pressure without obscuring the objective symptoms through voluntary muscular rigidity. Any hyperaesthesia of the skin is noted before commencing deeper palpation. Unless contra-indicated by being the site of the pain, it is my custom to commence palpation in the left iliac region, progressing up the left side of the abdomen to the left costal margin, then across the epigastrium to the right hypochondrium and then the right side of the abdomen to the right iliac region, finishing by palpating the hypogastric area. The position of the abdominal viscera is determined by bimanual palpation, the right hand performing the palpation.

Percussion and auscultation as applied to the abdomen yield results, in my view, of considerably less value from a diagnostic standpoint, than does palpation. While agreeing with Morgan[14] that no method which may aid in making a diagnosis or in throwing light on obscure conditions involving the gastro-intestinal canal should be completely ignored, I place greater reliance on results obtained by palpation than on

the findings elicited by percussion and auscultation. No doubt the succusion splash sometimes obtained over the epigastrium several hours after food has been taken would tend to suggest gastric dilatation with perhaps pyloric stenosis, but there are other means of determining this more accurately, viz., by X-rays and the stomach tube.

Urinary System. Apart from what is ascertained regarding the kidneys in the physical examination of the patient's abdomen, this system usually requires no further investigation beyond the ordinary qualitative urine tests, which comes under a section of the special examination.

Nervous System. A brief survey of the cranial nervous reflexes, particularly the light reflex, is made. The presence or absence of the knee jerks and the type of plantar reflex are noted. The condition of the abdominal reflexes may be conveniently ascertained when the abdomen is being examined.

Rectum and Anus. In a complete examination of the patient an inspection of the anus is made and a digital examination of the rectum carried out. I admit that where there is gross disease in this region it is usual for the patient to complain of some pain or discomfort referable to this site, but recently I had a case which has served to impress upon me the importance of making more routine rectal examinations. The patient, a male factory worker, Joseph H., aged 75, had for years been

troubled with epigastric discomfort and acid eructations. In spite of his age he had otherwise been in excellent health, and he continued at work till November, 1931. He then began to lose weight rapidly, the "indigestion", as he called it, became more evident to which the symptom of vomiting was added, and a condition of complete anorexia developed. The clinical picture suggested a malignant neoplasm, and while palpation of the abdomen yielded a negative result, I was anxious to investigate the stomach more fully, as apart from occasional shooting pains in the lower limbs his subjective symptoms were entirely gastric. He refused to have the stomach tube passed, and was averse, at his age, to go into hospital. I made a digital examination of the rectum, and found the prostate to be large, hard, and fixed, and, in view of the history, in all probability the site of malignant change. I suggested bringing out a consultant to see him and he agreed to this. Again no objective signs were found to suggest organic gastric disease, in spite of deep palpation over a very flaccid abdomen, and the consultant considered the patient was dying from malignant disease of the prostate. When the patient was closely questioned he admitted having increasing difficulty with micturition, but he thought that was common to all elderly men, so had not mentioned it beforehand. He later submitted to X-ray examination with negative result as regards the stomach. The prostate was too much involved to permit of operative removal.

At the conclusion of the physical examination it is usually

necessary to refer to the patient's history again, and supplement the information voluntarily given with interrogations based on the result of the physical examination. Usually it is possible at this stage to reach a tentative diagnosis, and if this is regarded as functional in origin it is my practice to prescribe a line of treatment for the patient - with the recommendation to return in a week's time. If there is then no improvement further investigation by special methods is adopted. It is not possible, nor does it seem to me desirable, to carry out chemical and radiological tests in each case of gastric discomfort as met with in general practice. Where, however, there is not a definite improvement with treatment, a policy of laissez-faire or the continued use of empirical methods seems equally unjustifiable, until further aids to a definite diagnosis have been utilised.

3. SPECIAL EXAMINATION:

Gastric Analysis.

I place this first in the list of special examinations because (i) it is a method which the general practitioner can, and, I think, should, avail himself of before having recourse to other special means of investigation, with, of course, certain obvious exceptions, such as a recent haematemesis, serious cardiac disease, etc., in which cases it may be inadvisable to risk passing a stomach tube; (ii) when carried out after a careful

clinical examination has been made fuller data are available to interpretate subsequent X-ray findings.

Undoubtedly of recent years there has been a tendency to under-estimate the value of the results of test-meal analysis. Thus at a meeting of the Medical Society of London[15] general disappointment was expressed with the help given by the test-meal in diagnosis. The controversy, too, which has taken place over the significance of lactic acid in the gastric contents has, perhaps, further tended to produce a certain amount of misgivings as to the interpretation of the findings. Thus J. D. Robertson[16] in his interesting monograph hopes that his observations will "deal a final blow to the views of the school of clinicians and biochemists who regard gastric analysis as being capable of an exact and quantitative interpretation". Bennett[17] admirably summarises the situation by expressing his regret that the high degree of precision in the diagnosis of cancer of the stomach which has been obtained by means of fractional gastric analysis combined with X-ray examination during many years past is not more widely appreciated. But, as he says, it is doubtful whether a revival of the theory of the supposed specific importance of lactic acid will help to make the knowledge of this more general.

Regarding the value and limitations of examination of the gastric contents, Einhorn[18] lays down certain guiding principles which I have found to be of value. On the whole he suggests that gastric analysis may be omitted whenever one is able

to arrive at a positive diagnosis without it, and in all acute conditions, the latter tending to amelioration in a short time. The large field in which these examinations are necessary comprises all chronic affections of the stomach with a doubtful diagnosis, that show but slight evidence of improvement. He also takes the view that gastric analysis is not absolutely necessary in chronic conditions which are apparently improving under the established regimen, and in the majority of purely nervous affections of the stomach. In these latter cases, however, I have usually found it advisable to pass a stomach tube, not for the psychological effect - though sometimes in neurotic cases this incidentally seems to be beneficial - but to see whether there is any gross secretory abnormality present.

While it may be open to controversy whether much useful information can be obtained from gastric analysis in cases of functional derangement, there should be no doubt, I submit, that in cases of suspected organic gastric disease information of priceless value may be obtained. Gastric carcinoma can often be definitely diagnosed as soon as it is suspected, gastric and duodenal ulcer may be detected, and the true pathological condition underlying many so-called cases of dyspepsia may be revealed.

It is now generally agreed that the simpler the test meal the better the results, and two methods are in general use:- (i) the Single-Hour Method; (ii) Fractional Gastric Analysis.

The Single-Hour Method. - This is the one I usually adopt. On a fasting stomach in the morning the patient is instructed to take one slice of toast without butter, and one pint of tea without milk, but with sugar if desired. At about 10 p.m. on the preceding evening the patient is asked to swallow one teaspoonful of currants. This is to test whether there is any gross retention or delay in the passage of food from the stomach to the duodenum. Exactly one hour after the morning meal, the stomach tube is passed. The one I use is attached to a Senoran's evacuator, and with this simple apparatus it is possible to recover the gastric contents without serious discomfort to the patient. It is, of course, of practical importance to secure the confidence of the patient before proceeding to pass the tube, and I always endeavour to allay any quite natural anxiety by telling him or her there is no chance of a choking sensation being produced providing they follow my instructions. The act of swallowing by the patient when the tube is at the level of the glottis enables the rubber instrument to enter the oesophagus with the minimum of discomfort.

The quantity of the material recovered is noted, as if this is excessive, - if, for instance, it exceeds a pint - one must decide whether this is due to defective emptying or to excessive secretion. If old food residue is present, such evidence would favour obstruction. If, on the other hand, the return in the evacuator is small, this may be due to a fault in the technique (e.g., the tube may become blocked) so too

much significance should not be placed on this without supporting evidence. The general appearance of the stomach contents is noted as an aid to diagnosis. Some of the material is then filtered prior to applying the qualitative and quantitative tests.

Chemical Examination of Single-Hour Test Meal. -

1. A piece of congo red paper is dipped into the filtered gastric contents. If it becomes definitely blue, free hydrochloric acid is most probably present, but one should remember that if lactic acid is present in considerable quantity a somewhat similar blue colour may be produced. (I prepare the congo red paper by pouring a 0.1 per cent. aqueous solution congo red dye on to a piece of ordinary filter paper, and allowing the latter to dry).

2. As a routine I next apply a test to ascertain definitely whether or not free hydrochloric is present. Gunzberg's reagent is used, which consists of an alcoholic solution of phloroglucin and vanillin. I have only a small quantity made up at one time as unfortunately it does not keep well. About two drops of the reagent are added to a few drops of the filtered gastric contents in a porcelain dish. Heat is gently applied until a dry residue remains. If free hydrochloric acid is present a definite red coloration appears, which is most marked round the irregular edges of the residue. This delicate test serves to eliminate the possibility of error in interpreting an indefinite congo red reaction and in my view should never be omitted.

3. Estimation of hydrochloric acid. 10 c.c. of the filtered gastric contents are put in a porcelain dish and a few drops of Topfer's reagent added. (This reagent consists of 0.1 gram dimethylaminoazobenzol dissolved in 100 c.c. alcohol). In the presence of hydrochloric acid a red colour appears. Decinormal sodium hydroxide is cautiously added from a burette until the fluid in the dish becomes a definite yellow. The number of cubic centimetres of N/10 caustic soda used is noted, and this is multiplied by 0.0365, which result gives the percentage of free HCl present.

The "combined" acid is estimated by adding a few drops of ½ per cent. phenolphthalein in alcoholic solution to the contents in the dish, and again adding alkali from the burette until a red colour is produced. Note is made of the number of cubic centimetres of N/10 alkali used in this second titration, and the result multiplied by 0.0365 to give the percentage of combined HCl. The total acidity is obtained by adding this result to the percentage of free HCl.

I have stressed the importance of first testing for hydrochloric acid with Gunzberg's reagent, as if lactic acid is present in considerable amount it is possible to get a reaction with Topfer's solution, which, of course, would be entirely misleading.

4. Test for lactic acid. - In the absence of hydrochloric acid, the test for lactic acid is carried out as a matter of routine. I do not now rely on Ufflemann's reagent as the results have often proved too indeterminate to be of much clinical value.

Since using a solution of ferric chloride, mercuric chloride, and hydrochloric acid as recommended by MacLean[19] I find it easier to ascertain the presence or otherwise of lactic acid. Two test tubes are taken, and about half an inch of the filtered gastric contents is added to one, and half an inch of tap water is poured into the other as a control. Two drops of the reagent are added to each. If lactic acid is present a characteristic greenish tinge is produced, whereas the test tube containing the tap water gives a scarcely perceptible colour.

This concludes the ordinary routine examination of the single-hour test meal. The significance of the different types of findings will be considered later.

The Fractional Method of Gastric Analysis. - From the point of view of the general practitioner, this method, owing to the fact that it takes from two to two and a half hours to carry out, can be utilised only in exceptional cases, though I readily admit it is of great interest and represents a considerable advance on the older but more practical method.

It is chiefly owing to the work of Rehfuss[20] in the United States that the fractional method has been evolved. The meal is prepared by adding two tablespoonfuls of finely-ground oatmeal to a quart of water and boiling till the quantity is reduced to one pint. This is strained through muslin, when it is ready for use. I usually have this meal prepared at my own home to ensure that it is properly made. The tube I use for

withdrawing the gastric contents is Ryle's[21] modification of the original Rehfuss tube, and I have found it to give every satisfaction. On the evening before the test I instruct the patient to take a glassful of milk containing two teaspoonfuls of charcoal - this has the same object in view as the currants given before the single-hour test. On the following morning the tube is swallowed and when the perforated end is well in the stomach (two circles on the tube show the approximate distance from the mouth to the fundus) a 20 c.c. record syringe is attached to the upper end of the tube and any fasting juice and contents withdrawn and measured. The tube is fixed to the cheek by adhesive strapping. The meal is now swallowed with the tube in situ. About 10 c.c. of the gastric contents are withdrawn at intervals of 15 minutes. My personal practice is to have twelve three-ounce medicine bottles, each numbered, in a box divided into the requisite number of compartments, and the samples for gastric analysis are put into these bottles in sequence. If, after 2½ hours there still remains more fluid in the stomach, the whole of the surplus is then aspirated. By testing each sample withdrawn with a few drops of iodine the presence of starch is ascertained. When no starch reaction is obtained, it is assumed that the rest of the meal has passed into the duodenum.

The quantitative tests are carried out with each sample in the same way as described for the single-hour test with the exception that the specimens are unfiltered, and only 5 c.c. are

used for each test. It is convenient to set down the results as a curve, and I have found the charts printed by Messrs Down Brothers useful for this purpose. The significance of the various findings in fractional gastric analysis will be referred to later.

Occult Blood in the Faeces.

Where this test is positive, the information thus afforded may be of considerable importance from a diagnostic point of view. In all cases which clinically or from gastric analysis suggest the presence of a peptic ulcer or malignant disease I make this test as a matter of routine. I have found the following method of applying the Benzidine Test very suitable:

Into each of two test tubes about ¼ inch of glacial acetic acid is poured. Pure benzidine is added in small quantities till a saturated solution is obtained in each test tube. To each a few drops of ozonic ether are added. A small piece of the faeces is mixed with a little water in a third test tube. The mixture is stirred with a glass rod and then boiled. After slight cooling the contents of one of the prepared tubes are added, and if blood is present a bluish or bluish-green tinge is produced, the depth of the colour depending on the amount of blood present. The control tube serves to show whether there is a definite change of colour. It is of course necessary to ensure that the patient is not subject to bleeding from the gums or nasopharynx, or that he has not been taking butcher

meat during the three days preceding the test.

All the tests which I have described are essentially simple to carry out, and the time required to secure the results is short, and yet it is the case that patients are sent to hospital with a request for admission before any chemical analysis has been attempted. I suggest that at least the single-hour method of gastric analysis should be carried out in all cases of suspected organic gastric disease, excluding the types of cases mentioned on pages 20 and 21, before recourse is had to radiography.

Radiographic Examination.

Within recent years the attitude of the profession towards radiography as an aid to the diagnosis of gastro-intestinal disease has greatly changed. Thanks to improvements in apparatus, in technique, and to the work of numerous investigators, the former sceptical attitude has been abandoned. There is now a strongly marked concensus of opinion among the profession that radiography is not only of the highest value in these conditions, but that it is quite an indispensable factor in the surgical treatment of the various gastro-intestinal affections. The previous critical and sceptical attitude was brought about by the inexpertness of the operators, and the difficulty of appreciating the differences between actual and pseudo shadows. As a consequence mistakes in diagnosis were frequent, but now it would be hard to over-

estimate the benefit to be derived from this means of investigation.

Unlike ordinary gastric analysis, which in my view should be carried out by the practitioner himself, X-ray examinations should be in the hands of an expert radiologist, as it is only after the study of hundreds of patients by radiographic methods, that the true significance of various abnormalities, as revealed by the fluorescent screen and radiograms, can be properly interpreted. Better still, as Paterson[22] suggests, if the practioner sees the patient with the radiologist, and discusses the radiological findings in conjunction with the clinical signs and symptoms. This, unfortunately, is for many of us a counsel of perfection, as a practitioner in an industrial area some miles from a hospital is not in a position to spend hours in such investigations. I am of opinion, however, that the radiologist should be made acquainted with the clinical findings and the results of chemical analysis before he carries out his own examination. This is not the view of all gastro-enterologists, as some, W. G. Morgan,[23] for instance, hold that the X-ray expert should be given no information beyond a request to furnish a report on the gastro-intestinal tract, in case, having the history, etc., he should unconsciously allow such details to influence him in his findings. My view is that such clinical data will enable him to correlate the result of his examination with the physician's findings. If, of course, there is no confirmation of the practitioner's tentative diagnosis, as

a result of the radiologist's report, a further examination may be subsequently required. Briggs and Hurst[24] hold that it is extremely unwise not to give the radiologist details as to what pathological condition is suspected, because the exact technique should vary according to the particular condition which is to be looked for.

Despite the extraordinary help radiography gives as an aid to the diagnosis of gastro-intestinal disease, it undoubtedly has considerable limitations. After all it is the shadow of the alimentary tract we see, and not the substance, and being in a state of more or less movement, the appearance varies repeatedly, and the resulting shadows are correspondingly altered. Hence constant care has to be exercised in order to distinguish a pathological distortion from a natural one.

It is usual to advise an X-ray examination only in those cases which present a clinical picture suggesting organic disease of the alimentary tract. In the case of the stomach and duodenum this practically resolves itself into an investigation for the presence of ulcer or malignant disease. The evidence provided may be direct - a deformity in the outline of the organ - or indirect - changes in the motor functions which may result from reflex disturbances in the activity of the pyloric sphincter. Modern technique has so far improved that it would be unwise as a rule to make a definite diagnosis of gastric or duodenal ulcer without confirmation by direct

X-ray examination. Indeed, in the case of gastric ulcer, Moynihan[25] asserts there are only two certain and unequivocal methods of making a diagnosis, - that of the radiologist and that of the surgeon.

A complete radiographic examination of the organs concerned in digestion includes the lower end of the oesophagus, the renal area and biliary tract, the fundus of the stomach, the pyloric antrum, the lesser curvature, the duodenum, the small intestine, the caecum and appendix, and the remainder of the colon. Barium sulphate is now generally used to make the opaque meal.

Where an investigation is required to estimate the capability of the stomach as regards its emptying power, a "motor meal" may be given at night and an X-ray examination made the following morning. If the barium is still in the stomach there is organic obstruction or motor inefficiency present.

It would be out of place here, I think, to enter further into the technique of what is, after all, a matter for the radiologist, rather than the concern of the physician, though I have thought it advisable to state in a general way, as I see it, the present position radiography occupies in its relation to the diagnosis of gastro-intestinal disorders.

Special Examinations of the Blood and Urine.

Cases which present symptoms of anaemia associated with persistent dyspeptic symptoms, should have a complete blood examination. As regards the red and white corpuscles, an estimation of their number may be conveniently carried out with the aid of a Thoma-Zeiss haemocytometer, and the haemoglobin percentage is readily calculated by means of the Haldane or Gowers Haemoglobinometer. These investigations, like gastric analysis, may be done by the physician himself. When a complete differential count is advisable, I recommend this to be secured through the services of a laboratory expert. Similarly, such investigations as blood-sugar estimations and Wassermann tests should only be entrusted to a skilled biochemist.

Regarding special urine examinations, such as the estimation of urinary diastase in suspected pancreatic disease, tests for urobilin and urobilinogen in possible hepatic dysfunction - here again it is a matter for the expert, and therefore we are not concerned with the exact technique employed.

I have endeavoured in this section to describe at some length the usual procedure I adopt in dealing with my gastric cases. In the next section I propose to discuss the various forms of dyspepsia, basing my remarks on the classification mentioned on p. 5, giving the clinical features of each variety, and describing the method I utilise to differentiate and treat each. Cases which appear to be of special interest will be referred to in some detail.

FUNCTIONAL DYSPEPSIAS.

General Considerations.

While for the sake of clarity I have arbitrarily divided this group into four subsections (p. 5), I quite realise that a very considerable proportion of the patients who come to consult the general practitioner suffer from disturbances which are unaccompanied by detectable pathological change, and often in which the most painstaking search fails to demonstrate any definite change in either tissue or function. Yet these patients suffer, and for the most part may be entirely relieved by properly directed treatment. Many of these conditions eventually lead to serious tissue changes; all of them destroy the sense of well-being in the patient and thus tend to affect adversely his efficiency, so that there is an economic loss to the individual and, indirectly, to the public.

In actual practice the accurate classification of functional gastric cases presents difficulties, which from a perusal of some text-books on Medicine would appear to be non-existent. It is, of course, comparatively easy to give a single clear-cut diagnosis when all the complaints of the patient are definitely related to some serious lesion, such as peptic ulcer or advanced gastric carcinoma. But, as Alvarez[26] says, in an illuminating paper on an analysis of 500 cases of dyspepsia, "how is one to classify the indigestion of a nervous psychopathic woman with migraine, constipation, mucous colitis, urticaria, hypertension, gall-stones, uterine fibromas, and a

husband with whom she cannot live at peace?" In this actual case the first therapeutic experiment was the removal of the gall-stones. As this produced only temporary improvement, the foods which were causing the urticaria and possibly some of the attacks of mucous colitis were next detected and eschewed. Again there was a little improvement, but the woman continued to be prostrated by severe attacks of headache, abdominal pain and vomiting. To make peace in the family the husband then left home, but without much improvement in the patient's condition. The basic cause of the symptoms appeared really to be due to migraine: the stones were impressive but apparently they had been silent and had had little to do with the case. The point to be noted is, that if the physician had lost track of the patient in the first few months after the operation, when she appeared to be cured, her case would have been classified as one of cholelithiasis.

It may be argued that the division of functional dyspepsias into motor, secretory, and sensory neuroses is quite artificial, as in cases in which there is some definite change in secretion, the chief defect may be found in some change in motor muscular activity. To me such criticism appears quite justifiable, but it still seems a suitable system under which to discuss the main types, provided one realises its practical limitations.

Maclean[27] divides the functional disorders into four groups: Hyperacidity, Hypoacidity, Dyspepsia of General Debility,

and Nervous Debility. But here again this classification is arbitrary in that the mere presence of hyper- or hypo-acidity is not necessarily a cause of dyspeptic symptoms. Indeed there would appear to be no perfect classification of functional gastric derangements, which is at once satisfactory as regards the theoretical and practical sides of the subject.

It is important to realise at the outset, that the small group of diseases comprised by peptic ulcer, cancer, and, more rarely, gastritis, constitutes for practical purposes all the organic diseases to which the stomach is usually susceptible. Where these can be definitely excluded a thorough examination of the other organs should be made before asserting the main trouble lies in the stomach.

While it appears justifiable to assert that gastric disorders are usually associated with, and dependent on, certain abnormal changes in the motor activity and neuro-muscular apparatus of the gastric mechanism, it must still be conceded, as Bennett[28] says, that disorders of motility, gastric atony, etc., are found in a very appreciable number of apparently healthy individuals.

Regarding etiological factors in the functional dyspepsias, there have been many put forward which, in my view, play but a small part in the causation of the derangement. Oral sepsis has been a favourite explanation, but I have found that removal of septic teeth is rarely of any benefit, though from the point of view of dental hygiene it is no doubt desir-

able. Dietetic errors, unless of a gross nature, in which case organic disease is more likely, are not basically the cause. Constipation, per se, is not an important factor, though admittedly it is often present with functional derangement. It is more likely to be due to the same cause which produces the dyspepsia. By far the commonest etiological condition in the functional dyspepsias I have found to be instability of the nervous system, whether congenital or acquired through overwork, worry, or fatigue. And it is the realisation of this fact which provides one with a rational method for managing these admittedly difficult cases.

I have already briefly referred (p. 2) to the relative increase of cases of functional dyspepsia in my practice of recent years, where unemployment has been a distressing feature in the life of the community. I attribute this increase in these cases to the worry and anxiety which is now a more or less constant factor in the working-man's household. From observation I do not consider these cases are due to modification of diet brought about by economic necessity, as in all instances there appeared to be an adequate food supply.

I have hitherto refrained from any detailed remarks regarding the anatomy or physiology of the stomach, considering such to be outside the scope of this paper. But the nerve supply of the organ, and its association with the motor and secretory activity of the stomach, are so closely associated with its functional disturbance that it seems advisable to

mention the salient features in this connection.

The stomach is provided with extrinsic nerves from two sources - the vagi and the sympathetic. The vagus is the motor nerve to the stomach, for it has been demonstrated that stimulation of the vagus fibres sets up contractions of the stomach wall. The sympathetic is the inhibitory nerve: when stimulated the sympathetic fibres cause a dilatation of the contracted organ. There is, in addition, a local nervous control, maintained by Auerbach's plexus of fibres, the main purpose of which is to expedite conduction, to correlate the activities of the different regions, and to prevent spasticity. The automatic rhythmical contraction of the stomach, then, is inherent in the muscular coat of that organ, and is merely regulated by impulses from the central nervous system passing down the vagi, and from the sympathetic system by way of the splanchnic nerves. Many factors, however, physical, mental, and emotional, may tend to throw the machinery out of balance. Cannon,[29] in his experiments on digestion in the stomach of the cat made the interesting observation that whenever the animal became enraged or excited, stomach movements immediately ceased.

Regarding the gastric response to food, Pavlov[30] realised that the main secretion of the stomach occurs when food has entered it. He showed that even section of the vagus nerve does not prevent such a flow, and that various foods differed in the degree to which they were capable of exciting such

secretion. These observations have been confirmed by the work of many physiologists, but the problem remains to produce a satisfactory explanation. Pavlov himself, convinced by the failure of vagal section to arrest such secretion, that the mechanism could not depend on the central nervous system, believed it must be due to a short nervous reflex. The more modern "hormone" theory, in which a supposed hormone "secretin" was discovered which has the effect of stimulating secretion by the pancreas, has not been accepted by all authorities, and indeed Bennett (31) is strongly opposed to this hypothesis.

It will thus be seen that there is still dubiety as to the exact modus operandi of the complicated process of digestion and gastric secretion as it occurs in the normal stomach, and I suggest it is rational to presume that factors which tend to upset the nervous system of the individual frequently react on the delicate mechanism which controls the gastro-intestinal tract, to produce a functional derangement.

The elucidation of problems as complex as those concerned with the genesis of abdominal pain is not helped by vague theorising, and it would be quite out of place to attempt any final settlement in so obscure a field, but in concluding these introductory remarks, I should again like to emphasize the great importance of ascertaining the presence or otherwise of organic gastric disease, before attributing symptoms to a purely functional derangement of the organ.

(a) MOTOR NEUROSES.

Gastric Atony. (Myasthenia gastrica).

In this condition there is a diminution of the elasticity and contractile power of the muscular coat of the stomach, whereby the organ is rendered unduly distensible, and is thus prevented from emptying itself within the normal period of time. It follows from this definition that more or less gastric dilatation results unless the disorder is rectified at an early stage.

Etiology. Depending on two main causes, it is convenient to divide cases of gastric atony into two types: (a) Primary Atony, and (b) Secondary Atony.

Primary Atony: The quantity and quality of the food are the chief factors in the production of this type. Indiscretions in quantity of food taken over a long period tend to produce atony: thus the bon vivant who habitually over-eats is peculiarly susceptible to gastric myasthenia, due to degenerative changes in the hypertrophied muscular coat of the stomach. Needless to say in my practice, where the industrial slump has been in evidence for the past few years, it is qualitative dietetic errors which account for most of my cases. On investigation I find that where carbohydrates are habitually swollen with excess of tea or other fluids, symptoms of atony may supervene: the stomach wall becomes unduly stretched and loss of muscular tone results.

Many writers regard the use of animal food as particularly prejudicial to the muscular integrity of the digestive organs, but my experience in this respect is similar to that of Soltau Fenwick[32] who regards meat as far less harmful than a diet which is largely composed of liquids, starches, or vegetables. It is inadvisable to dogmatise, but generally speaking I have found that meat eaters are chiefly liable to secretory disorders (hyperacidity, etc.), whereas those who eat substances which throw the chief stress of digestion upon the intestines, are more likely to suffer from motor derangements of the stomach. In my cases I have found the taking of tea in excessive quantities to be too constant a factor to be a mere coincidence in the production of myasthenia. Whether this is due to an excess of tannin in the beverage or to too great a quantity of fluid consumed over a prolonged period I cannot state with certainty, but in my view it is the latter factor which plays the main part in tending to gastric atony, and for this reason: patients who habitually imbibe large quantities of quite weak tea may present symptoms of gastric myasthenia, even when they show no other dietetic idiosyncrasy. For a similar reason inveterate beer drinkers tend to suffer from this condition, and one of my most obstinate cases (vida infra) appeared to be due to this. Regarding excessive tobacco smoking, I have not satisfied myself that this is per se an etiological factor of much importance, though as a cause of gastric secretory disturbance it plays a prominent part.

Morton (33) attributes the undoubted fact that gastric atony is less common nowadays among young women to the freer and fuller life, both physical and mental, which they enjoy compared with those of previous generations. This seems a rational explanation, for it is recognised that an outdoor life with its associated physical exercises produces a more robust young womanhood.

Worry and anxiety are well-known factors which may contribute to the production of atonic dyspepsia. In such cases a very slight dietetic indiscretion may cause symptoms which normally would pass unnoticed. Cases due to this cause I have found to be especially common in this district of recent years, and they respond to treatment only in so far as the initial cause of the mental anxiety can be alleviated or removed.

Secondary Atony: While this type is naturally more common, in that it is associated with different diseases - phthisis, chronic bronchitis, cardiac, hepatic, and renal disease, etc., - it is of importance to note what is the primary disease so that a rational line of treatment may be adopted. In this connection I wish to stress the importance of making a thorough pulmonary examination where there are signs of gastric myasthenia associated with a generally debilitated state and perhaps a slight cough. A case of this type will be mentioned in some detail later.

Chronic gastritis, from whatever cause, is almost invariably followed by, or associated with, atony, the latter con-

dition in such cases being due to the chronic inflammatory changes spreading into the muscular coat.

Symptoms: Usually the onset is insidious and the condition tends to progress until a state of food retention exists. For descriptive purposes I propose to discuss the usual symptoms in two sections, though clinically the one gradually merges into the other: (i) Pre-dilatation stage of atony; (ii) Dilatation stage with food stagnation and later retention.

(i) Pre-dilatation stage: There is no pathognomonic symptom at this stage to point to a definite diagnosis, but a history carefully taken is a great help as much from the negative facts elicited as from the positive. The severity of the symptoms of course vary with individual cases, but all complain of epigastric discomfort rather than acute pain. A feeling of abdominal distension is usually present, and a sense of weight in the epigastrium. Very frequently I find the patient has already discovered that the intake of fluids, such as soup, readily produces these uncomfortable sensations, and he has therefore modified his diet somewhat before seeking advice. Flatulence, sometimes with acid eructations, is also a common symptom. But even where occasional "heartburn" is complained of in these cases I have usually failed to find, on gastric analysis, any corresponding excess of free hydrochloric acid present in the gastric juice. No doubt the flatulence is frequently due to aerophagy. The absence of vomiting is a negative

factor of some importance in the diagnosis as I have not seen it in an uncomplicated case of gastric myasthenia in the pre-dilatation stage. Where there is a history of vomiting in these cases I have found that it has been artificially induced by the patient's finger being inserted into the throat. This rather drastic treatment certainly relieves the patient of his gastric discomfort for the time being.

The appetite, good to begin with, ultimately becomes poor, as after a few mouthfuls the patient experiences a feeling of satiety. Where, however, there is hyperacidity or gastrosuccorrhoea associated with the atony, the appetite may be increased, as in this type of case the many uncomfortable sensations that ensue during the digestive process are immediately relieved by the further ingestion of food.

Constipation is a most constant symptom, due to a corresponding intestinal myasthenia. The atonic bowel becomes distended with fermentative gases and thus the abdominal discomfort is greatly increased. Thirst is not present. Indeed, there may be an aversion to fluids, as they tend to increase the dyspeptic symptoms.

The degree of mental and physical lassitude which frequently accompanies these cases depends largely on the cause of the atony - thus where it is secondary to a systemic disease such as tuberculosis or cardiac disease the clinical picture is naturally altered or dominated by the presence of such a condition. But even in primary atony I have often noticed that the

degree of mental lassitude appears to be in excess of the physical symptoms.

Physical Signs: The tongue is usually moist and furred, but a more characteristic condition of the organ is where it is flabby and indented along its margin by the teeth. The patient frequently exhibits an unhealthy appearance, modified, of course, by any accompanying organic disease.

Though at this stage inspection of the abdomen is negative, splashing on succussion is, however, obtainable from the commencement of the meal until long after it should normally have left the stomach. If I can elicit a gastric splash an hour and a half or more after a meal, I regard it as a certain indication of impairment of the muscular power of the stomach - providing there are no other symptoms pointing to a stenosed pylorus or organic disease - the duration of the succussion sound indicating roughly the degree of impairment of the gastric musculature.

Regarding the results of gastric analysis, these are inconclusive from the diagnostic point of view. In the primary type there may be no change noted, beyond the free hydrochloric acid remaining present an abnormally long time. In the secondary type I have sometimes found hyperchlorhydria to be present.

(ii) Stage of Dilatation: The tendency is for every case of gastric atony to terminate in dilatation of the stomach, though the onset is quite insidious. Often the patient only

comes under one's observation when a degree of dilatation is present, and then the symptoms are somewhat different from simple atony, so that it may be possible to form a tentative diagnosis prior to the physical examination. At this stage the epigastric symptoms of discomfort are increased, and now it is not only liquids which cause a sense of weight and fulness, but also solid and semi-solid food. The actual location of the pain I find is almost characteristically indefinite: sometimes the chest, back, and abdomen are all complained of as being sites of discomfort. Vomiting, which is rare in the first stage, is now a common feature; it has a sour smell due to the fermentation process occurring in the stagnated gastric contents, and when allowed to stand it typically separates into three layers: a frothy top layer, a middle opaque section, and at the bottom undigested food which is seen to be undergoing fermentative changes.

Naturally the patient exhibits signs of failing health, loss of weight, tachycardia on exertion, with perhaps dyspnoea. To some extent, the hepatic functions become disorganised due to the toxic products of food decomposition getting into the portal circulation. Indeed the whole system may be so deranged that in a cursory inspection the presence of a ptosed, dilated, and poorly functioning stomach may be overlooked, unless the possibility of gastrectasis due to atony is kept in mind.

Physical Signs in stage of Dilatation: On inspection of the abdomen with the patient in the upright position, the

stomach may be seen to be lying abnormally low. In cases which are somewhat advanced, the emaciation of the abdominal wall serves to emphasize the enlarged flabby viscus, appearing as a protuberance low down in the abdomen. Owing to the diminished muscular power of the organ, active peristalsis is not a feature of these cases, and, indeed, if this is found present it would strongly suggest some organic obstruction rather than a simple myasthenia with dilatation. Succussion is obtained long after the stomach should be normally empty. If a tube is passed and air pumped into the viscus, the dilated organ may be seen extending as far as, or below, the umbilicus.

If I decide to give a test breakfast it is my custom to advise a teaspoonful of currants to be swallowed about 10 p.m. the night before. In a typical case of atony with dilatation and retention it is usual to find a few currants floating about in the Senoran's evacuator, pointing, of course, to gross delay in the emptying of the organ.

Regarding the general results of gastric analysis in these advanced cases usually I find some diminution of the free hydrochloric acid, but often there is no marked reduction in the total acidity - due to the presence of organic acids resulting from gastric fermentation. Chronic gastritis is often present and this is suggested where the free hydrochloric acid is diminished.

In doubtful cases X-ray examination is of use in demonstrating the presence of the dilated, slowly-emptying organ.

Prognosis: In cases of primary atony obtained in the pre-dilatation stage I am sanguine that a cure can usually be effected, though I insist that treatment must be maintained long after the symptoms have disappeared. Even where there is some degree of dilatation so much benefit can be derived from a more rational mode of living that it is well worth while endeavouring to obtain the patient's co-operation in this respect. In the secondary types of myasthenia the prognosis must necessarily be more guarded as the extent and nature of the primary disease must exercise an important bearing on the power of the stomach to respond to specific treatment for the organ. But I always assure my patients with gastric atony, that if they are prepared to follow instructions their abdominal discomfort will certainly be considerably alleviated.

Diagnosis: A full clinical history and the result of the physical examination are usually sufficient to suggest the diagnosis. The points I would lay stress on, briefly, are: the disinclination to take fluids owing to the resulting dyspepsia; the discomfort rather than pain after food; gaseous eructations; succussion long after the stomach should be empty; the absence of visible peristalsis. Regarding the differential diagnosis, the following may, in some points, simulate myasthenia: Pyloric stenosis, gastric neurasthenia, gastroptosis, and chronic gastritis.

Pyloric stenosis: Whether from ulcer or carcinoma, the pain is usually more severe after solids than after liquids, even in the early stage. Visible peristalsis is a further differentiating factor, while, in cancer, the rapidity of loss of weight is disproportionate to the length of time the patient has complained of symptoms. X-ray examination in these cases affords valuable means of corroborating or otherwise the presence of organic narrowing of the pyloric region.

Gastric neurasthenia: I have found cases of this condition to present some difficulty in that they may have similar subjective symptoms as regards gastric discomfort, but a careful history serves to distinguish the two conditions. Where headache, insomnia and depression are also present in an introspective patient, combined with the fact that the symptoms vary from day to day without any apparent reason, the probability is that neurasthenia is the essential factor here.

Gastroptosis: The physical signs are a help here, though of course myasthenia may be present also. X-rays may be utilised in a doubtful case to demonstrate the ptosed condition of the abdominal viscera, but I do not usually regard this as a necessary procedure.

Chronic gastritis: The etiology is different; there is no motor insufficiency (except in an advanced stage) and consequently no abnormal splashing. Gastric analysis serves to demonstrate an excess of mucus.

TREATMENT: I place greatest reliance on suitable dietetic measures as being the most important factor in treating cases of gastric atony. And I have come to the conclusion that specific directions must be given to the patient to achieve the best results. It is my custom, therefore, to write out a diet sheet for the individual case with this end in view. General points to be observed are that small meals should be taken at fairly frequent intervals, with limitation in the amount of fluid consumed. Where possible a rest in the recumbent position before and after the meal is helpful. Depending on the result of gastric analysis, the diet should be modified where there is hyperchlorhydria or subacidity, but in the majority of ordinary cases I find little change in the constitution of the gastric juice. Fresh air and moderate exercise are important details to be remembered in the treatment, as it is only by building up the system generally that a permanent improvement or cure can be effected. Associated constipation is, I consider, best treated by suitable dietetic measures, with, in certain cases, abdominal gymnastics (See p. 157).

In the later stages of myasthenia, where there is food stagnation, I find that gastric lavage is helpful to begin with, and for this purpose I use the Senoran's evacuator and tube, with warm water and sodium bicarbonate (two drachms to the pint). Not more than a pint is allowed to run into the stomach at a time, but the process is repeated until the returned water is clear. Twice a week is usually sufficient for lavage, and gener-

ally in two weeks it can be stopped altogether.

The question of electrical treatment is a vexed one, as authorities differ concerning its utility as a remedial measure. Personally I have had no experience of intra-gastric faradization, but as excellent results can usually be obtained by the methods already mentioned I see no reason why this apparently doubtful procedure should be adopted.

It is usual to advise elevating the foot of the bed about twelve inches, as by this means the pelvis is raised above the shoulder level and the weight is removed from the greater curvature of the stomach, thus facilitating the emptying of the organ. Only one pillow beneath the head should be used, of course, or else the object of the bed-raising is defeated. Regarding medicinal measures in myasthenia, I have found these to be disappointing, and beyond general tonics I do not prescribe drugs in cases of uncomplicated primary gastric atony. Where the atony is secondary to some general disease, cardiac, renal, etc., it is of course necessary to deal with this at the same time, as otherwise the gastric condition will fail to respond adequately to treatment.

It may be appropriate here for me to give the general regimen I adopt with atonic cases, though it must be understood that details are slightly modified to suit individual cases:

7 a.m. One half pint of warm water: after drinking a rest of a quarter of an hour on the right side.

7.15 a.m. Abdominal gymnastics. (A sheet is given to the

patient with these exercises in detail: see p. 157). A quick tepid bath followed by a brisk rub down.

Breakfast: Orange juice without sugar; six ounces of fine oatmeal well cooked, with three ounces of milk. Two ounces of coffee without sugar, and small quantity of milk. One slice of toast with butter.

11 a.m. Six ounces of warm broth, free from excess of fat.

1 p.m. A broiled lamp chop, or stewed steak, or small helping of white meat of chicken. A baked white potato or three ounces of baked macaroni, with, if desired, a small portion of well-cooked peas or beans. Two cracknel biscuits with butter. Six ounces of water to be sipped during the meal. If possible rest half an hour lying down.

4 p.m. Five ounces of tea, with milk if desired, and half a slice of toast, with butter.

6.30 p.m. An egg cooked anyway except fried. Two slices of bread with butter. A baked apple. Water - six ounces.

9 p.m. If desired, buttermilk six ounces, with one or two plain biscuits.

In such a daily regimen an attempt is made to build up the general health and to correct the associated constipation. If the latter still persists it may be necessary for

a short time at any rate to prescribe a laxative to supplement the diet and exercises. In this connection I have found Phenolphthalein in combination with a mineral oil, as in Agarol Brand Compound, quite useful. A tablespoonful of this emulsion taken at bedtime is generally sufficient.

Case Histories.

CASE 1. Mrs E., housewife, aged 41. Family history negative. No history of any illnesses of importance. She was an interesting case in that her subjective symptoms were atypical of simple gastric atony. For about five years she stated she had been troubled intermittently with epigastric discomfort occurring within an hour after taking a meal, the pain being sometimes so severe that she "had to walk the floor". At other times she had a feeling of heaviness after meals.

Physical examination on 26.4.32 revealed a well-nourished, healthy-looking woman, tending to plethoric type, and in spite of history of severe pain the previous night, she looked remarkably fit. Succussion sounds obtained though nearly two hours had elapsed since she had had any fluid. Liver dulness not increased; gall-bladder could not be palpated and there was no tenderness in this region. Examination of the abdomen showed slight generalised epigastric tenderness and also slight pain on palpating the right iliac region. At subsequent examinations I failed to elicit any tenderness in the latter locality. Splashing from about an inch above the umbilicus to three

fingers' breadth below that point.

Gastric analysis showed free hydrochloric acid 0.098 per cent., and total acidity 0.229 per cent. Urine and faeces showed no abnormality. Wassermann test was negative. Circulatory system presented no abnormal features.

She was put on an "atonic" diet and at first did not appear to derive much benefit. Indeed she said the acute pain still troubled her. She was sent to the Infirmary for a gastro-intestinal X-ray report on 10.5.32. This stated "No ulcer found; very slight irregularity of the duodenal cap; gastric atony present." The patient was then reassured there was no special evidence of organic disease and she improved steadily thereafter. Up to six months after the X-ray examination there had been practically no discomfort, though her diet was still based on the regimen before mentioned.

In this case I consider she had unconsciously exaggerated the severity of the subjective symptoms. Possibly her age (41) affected her nervous system to some extent.

CASE 2. J.H., male, aged 72; saddler. Family history negative. For at least ten years had been troubled with intermittent attacks of epigastric discomfort within half an hour after meals, otherwise he had been fairly healthy. Definite alcoholic history, with excessive beer-drinking. He complained of a feeling of distension after meals, but there was no acute pain. Appetite poor. Occasional vomiting.

Belching of flatulence frequent. At times vertigo after meals. Constipated. Examined 30.11.31. Patient 5 ft. 8 in. in height, and weight 11 st. 10 lbs. Face somewhat swollen in appearance; dilated venules on nose; conjunctivae congested; in fact his appearance was compatible with the alcoholic history. Chest well formed, though tending to be slightly barrel shaped: some emphysema present. Circulatory system: second aortic sound accentuated; radial vessels hard and readily palpable, suggesting old-standing arteriosclerosis. Blood pressure 160/90. Wassermann negative. Liver dulness extended full to right costal margin. Gall-bladder could not be palpated. Spleen and kidneys presented no abnormality on palpating over their sites. Urine and faeces normal: no occult blood in stools.

Abdomen pendulous in appearance. Splashing sound elicited two hours after a meal from mid-epigastrium to about three inches below umbilicus. No signs of visible peristalsis at any examination.

Gastric analysis: free hydrochloric acid 0.064 per cent., combined acidity 0.075, and total acidity 0.139 per cent. Some excess of mucus and undigested particles present. Evidence of gross food retention, in that currants swallowed the night before were present in the test-breakfast withdrawn.

A diagnosis of gastric atony with dilatation, associated with chronic gastritis and constipation was made.

Treatment: Gastric lavage was carried out twice, but he would not continue with this treatment. His diet was

modified to the "atonic" type, which appeared to relieve the constipation. His age and general condition rendered abdominal gymnastics inadvisable. He improved considerably, and a year later he was still feeling the benefit of the treatment, though the physical signs were little altered. In this case the symptoms were allowed to continue for so many years that hopes of a complete cure were never entertained. Medicinally, dilute hydrochloric acid (half a drachm) in four ounces of water was prescribed to be taken with meals in view of the accompanying subacidity.

CASE 3. A.L., male, aged 34; brassmoulder. History of tuberculosis on father's side, though both parents alive and well. In good health until seven years ago when he developed a cough, and noticed that his stomach was becoming more readily "upset" by slight indiscretions of diet, until latterly he was hardly ever without some discomfort after meals. The pain, never severe, was epigastric in situation, and occurred within an hour after a meal. Definite loss of appetite. No vomiting but frequent nausea. Readily tired on any physical exertion. Complained of palpitation on walking up the short hill outside my surgery.

Physical examination revealed a rather tall spare man of sallow complexion. Height 5 ft. 10 inches; weight 9 st. 1 lb. Within the previous two years he told me he had lost a stone in weight. Inspection of chest showed fairly marked infraclavicular hollows on both sides with some relative diminu-

tion in expansion of right upper chest on deep inspiration. Percussion yielded a duller note over right apex, and auscultation showed signs of bilateral apical catarrh, more marked on right side. The sputum was negative for tubercle bacilli, but the X-ray report on the chest stated there were signs of tuberculosis in both lungs, and this agreed with the clinical findings.

The circulatory system was normal apart from rather soft cardiac sounds and tachycardia on exertion. Blood pressure 130/70.

Examination of the abdomen showed panniculus spare, and costal arches prominent. Succussion sounds elicited two hours after a meal as far as level of umbilicus. No visible peristalsis. Gastric analysis: free hydrochloric acid 0.113 per cent., total acidity 0.229: a normal degree of acidity. No evidence of gross retention of food, though there was definite delay in the emptying of the organ. X-rays revealed a somewhat dilated atonic stomach with no signs of organic disease.

A diagnosis of pulmonary tuberculosis, with secondary gastric atony, was made.

Treatment: For some months the patient received sanatorium treatment, and when next examined by me the pulmonary signs of disease were less in evidence, though the cough was still troublesome. He was advised to take small, frequent, nutritious meals, and cod liver oil inunction was

tried to combat the primary disease. The stomach symptoms did not entirely disappear, but he found that by adhering to the diet scheme he could carry on in comparative comfort. He was advised to give up his old trade (he was a brassmoulder) as the fine dust associated with such an occupation was obviously prejudicial to the health of a tuberculous subject.

Idiopathic Achalasia of the Cardia (Cardiospasm).

It is an established physiological fact that the sphincter at the cardiac end of the stomach is normally closed, and that it relaxes with each peristaltic wave which reaches it. The term cardiospasm is therefore unfortunate, in that it presupposes that normally the sphincter is relaxed. In an attempt to correct this impression Hurst[35] recommended that the condition should be referred to as achalasia of the cardia, thus suggesting that in this condition the cardiac sphincter fails to relax when food is swallowed, thus causing a temporary delay in the passage of the bolus from the oesophagus to the stomach.

Etiology: It would be inappropriate here to detail the different theories which have been evolved to account for the onset of achalasia. Although Hurst is perhaps the chief present-day supporter of the failure-to-relax theory, H. D. Rolleston advanced this opinion as long ago as 1895, while in France a similar view has long been held by Mathieu and

Sencert:[34] these authorities are quoted by Bennett.[12]

Assuming, then, that the condition is not a real spasm, the suggestion that it is due to something interfering with the balance of the vago-sympathetic nerve reflex to the sphincter seems a rational explanation. But even here the ultimate cause of the inco-ordination is not made apparent.

Morbid Anatomy: No change may be found in the sphincter. (In an early case I had which will be detailed later, X-ray examination failed to reveal any gross pathological change in the appearance of the oesophagus or stomach). Where the condition has progressed, dilatation of the gullet results, the distended walls of which may become varicose in time, with haemorrhage as a possible complication.

Symptoms: The commonest symptom complained of is a feeling of the food occasionally sticking in the retro-sternal region. This discomfort may be slight in character and extend over a period of years. Later, regurgitation of solid food may occur, though liquids may pass through into the stomach cavity. In the final stage, when a large oesophageal sac has formed, regurgitation only occurs towards the end of a meal, as the adventitious sac accommodates the first part of the food taken.

In the early stage the nutrition of the patient is unimpaired, but later it is obvious that marked emaciation must inevitably result as sufficient food is not getting through,

and definite evidence of malnutrition occurs. One of the worst cases I have seen was that of the mother of a medical friend of mine, Mrs G., aged 60. For over ten years she had been troubled with increasing difficulty in getting food to pass through the lower part of the oesophagus. She would not submit to any treatment, but it was painful to see her attempting a meal when she was making a social call. Latterly, of course, she refrained from taking food outside her own home. The degree of emaciation became very marked, and finally she developed symptoms of cerebral softening, from which she died, though undoubtedly malnutrition was a factor in undermining her constitution. About three months prior to death she had a haemorrhage from the gullet, probably due to rupture of one of the dilated oesophageal veins.

Diagnosis: When a patient comes with a history of perhaps years of dysphagia, the diagnosis is comparatively easy. But in recent cases, where there has just developed a difficulty in getting food to pass into the stomach, it is of importance to exclude, if possible, the presence of a commencing organic stricture. I lately had a case, Mrs K., aged 52, which presented some difficulty. Her complaint, when seen in June 1932, was simply that her "food seemed to stick in the back of her throat", which symptom had been present about four weeks. Before seeing me she had been attended by a physician in another district who, she told me, had passed a stomach tube without any difficulty. Her appetite was good, though of

necessity she could not completely satisfy her desire for food. The only suggestive factor in her case appeared to be that her loss of weight was out of proportion to the duration of the symptoms. I unfortunately was away for a month after seeing her, but during my absence I arranged for her to be examined at the Royal Infirmary. She was admitted for observation and radiographic examination. On my return I found that no actual oesophageal or gastric disease had been discovered, but that there was an inoperable post-cricoid carcinoma which accounted for the dysphagia. A gastrostomy was performed and she was sent home where I attended her for a few weeks, but later she was admitted to the Cancer Hospital. In spite of X-ray therapy she died about six months from the commencement of the symptoms. At no time could I discover any definite glandular enlargement, though latterly she had considerable pain in the cervical region.

I mention this case to show that even extra-oesophageal carcinoma may simulate, in an early stage, some of the signs of a simple achalasia of the cardia.

Regarding the differential diagnosis, the introduction of a large bougie generally will meet with less resistance than will the passage of a smaller one. The reverse is true of organic stricture. In chronic achalasia, where the condition has progressed to the stage of oesophageal dilatation, the diagnosis may be confirmed by X-ray or oesophagoscopy. In carcinoma of the cardia, where it is still possible to give a

test-meal and pass a stomach tube, there may be achlorhydria, while in achalasia it is unusual to obtain no free hydrochloric acid on gastric analysis.

Prognosis: When got before changes have developed in the gullet, the prognosis is good, but after dilatation, though much benefit from mechanical treatment may be obtained, a more guarded opinion should be given.

Treatment: I think it is a mistake to withhold solid food in these cases as fluids do not help the sphincter to relax. Therefore bland, non-irritating food should form the basis of dietetic treatment, and in early cases more drastic methods may be unnecessary. (Such a type of case will be detailed later). I have not found the exhibition of such an antispasmodic as belladonna to be of much service, but I have prescribed bromides with benefit where there was an accompanying degree of nervous excitability.

When the condition has so far progressed that mechanical dilatation is indicated, by far the simplest method is to pass a rubber tube weighted at its lower end by mercury. The tube may overcome the resistance at the sphincter by its weight alone. When passed it should be left in position for at least a quarter of an hour. The operation may be repeated at intervals of a few days.

In the very advanced type of case, where there is a large sac, and where it is difficult to locate the cardiac open-

ing, I am of opinion that unless X-ray or oesophagoscopy facilities are available, it is advisable if possible to delay blind exploration. The ingenious method of getting the patient to swallow a thread with a glass bead fixed to its end, some twelve hours before attempting to pass a bougie, which interval of time should ensure the bead passing the cardia, and then, after pulling the thread taut, threading an olive-headed bougie over it thus enabling the tip of the staff to be guided to the cardiac orifice, may be necessary in an emergency, but I feel that where such a technique is necessary, the patient should be in an institution or Home where further facilities are available if required. This applies also where further dilatation is carried out by means of a rubber bag, which should be fitted to the bougie just above the olive. Dilators of this type have been elaborated by Plummer[35] and others.

Although such treatment is of necessity uncomfortable from the point of view of the patient, and delicate from that of the operator, as great care must be exercised not to overdilate where the walls of the oesophagus are already thinned, it is infinitely preferable to a gastrostomy, which, while leaving untouched the primary condition, substitutes for it an unsatisfactory method of artificial feeding.

Case History.

Mrs W., aged 48; married; housewife; nervous temperament; one child. Family history negative. Past history:

diphtheria in childhood, otherwise had been healthy. For some months had had discomfort associated with the feeling of a lump at the lower end of the gullet when swallowing food. In spite of this she appeared well nourished, of good complexion, and she was of opinion that she was gaining weight. Oral examination quite negative. Knee jerks slightly hyperactive. Examination of abdomen was negative: no epigastric tenderness elicited. Examination of gastric contents 25.11.31 showed a definite hypoacidity: free hydrochloric acid 0.040; total acidity 0.098 per cent. (Gunzberg Test for free HCl was positive). A slight delay at the level of the cardia was noted on passing the tube. Blood pressure 135/80. Blood Wassermann negative. Urine and faeces negative. X-ray examination failed to reveal any dilatation of the oesophagus and the stomach showed no abnormality.

A diagnosis of early achalasia was made, associated with hypoacidity.

Treatment: Very bland semi-solid diet, taken in frequent small meals was ordered. Ac. hydrochlor. dil. m. xxx in half a pint of orangeade was taken with meals in view of the subacidity. At bedtime sodium bromide gr. xx was prescribed to allay the nervous tendency which was a feature of this case. She improved without the necessity for mechanical dilatation. Ten months later she developed mastoid disease, for which she underwent a radical operation, with satisfactory result. When seen thirteen months after commencing the diet-

etic treatment she appeared to be quite well, her weight had been maintained, and there was symptomatic relief of the dysphagia.

Aerophagy.

This condition is a psychoneurosis and in severe cases it persists long after any primary gastric discomfort has disappeared. As Hurst (36) says, aerophagy may be regarded as a visceral tic, and, like all hysterical symptoms, it can readily be cured by psychotherapy. The condition is characterised by periodic attacks of noisy belching, which occurs as a voluntary act, at first induced to relieve an uncomfortable sensation of distension in the stomach. The air is drawn into the gullet or stomach, and then, by contraction of the abdominal muscles, and diaphragm, the accumulated air is expelled with a loud noise.

Physical signs: These may be negative, though during an attack the stomach is found to be unduly tympanitic to percussion. Palpation reveals no marked tenderness.

Treatment: I do not usually rely on drugs for this condition, but rather explain fully to the patient that the cure rests entirely with herself. (The condition appears to be much commoner in females than in males). She is told that the air-swallowing can and should be controlled and that it is not due to fermentation of food. Very often such a re-

assuring statement is more efficacious than the prescribing of nerve sedatives, though occasionally, where there was a marked nervous instability I have given bromides with benefit, but without psychotherapy the habit tends to persist.

Case History.

Mrs McA., aged 45; housewife; married; four children. Highly neurotic temperament. Shortly after her first confinement she developed a right-sided facial paralysis, and this has never completely cleared up. Domestic life not very happy. She complained of frequent "belching of wind", which symptom had been annoying her for several months. Associated with this she had a sensation of gaseous distension in the epigastric region. No nausea; no vomiting. Occasional palpitation after meals.

Physical examination revealed a somewhat spare woman of medium height. She had false teeth; tongue moist but coated at the back. No evidence of goitre. Chest examination negative. Heart sounds somewhat soft; pulse 90. Blood pressure 130/75. Knee jerks active. Abdomen showed no particular abnormality. Urine and faeces normal. Gastric analysis showed a total acidity of 45 (Ewald scale), with free HCl 35.

A diagnosis of aerophagy associated with neurasthenia was made.

Treatment: The condition was explained to her, and a bland nutritious diet was advised, with rest before and after

meals whenever possible. Emphasis was laid on the necessity for her co-operation by avoiding air-swallowing. Under this régime the eructations became less troublesome, and when seen six months after commencing treatment she had obtained a symptomatic cure as regards the aerophagy.

Pylorospasm.

As its name implies, this is a spasmodic contraction of the pylorus, without organic disease. In my experience it is rare to get a case of primary pylorospasm: it is far more apt to be secondary to some organic disease than to be primarily a functional disorder, and for this reason it is of the utmost importance to investigate fully a case presenting the symptoms of this condition.

Etiology: The exact etiology is obscure, though it is probably another evidence of vagotonia, as the condition has been induced in animals by stimulation of the vagus (Morgan).[37] While recognising, then, that pylorospasm may be a pure neurosis, it is more usually the result of reflex stimulation caused by disease elsewhere, as in gastric ulcer, appendicitis, cholecystitis, and, in women, disease of the pelvic organs.

I have usually found hyperchlorhydria to exist in cases which present signs of pylorospasm but as hyperacidity is present in many other types of gastric disorders it would

be inadvisable to attach much importance to this as an etiological factor.

Symptoms: Pain is the most prominent symptom, and it may be extremely severe. It is epigastric in situation, perhaps radiating to the right side, and occasionally it may spread over the abdomen. It is spasmodic in character, associated as it is with an increase in the peristaltic contractions of the gastric musculature. Vomiting is not uncommon, where the spasms are severe, and this may relieve the pain

Physical Signs: In simple idiopathic pylorospasm nothing abnormal may be noted on inspection of the abdomen. I have not seen visible peristalsis in such a case, though it may be present in a thin patient. Where there is definite visible peristalsis organic stricture producing a stenosed pylorus is a more likely explanation of the symptom. On palpation there may be much epigastric tenderness elicited, and it may be possible to palpate the peristaltic waves or to feel the pylorus as a small hard swelling if it is in a state of spasm at the time of examination. Palpation of the other regions of the abdomen should be carried out with care, especially the right hypochondriac and right iliac parts, in view of the possible association of cholecystitis or appendicitis with the condition under consideration.

It would be misleading to suggest that haematemesis may be regarded as a symptom of pylorospasm but some months ago

I had a case, fuller details of which I propose to give later, where, in a gastric crisis associated with tabes dorsalis, there was a severe haematemesis without any other sign of peptic ulcer to account for this symptom. I venture to think that violent pylorospasm played an important part in the production of the haemorrhage. In this connection it may be of interest to note that Bennett[38] has recorded three cases of this phenomenon associated with early locomotor ataxia.

Gastric analysis: Usually there is an excess of acidity in the gastric juice though this is not pathognomonic; but I endeavour to give a test-meal in these cases, as where there is an achylia the strong probability is, one is dealing with an organic stricture. So gastric analysis may be a useful aid to differential diagnosis.

Diagnosis: It is necessary to ascertain as definitely as possible the presence or otherwise of organic disease before arriving at a positive diagnosis of simple pylorospasm, and to this end a radiological examination is of great importance. Indeed I venture to think that no case should be labelled idiopathic pylorospasm (a rare condition) before every diagnostic aid has been invoked. Where the stomach is found to be free from abnormality, the gall-bladder and appendix regions should be examined with some care. I lately had a case, which I propose to mention in detail later, which presented many of the symptoms of an ulcer in the pyloric region, but which on further examination was found to be secondary pylorospasm due

to chronic appendicitis. In this connection Aaron's sign (39) is of some practical interest: it consists of a referred pain in the epigastrium, left hypochondrium, umbilical or praecordial region from continuous firm pressure over the appendix. Aaron has found this sign valuable in deciding where and when not to recommend operation for chronic appendicitis. He has found this test to be repeatedly confirmed by subsequent operation, and he considers the referred pain in these cases to be due to spasm of the pylorus or duodenum.(40)

Einhorn and Scholz(41) have elaborated a mechanical type of diagnostic test with a fine copper wire covered with braided silk to which a metal ball is attached at one end. This apparatus is called a delineator string. It is introduced into the stomach in the evening and left in situ overnight. X-ray examination takes place next day. If the ball is still in the stomach the obstruction is probably organic. If the ball is in the intestine but the string is found to be irregular (having a zig-zag appearance) then some degree of spasticity of the pylorus is presumed to be present.

I have not had practical experience of this ingenious test, but I suggest that a careful clinical history combined with expert radiography should suffice to establish a diagnosis.

Treatment: The diet, as in achalasia, should be bland and non-irritating. Small meals at frequent regular intervals appear to be better tolerated than larger meals thrice daily. Heat applied to the epigastrium during an attack of severe

pain is helpful, and if vomiting is a feature the application of a mustard leaf to the same region may afford considerable relief. I have found the prescribing of belladonna in the form of atropin gr. 1/100 distinctly of benefit in relieving the spasm. Later, when the acute symptoms have passed off, the following type of diet is suggested:

8 a.m. Grape fruit juice without sugar; one egg cooked any way except fried; a slice of toast with butter. One cup of weak tea.

12 noon. Fresh fish boiled or steamed; or small portion of a lean broiled lamb chop. Vegetables: fresh green peas cooked until very soft, or well-cooked carrots. If potatoes desired, a very small quantity well mashed. Baked or tinned pear, or the soft part of a baked apple. A glass of water.

4 p.m. A slice of thin toast or bread and butter. A cup of weak tea.

6 p.m. Small portion of fish if not taken at dinner; or one egg; white bread and butter. A glass of water.

In the early morning and between meals, a glass of water.

At bedtime I have found the administration of olive oil in the form of an emulsion with Horlick's malted milk and hot water - the so-called "Orzone" - as recommended by Morton,(42) to be of benefit in some cases. As he suggests, it not only helps to relieve any tendency to spasm, but also it may coat

the mucous membrane and control to some extent the excessive flow of hydrochloric acid so often present in pylorospasm.

Case Histories.

CASE 1. M.B., male, aged 45; riveter. Family history negative. History of gastric discomfort intermittently for years. He had been a heavy alcoholic drinker and was still a fairly heavy smoker. Present attack of gastric symptoms had lasted eight weeks; pain in epigastric region usually $2\frac{1}{2}$-3 hours after meals. The pain was at times spasmodic and so acute as to cause vomiting. The taking of more food sometimes afforded him temporary relief. Appetite not good.

Physical examination revealed a man of medium height, of sallow complexion and under-nourished appearance. His weight, he told me, had been practically stationary for a number of years. Masticating surfaces in unsatisfactory state: very few teeth, and these carious. Circulatory and respiratory systems negative. Blood pressure 120/90. Blood Wassermann test negative. Urine and faeces negative. After an Ewald test-breakfast on 12.7.32, the following result was obtained: Gunzberg test positive for free HCL. Free HCl 0.228; total acidity 0.332 per cent.: a definite hyperchlorhydria. No evidence of gross food retention. Some excess of mucus. Examination of the abdomen: tenderness elicited on palpating over mid-epigastrium. Palpation over right iliac region produced no local discomfort but patient experienced a slight pain in epi-

gastrium. No visible peristalsis at time of abdominal examination.

I felt that further investigation was advisable so patient was sent to the Infirmary, and on 16.8.32 an X-ray report on the gastro-intestinal tract was received. This stated: "Stomach somewhat ptosed; peristalsis unduly active; no evidence of ulcer in stomach or duodenum. The appendix appears diseased. The caecum is movable".

After this a Fractional Test-meal was carried out with the following result: Fasting juice 15 c.cs. No bile or charcoal. Free HCl 5; total acidity 10. At ¼ hr. free HCl 2.5; total acidity 15. Thereafter it rose to 35-40 for free HCl, and 55-60 for total acidity at ¾ hr. It gradually fell to 20 and 45 respectively at 2¼ hours.

Diagnosis: Pylorospasm secondary to chronic appendicitis. There was probably an accompanying degree of chronic gastritis, but this in itself did not appear to account for the acute spasms of pain, and the response to atropin was such as to point to some spasm of the pylorus.

Treatment: He would not submit to appendicectomy, so treatment on medical lines was adopted. A very bland diet was ordered, and later a fuller diet, similar to that detailed on page 71 was advised. 1/100 gr. atropin half an hour before breakfast and a similar dose before the evening meal was given to relax the pylorus. Under continued treatment he made slow

but satisfactory progress. The edentulous condition of his gums was pointed out to him as a contributory factor towards his ill-health, and early dental treatment was strongly urged. In view of the tendency to hyperacidity he was advised to stop or curtail smoking.

Discussion: From a physician's point of view this type of case is unsatisfactory, in that permanent relief of the patient's symptoms is scarcely to be looked for. The unhealthy appendix is still present and liable to upset normal gastric function, apart altogether from the possibility of the supervention of an acute appendicitis. At the same time it has to be admitted that the patient has considerably improved during the past six months, and has had no further symptoms attributable to gastric dysfunction.

CASE 2. W.B., male, aged 57; engineer; married; no children. Father died when 70 from Bright's disease; mother died at 60; actual cause of death not known, but patient said she had severe "lupus of the face" before she died. He has one brother in an asylum with "general paralysis".

Though never robust, patient had been in good health up to six years ago, when he had a severe attack of abdominal pain with vomiting. At that time he was living at Renfrew. His medical attendant was called in, and advised removal to hospital, where patient was kept for three weeks. A thorough X-ray examination of the gastro-intestinal tract was carried out, but he was told there was no sign of organic disease; this

was confirmed by the report his doctor received. A year later he removed to Perth, where he had more gastric attacks, at intervals of a few months. He now began to feel some weakness in the lower limbs, associated with intermittent shooting pains. His medical attendant referred him to the dispensary for venereal diseases attached to the hospital, where he had a series of injections. I am unable to state the result of any blood examination at that time. Two years ago he came to this district, and when I examined him he showed typical clinical signs of tabes dorsalis: loss of the deep reflexes, small pupils showing the Argyll-Robertson phenomena; Romberg's sign positive. His gait showed that he had reached the ataxic stage of the disease. His blood Wassermann was indefinite, but this may have been due to the anti-specific treatment he had received elsewhere. The cerebrospinal Wassermann test was negative. His blood pressure was 170/100, and there was definite thickening of the radial arteries. Respiratory system negative. I referred him to the dispensary for venereal diseases, and his case was regarded as one of locomotor ataxia, and further treatment was carried out.

On 1.5.32 I was sent for and found him in great pain and vomiting large quantities of coffee-ground material, though before I arrived he had had a definite haematemesis. The pain was spasmodic in character and epigastric in situation. I felt what I considered to be accentuated peristaltic waves over the stomach region. Under treatment (<u>vide infra</u>) he soon im-

proved and within a week he felt so much better that he was anxious to get up. By now there were no signs whatever of epigastric tenderness, and apart from his deranged nervous system he had no pathological symptoms.

It is now ten months since the acute attack, and I have seen him on several occasions within this period. He has remained entirely free from stomach symptoms, though for some months he has been on ordinary diet. Gastric analysis revealed a subacidity: free HCl 15, and total acidity 30. No occult blood apart from that found in the specimen examined after the acute attack. No occult blood in the faeces.

Diagnosis: Gastric crisis with severe pylorospasm associated with tabes dorsalis.

Treatment: At the time of the attack his pulse was poor, and he was somewhat collapsed. Heat applied to the abdomen did not afford relief so, at some risk, morphia gr. $\frac{1}{4}$ combined with atropin gr. 1/100 was given, and within half an hour he felt much easier. Some ice-chips were given to be sucked, and the next day small amounts of peptonised milk were allowed, and were found to be well tolerated. There were no facilities for regular rectal feeding, and fortunately this did not prove necessary. Atropin gr. 1/100 twice daily was prescribed and the diet gradually supplemented, until he was on a regimen such as that detailed on page 71. A month later antispecific treatment was resumed.

Discussion: I realise, of course, that it is quite possible for a tabetic patient to have a peptic ulcer at the same time, and there is no doubt that haematemesis is a commoner symptom of ulcer than it is of a gastric crisis. Hurst,(43) indeed, definitely states that "the occurrence of haemorrhage should be regarded as conclusive evidence against tabes being the sole cause of the symptom, though at one time it was erroneously believed that a true tabetic crisis could give rise to haematemesis." But I venture to think that in this case, where (i) there were no gastric symptoms in between the severe visceral crises; (ii) there was absence of radiological evidence of gastro-intestinal disease; (iii) there was the absence of the typical history of an ulcer diathesis, it is justifiable to conclude there may be another etiological factor to account for the haemorrhage, and I incline rather to the view of Bennett, before mentioned, that the severe gastric contractions during the crisis, and which could be felt clinically, produced a spasm of the pylorus so violent as to cause haemorrhage from the local blood vessels. Incidentally the patient had signs of arteriosclerosis, his systolic blood pressure was 170, and these factors may have been contributory agents in the production of the haematemesis.

(b) SECRETORY NEUROSIS.

Hyperchlorhydria.

Whether hyperacidity should be considered a disease sui generis or not is difficult to decide. It is clearly recognised nowadays that a person may have a gastric acidity well above the average without it necessarily producing any subjective symptoms. On the other hand, in my experience, it is more usual to find that a patient with a total acidity of say 70 or over, and where an exhaustive physical and radiological examination has failed to detect any anatomical lesion, complains of a characteristic train of symptoms which it may seem fair to assume has its origin in hyperacidity. But against this hypothesis experimental evidence has been adduced to show that even with a sudden large concentration of free hydrochloric acid in the stomach no epigastric pain or discomfort may be experienced: I refer to the work carried out by Bennett and Dodds[44] in the laboratories of the Middlesex Hospital. Briefly, they state that the average amount of "resting" juice one can withdraw from a normal fasting stomach is roughly about 10 cc. every ten minutes. If a hypodermic injection of histamine is now given the subject shows an immediate flushing of the face, but no gastric symptoms. Aspiration of the gastric contents now reveals dramatic effects; for twenty to thirty minutes the stomach will pour out large quantities of a juice stronger than can be collected in any other manner.

An average secretion, ten minutes after the histamine injection is 120 c.c. of juice containing 0.36% hydrochloric acid, yet the secretion of this juice is not attended by the least epigastric pain or discomfort, no matter whether it be aspirated or left in the stomach. In view of these facts Bennett[45] states "it seems irrational to accept hyperchlorhydria as a pathological entity, or as an explanation of the abnormal symptoms in any patient."

I have quoted these experimental details to show that the problem is not so simple as it may at first appear, but I venture to suggest that where excessive acidity has been present for months or years it may produce symptoms from some subtle pathological change in the gastric mucosa, whereas the sudden experimental increase in the acidity of a stomach which has hitherto been secreting normally may fail to elicit abnormal gastric sensations. I therefore propose to deal with hyperchlorhydria as a condition which, per se, is capable of producing symptoms.

Etiology: Depending on the nature of the exciting factor, it is convenient to divide the condition into (a) primary, and (b) secondary hyperchlorhydria.

(a) Primary Hyperchlorhydria: It is usual to attribute to dietetic indiscretions an important place in the causation of primary hyperchlorhydria, and certainly persons who habitually take excess of foods flavoured with condiments, or who are excessive smokers or drinkers of

alcoholic beverages, - these individuals are liable to suffer sooner or later from hyperacidity. But I regard the psychic factor as of equal importance in the production of this functional derangement. Of recent years I have gone into the dietetic history of many cases of hyperchlorhydria, and very frequently I have come to the conclusion that worry and anxiety were the only demonstrable reason for the gastric symptoms. I am of course aware that this is a well-recognised factor in the production of the functional dyspepsias, but it seems to be particularly prone to result in hyperchlorhydria. It is needless to stress the importance of realising this from the point of view of successful treatment.

Insufficient mastication of ordinary food through habit or absence of teeth may be a contributory factor, while insufficient cooking of food, particularly excess of hard vegetables, may produce the symptoms of hyperacidity. Lastly, any condition tending to exhaust the nervous system indirectly such as masturbation, haemorrhage, living in badly ventilated rooms - these and similar factors may play an unsuspected part in the production of primary hyperchlorhydria.

(b) Secondary Hyperchlorhydria: Duodenal ulcer is perhaps the commonest single organic disease to be accompanied by secondary hyperchlorhydria, though a gastric ulcer, particularly in the pyloric region, may be associated

with hyperacidity. Among the extra-gastric causes, appendicitis, cholecystitis, cerebral tumour, and, in women, pelvic disease, should be remembered as often having an accompanying degree of hyperchlorhydria. Exactly how this reflex action on the gastric juice is brought about is still a matter of dubiety.

Symptoms: Usually I find the patient is unable to state definitely just when subjective symptoms first occurred, the onset is so insidious. Sooner or later, however, pain occurs, and this is the most constant symptom. To begin with it is rather a sensation of discomfort than actual pain. It may be characterised as a burning sensation in the epigastrium which is relieved later by acid eructations. Later definite pain occurs, and usually within two hours after taking a meal. An interesting feature of the pain is, that meals consisting largely of carbohydrates produce more discomfort than those which contain a preponderance of proteid food. A probable explanation of this is the recognised fact that there is a strong chemical affinity between free hydrochloric acid and albumin, the compound formed when the two are brought together being of considerable stability, and not endowed with the irritant properties of the uncombined acid.

The pain or severe discomfort is usually relieved by taking food, for this reduces the concentration of the acid. Indeed the patient may experience a hungry sensation, and the clinical picture in this respect may then

be typical of the so-called "hunger-pain" of duodenal ulcer. I am therefore unable to regard hunger-pain as pathognomonic of an ulcer of the duodenum, as I have repeatedly found this symptom in simple hyperchlorhydria. This will be further considered when dealing with the differential diagnosis. Acid eructations are frequently complained of. As the acidity rises peristalsis becomes more and more active, but the pylorus remains more or less closed, and thus the pressure inside the stomach tends to increase. To a less extent the cardiac sphincter is also involved. This sphincter, being the weaker of the two usually yields first, when a copious gaseous eructation occurs, and the patient experiences temporary relief.

Vomiting: This is not a common feature, but the patient may at times artificially induce it with his finger in the throat, as experience teaches him that he will thus obtain rapid ease.

The appetite in an uncomplicated case is usually good, owing to the hyperacidity acting as an appetizer, and hence these patients rarely look undernourished. Indeed considering the pain and discomfort they so frequently suffer from they appear to be remarkably fit. Constipation is a fairly common symptom.

Many writers state that vertigo is frequently present in these cases, but in my experience it is too inconstant

a factor to be of any real significance from a diagnostic point of view.

Physical Signs: During an attack there may be generalised epigastric tenderness on palpation, but this varies with the severity of the attack. In the intervals between the acute symptoms, in an uncomplicated case, the abdomen presents no abnormal features. In short, the physical signs are so indefinite as to be of little use diagnostically without gastric analysis. Of course, where the hyperacidity is secondary to organic disease, the physical signs will be correspondingly altered.

Chemical Analysis: This is really necessary before a definite diagnosis can be made. The fractional method or the ordinary Ewald test breakfast may be used. Where the one-hour method is adopted and gives a normal result in spite of a typical history of hyperchlorhydria, it is advisable to try the fractional method, as it is recognised that the hyperacidity may develop at any period during digestion, and it is possible that the single test has missed the high acidity stage. For this reason, then, the fractional examination of the gastric contents is the method of choice, but from the general practitioner's point of view it is not always practicable, owing to the time required for such a test. I suggest, therefore, that where, as a result of single-hour analysis, the result is "positive" - where

there is definitely a hyper-normal acidity, with no other pathological signs - the case may be regarded as one of hyperchlorhydria, but where the result is "negative," it does not necessarily follow that the patient is not subject to hyperacidity at some period of the digestive process.

Prognosis: Early uncomplicated primary hyperchlorhydria is usually susceptible of cure with care and patience. The more chronic variety may be greatly relieved by suitable treatment, though relapses may occur. Regarding the secondary type, the prognosis depends on that of the primary disease.

Diagnosis: The main points to note are: post-prandial discomfort, relieved by taking food or alkalies; pyrosis; keen appetite; fasting stomach empty; increased hydrochloric acid in gastric secretion.

Before the condition can be definitely regarded as primary hyperchlorhydria, the following diseases or derangements must be excluded: peptic ulcer (gastric or duodenal); cholecystitis; chronic appendicitis; chronic hypersecretion.

Peptic ulcer: The symptoms are usually more constant and severe, and I would lay particular stress on the small localised area of particular tenderness so frequently found in ulcer. Again, a continuous secretion of gastric juice rather than simple hyperchlorhydria usually accompanies

peptic ulcer. Radiological examination may provide very useful confirmatory evidence.

Cholecystitis: In this condition the administration of alkalies fails to afford the relief which such treatment gives in simple hyperchlorhydria; and the pain radiates chiefly to the right hypochondriac region and perhaps to the angle of the scapula posteriorly. If there is jaundice, or a history of jaundice, the condition is more likely to be due to a deranged gall-bladder.

Appendicitis: This does not usually simulate a simple hyperchlorhydria so frequently as do the above-mentioned two conditions. The pain and tenderness in the right iliac region, unaffected by the taking of food, and unrelieved by alkalies should point to appendicular disease.

Hypersecretion: Here the symptoms may be similar to hyperacidity, and it is only by passing a stomach tube in the morning before the first meal that the presence or otherwise of a fluid rich in free hydrochloric acid can be ascertained. Again, dilatation of the stomach is common in chronic hypersecretion, whereas it is unusual in simple hyperchlorhydria. A poor appetite and excessive thirst, which are frequent symptoms in hypersecretion, are further aids in the differential diagnosis.

TREATMENT: It is essential in my view to ascertain if the patient has any particular worry or anxiety, as this

may be a potent factor in maintaining the secretory derangement in primary hyperchlorhydria. If such can be found and removed an important obstacle to cure has been overcome. In a case I had some time ago the dread of cancer seemed to be the exciting factor, and when this fear was removed the symptoms became less troublesome, and ultimately she obtained complete relief.

Dietetic treatment is of course of considerable importance, as it is advisable to suggest a diet which does not tend to unduly stimulate the secretion of gastric juice. For this purpose I select a diet based on this principle, as follows:-

Breakfast: Fine oatmeal or cream of wheat (well-cooked) eaten with milk or cream, without sugar. One or two eggs cooked any way except fried. A slice of cold toast with butter. A cup of freshly made tea with plenty of milk, but no sugar.

Dinner: Chicken, beef or lamb all to be cooked very soft - preferably stewed. Potato thoroughly baked or boiled; cauliflower or broccoli or carrot well chopped before cooking. Baked apple or tinned pear. If salad is desired it should be made simply of olive oil and salt to which may be added a small quantity of lemon juice. Eight ounces of water.

Afternoon: Plain biscuit or two; cup of weak tea with plenty of milk but no sugar.

Supper: Steamed or boiled white fish; stale bread and butter or toast; weak tea with plenty of milk and no sugar.

At bedtime an emulsion of Horlick's malted milk with olive oil is often well-tolerated.

Condiments and alcoholic beverages, strong tobacco, and sweet foods are forbidden as tending to stimulate gastric secretion. Where possible I suggest a rest period of half an hour after meals.

In the average working-man's household it is not possible to have such a dietetic scheme carried out to the letter, especially in times of economic stress, but I make a point of giving these cases a diet-sheet based on such an outline, as I find that, though in parts it is impracticable, it at any rate serves to prevent a conscientious patient from going far astray in the matter of meals. The tendency to take an excessive quantity of fried food is very prevalent, and a diet based on the lines above-mentioned should serve to cut out this common indiscretion.

Foods rich in carbohydrates are less well tolerated than proteid foods, but the proteins should be fresh rather than preserved: thus ham, bacon, smoked fish etc. should be excluded from the diet.

Where gastric dilatation is associated with the hyperchlorhydria, it is inadvisable to have a vegetable oil in the dietary, for while it certainly diminishes the acidity, it also tends to aggravate the stasis in an organ which is already hypomotile. On the other hand, in simple uncomplicated hyperacidity, peristaltic movements are usually overactive, and the administration of olive oil in any form is doubly helpful.

Constipation is a frequent accompaniment of hyperchlorhydria, and where the condition persists in spite of endeavouring to inculcate a regular habit, abdominal gymnastics may prove helpful when the acute symptoms of hyperacidity have passed off. This will be more fully discussed later. Emulsio magnesiae (B.P.C.) taken at bedtime is useful both as an antacid and gentle purgative in cases which require further specific treatment for the intestinal stasis.

Medicinal treatment: It is my practice to supplement the psychotherapic and dietetic treatment with suitable alkaline medication in the early stage, but I endeavour to discontinue drugs long before the diet scheme has been abandoned.

Much has been written in condemnation of the prescribing of sodium bicarbonate in hyperacidity in that, while it gives temporary relief by liberating carbon dioxide which in turn helps to relax the pyloric sphincter and thus allows some of the food to leave the stomach, it is follow-

ed by a secondary rise in the amount of free hydrochloric acid secreted. This is certainly a theoretical objection to the drug, but for a number of years I have used it in conjunction with the carbonates of magnesium, calcium, and bismuth, and I have found on the whole that such treatment was justified by the results obtained. Certainly such a powder tends to produce some gaseous distension of the stomach, but the consequent eructation of gas affords the patient relief from a troublesome epigastric sensation common in cases of hyperacidity. As an alternative I have found the administration of "triple" phosphate in drachm doses helpful in many instances.

Belladonna or its alkaloid has an inhibitory action on the secretion of gastric juice, and therefore it provides another rational means of treating hyperacidity, but in my hands alkaline medication has yielded better results, and I prefer to reserve atropin or belladonna for cases which have an accompanying degree of hypersecretion.

Regarding lavage, I do not consider this a method of choice in simple hyperacidity, though where gastrectatis is present it may be very helpful.

The treatment of secondary hyperchlorhydria must be based on the nature of the primary disease. Where an apparently simple hyperacidity fails to respond to appropriate treatment it is probable there is an undiscovered organic reason for this, and I suggest an X-ray examination, even

in the absence of clinical symptoms pointing to more serious disease.

Case Histories.

CASE 1. R.A., male, 49; machinist; married. Family history negative. Apart from hernia seven years ago, and acute appendicitis five years ago, both of which had been operated on, he had been well. The appendicitis occurred as an acute attack, and was immediately treated surgically. He remained free from symptoms for several months after this, and then he began to have intermittent attacks of gastric discomfort from one to two hours after meals. He characterised the pain as dull, and it extended over the epigastrium. It was followed by acid eructations and excessive belching. Apart from being a fairly heavy smoker his habits were temperate. He had no particular worries, but was inclined to hurry his meals.

Examination of chest organs and abdomen was negative. Blood pressure, urine and faeces all within normal limits. Test meal was given 23.1.32., with the following result: free HCl 65; total acidity 80. This man did not submit to X-ray examination as he wished to try the effects of medical treatment first. He was put on a dietetic regimen similar to the one previously outlined, with alkaline powder. Smoking was curtailed and he agreed to rest a quarter of an hour after meals. He obtained a symptomatic cure, and up to eighteen months after treatment he was keeping fit.

Whether the hyperacidity was actually present before his appendicitis, or whether it developed quite apart from this, I am not prepared to state. Realising the affinity between gastric secretion and disease of the appendix one might feel justified in regarding the case as a hyperchlorhydria secondary to a previously diseased appendix. It would have been interesting to have a gastric analysis performed before his operation, though at that time he had no subjective gastric symptoms. If the case is really a secondary hyperchlorhydria, then the removal of a diseased appendix cannot be regarded as a cure of an associated hyperacidity.

CASE 2: J.A., male aged 38; factory worker; married. Family history not significant. Apart from history of attacks of "indigestion" for about three years he had been in fair health, though not of robust type. His complaint was that soon after eating he felt a sense of gastric fulness, followed about an hour later by acid eructations. He was clear that condiments and raw fruit aggravated his symptoms. He was of a nervous disposition, and had been worried by long periods of unemployment.

Patient was of spare build and sallow complexion, and habitually wore an anxious expression. His weight had been stationary for years. Examination of the chest organs was negative. Beyond slight epigastric tenderness the abdomen revealed no abnormality. Blood pressure 130/70; urine and

faeces normal. Wassermann blood test negative. Gastric analysis 25.2.32: free HCl 68; total acidity 84; no evidence of gross food retention; no excess of mucus. X-ray examination revealed no gastro-intestinal abnormality.

A diagnosis of simple hyperchlorhydria was made and he was put on an appropriate diet. It is over a year since I first examined him and he has been on a modified diet during that period; also he has been in steady employment during this interval. He has secured symptomatic relief, and his general health has improved, but I attribute this as much to freedom from worry as to the alteration in his diet. No alkaline medication was necessary in this case.

Hypersecretion. (Gastro-succorrhoea.)

There is a tendency in some modern text-books on gastroenterology to regard hyperchlorhydria and hypersecretion as synonymous terms, but I suggest this is fallacious. In other words, it is wrong to assume that excess of secretion must necessarily entail an excessive concentration of free hydrochloric acid as well, though I admit in my experience it is more usual to find this occurring than diminished acidity with hypersecretion. Clinically it is only after careful investigation that a diagnosis of hypersecretion is justified, and in general practice I am of opinion that a positive finding of gastro-succorrhoea is rarely possible without a fractional test-meal.

For descriptive purposes it is convenient to divide the condition into two types, intermittent and chronic, though clinically the one may merge into the other imperceptably.

Etiology: In the intermittent form the causation may be similar to that of hyperchlorhydria: worry, excitement, dietetic indiscretions over a long period etc. The patient is usually of the emotional, neurotic type. In the chronic form it is more probable that organic disease is responsible, especially gastro-duodenal ulceration, gall-bladder disease, or appendicitis.

It seems a safe axiom, where chronic hypersecretion is concerned, to regard it as a manifestation of organic disease - either intra-or extra-gastric - unless exhaustive examination fails to locate such. Even then, the possibility of having overlooked disease in a remote organ should not be lost sight of.

Symptoms: A feature of the intermittent type is, that the patient may go to bed in his usual health, but at perhaps one or two a.m. he wakens with severe epigastric discomfort, which may result in copious vomiting of sour-smelling fluid containing free hydrochloric acid. Instant relief may be afforded by the vomiting. He may have repeated attacks of this type and then remain well for months. The tendency, however, particularly if there is an organic basis, is for the condition to merge into the chronic type.

The symptoms of chronic hypersecretion may be very similar to those of simple hyperacidity, but I am of opinion there is more nocturnal discomfort than in hyperchlorhydria. Here again, the "hunger-pain" may be a feature, which is relieved by taking a biscuit. The other symptoms of hyperchlorhydria already enumerated, are usually present in hypersecretion so that there is really no pathognomic sign to differentiate the two conditions as far as subjective symptoms are concerned.

The physical signs of hypersecretion, qua hypersecretion, are also indefinite, though if there is a primary disease, such as ulcer, present, the physical examination may provide signs pointing to this, but it would neither confirm nor deny the presence of gastro-succorrhoea. It is only by careful investigation through chemical analysis that the diagnosis can be established beyond doubt.

Chemical Analysis: On passing a tube into the fasting stomach in a case of chronic hypersecretion, the tube will evacuate a considerable quantity of light greenish-yellow fluid which gives the usual reaction for gastric juice. If there is organic stenosis also present, the fluid may be mixed with some undigested food. If, through further investigating by the fractional method of gastric analysis, after three hours there is still a bulky residue which chiefly consists of clear juice and with very little of the test-meal, it seems fair to assume that hypersecretion is definitely present.

Apart from the useful information one may derive from a fractional test regarding the presence or otherwise of hypersecretion, the nature of the acid curve obtained may be of real importance in suggesting the possible presence or otherwise of hypersecretion, the nature of the acid curve obtained may be of real importance in suggesting the possible presence of juxta-pyloric ulceration. Over a year ago I had an interesting case (details on p.97) which clinically might have been one of ulcer or early carcinoma, but which on the first X-ray investigation showed no organic abnormality. The chart of the fractional analysis yielded a "plateau" curve suggesting an ulcer in the duodenum or pyloric region, and this was confirmed by a later radiological examination.

Diagnosis: This I do not consider to be easy in spite of the assertion of certain writers. Soltau Fenwick,(32) for instance, states "chronic hypersecretion is a complaint that is easy to recognise if attention is bestowed on certain characteristic symptoms of which the following are the most important; (i) After many remissions the dyspepsia has become permanent and defies the usual methods of treatment; (ii) Pain or discomfort ensues regularly 2 or 3 hours after a meal, or at other times when the stomach is almost devoid of food; (iii) Vomiting often occurs at the crisis of an attack of pain and is especially frequent between 1 and 2 a.m. (iv) Appetite increased, and severe thirst may be experienced after vomiting; (v) Urine scanty with

excess of phosphates and deficient in chlorides; (vi) Patient steadily loses flesh and strength and may exhibit marked cachexia. (vii) Stomach is dilated and signs of pyloric stenosis or pylorospasm are present," etc. I suggest that none of these signs are pathognomonic of the derangement, and even if they are all present in a case it would be inadvisable to call it hypersecretion without (a) ascertaining that there is excessive fasting juice and (b) excessive secretion of juice after say two and a half hours from the administration of a test-meal. Where these conditions are fulfilled one can definitely assert the condition is not one of simple hyperchlorhydria or neoplasm, but that hypersecretion is present.

Where there is no sign of gastric or duodenal ulcer to account for the gastro-succorrhoea, the possibility of a diseased gall-bladder or appendix should be remembered, as, having diagnosed hypersecretion, it is of still more importance to ascertain the factor which may be producing the excessive secretion.

Prognosis: In the intermittent type, where no organic disease is present the outlook is good. The chronic variety may be benefitted by treatment, but a guarded prognosis should be given as relapses are common, especially where organic disease is the basis of the hypersecretion.

Treatment: In simple intermittent hypersecretion,

where the patient is neurotic, much can be accomplished by regulating the mode of life and removing as far as possible mental strain and excitement. I usually advise a diet based on that for simple hyperchlorhydria. Where vomiting is a feature lavage is most helpful. I have found washing out the stomach with sodium bicarbonate, two drachms to the pint, quite satisfactory. Where there is a high percentage of free hydrochloric acid, alkaline medication alternating with belladonna, or combined with it, helps to limit the secretion of juice and lower the acidity.

Where the case is chronic, and an organic cause has been located, the treatment must necessarily be influenced by the site of the primary disease, though even here lavage may afford considerable relief.

Case History.

J.B., male, aged 59; unemployed engineer; single. Family history not significant. As regards habits, he was a fairly heavy tobacco smoker: 3-4 oz. being smoked per week. At the week-ends, when working, he was in the habit of taking beer, and occasionally spirits, though not to excess. He had been a healthy man apart from attacks of epigastric discomfort which had occurred intermittently over a period of years. For the past two months the attacks were more frequent and severe. Pain occurred about one and a half hours after taking food, and this was to

some extent relieved by taking more food. He was often wakened in the night with pain, and vomiting was occasionally present. When this took place it was copious, but he secured relief. His appetite was poor, and latterly he was afraid to eat, knowing that epigastric pain would result. Thirst was not a feature. Acid eructations frequent. Constipation present.

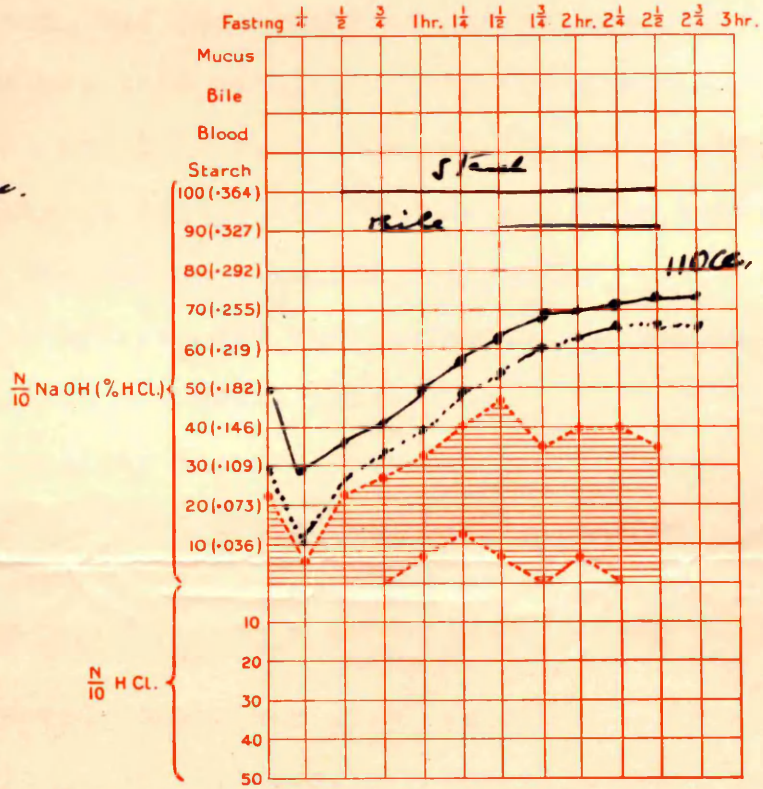
General appearance on 23.11.31 was that of a man somewhat pale and emaciated looking, though he stated he had always been sallow and that his weight had not appreciably altered for years. Height 5 ft. 8 ins; weight 8 st. 10 lbs. Teeth showed marked caries associated with pyorrhoea. Tongue moist but furred. Some degree of arteriosclerosis. Cardiac sounds of good quality, though second aortic sound accentuated. Respiratory system negative. Abdomen showed panniculus spare. Definite tenderness on palpating over epigastrium, particularly in middle line. Lower abdominal region negative to palpation. On 24.11.31 he had an Ewald test-breakfast with the following result: free HCl 0.31 per cent; total acidity 0.35 per cent. The quantity withdrawn in the evacuator was 200 cc. and no doubt more could have been secured. There was no evidence of gross food retention, in that currants swallowed the previous evening were not present. No occult blood in gastric contents or faeces.

In view of the definite hyperacidity and localised

GASTRO-INTESTINAL ANALYSIS.

Name of Patient J. B. Ward ~~Bed~~

1. FRACTIONAL TEST-MEAL. Date 10-12-31



Fasting Juice.

Volume. 80 cc.

Cells.

One Hour Fraction.

Free HCl.

Active HCl.

Total Chloride.

——— : Total Acidity

..... : Free HCl.

The shaded area represents the limits for free HCl. in 80% of normal people, and average rate of emptying (2-2½ hours).

——— represents free HCl.

——————— represents total acidity.

Summary.

2. FÆCES.

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pain he was sent to the Infirmary for further examination, as peptic ulcer was suspected. His general appearance had suggested to me the possibility of a malignant neoplasm, but the result of gastric analysis appeared to exclude this condition, but the question of ulcer remained.

On 15.12.31 I received the X-ray report which was negative except for "ptosis and poor tone of stomach." He was put on alkaline medication, the diet for hyperchlorhydria, and was instructed to return to the Infirmary in from 4-6 weeks time.

After the one-hour test, and before receiving the X-ray report I carried out a fractional test with the following result: fasting juice withdrawn 80 c.c. After two and three quarter hours, when most of the meal had been removed, there was still an appreciable quantity of hyperacid juice (110 c.c.). I append a chart which readily shows the appearance of the acid curve. The condition, then, was definitely one of hypersecretion, though the exact cause of this was still in doubt. The radiologist's report did not suggest ulcer as the primary factor, and there were no physical signs pointing to gall-bladder or appendix disease. Gastric lavage was carried out with beneficial result, and he remained fairly well until May 1932, when he had a recurrence of the acute symptoms. With lavage etc. he again improved but relapsed in June. He was again X-rayed (17.6.32) and the physician wrote to me:

"X-ray shows what it missed before! Large chronic ulcer in lesser curvature near pylorus. This new finding bears out the fractional finding of a plateau curve".

The patient was admitted to a surgical ward shortly afterwards, but as his symptoms were then in abeyance he was kept in bed on a gastric ulcer diet and was later discharged without operation. It is now a year since he was in hospital, and he is keeping well, though still on a post-ulcer régime.

Discussion: This case seems interesting because (i) From the results of the one-hour test and first X-ray examination a diagnosis of simple hyperchlorhydria may have been made, with some justification, as the clinical symptoms were those of hyperacidity, but the occasional copious vomiting and the tendency to localised pain suggested further investigation. (ii) The fractional test proved there was a definite degree of hypersecretion, and the type of acid curve suggested juxta-pyloric ulcer. The second X-ray examination succeeded in comfirming this. (iii) In this case it was the fractional test which was the most important factor in arriving at a diagnosis.

Hypochlorhydria.

I do not propose to devote much space to this condition as, where it is quite definitely short of complete achlorhydria, it may be of little clinical significance. Indeed, I have not satisfied myself that hypochlorhydria, per se,

does produce any characteristic symptoms. It is admitted that a total acidity of under 40 (Ewald scale) is below the average, and yet this may be the normal for the individual. In cases of gastric neurasthenia I have often found a low total acidity, but in this condition the findings are so variable that it would appear to be illogical to attribute any subjective symptoms to diminished gastric acidity.

There is little doubt that emotional disturbances can easily produce some degree of inhibition as regards gastric secretion, so where a condition of subacidity is found in a neurotic subject, an endeavour is made to allay the patient's anxiety. As a routine, dilute hydrochloric acid is prescribed, in a dose ranging up to a drachm, to be taken in half a pint of water or orangeade, with meals. It is realised that such dosage is inadequate as regards appreciably affecting the gastric acidity, but nevertheless I have found in many instances that the patient has felt an improvement in his general condition. Quite possibly the mere administration of some therapeutic agent in such a type of patient has a definite psychological value.

Achlorhydria and Achylia gastrica.

It may be as well to state that these two terms have been used by some writers indiscriminately, with resulting confusion. The conditions are not actually synonymous, although both indicate an absence of free hydrochloric acid in the gastric juice. Achlorhydria refers only to an absence of free HCl:

but in achylia gastrica there is a superadded deficiency of enzyme-like substances acting in an acid or neutral medium, although, strictly speaking, it implies a cessation of all gastric secretion. It therefore follows that all cases of achylia gastrica have achlorhydria, though the converse may not hold.

Absence of free HCl after a test-meal is not incompatible with good health, but the condition may not be a true achlorhydria, for, as Hunt (47) says this may be the result of using a meal with insufficient stimulating properties, or to lack of care in the examination. Probably the most powerful stimulus of gastric secretion we possess is histamine, and the use of this substance has shown that quite a high proportion of cases of so-called achlorhydria are in fact false or apparent only, and hydrochloric acid is quite freely excreted after the subcutaneous injection of 1 c.c. of histamine in a strength of 1:1,000. In true achylia gastrica no such result is obtained.

Whilst realising, then, that there are two forms of anacidity, I propose to deal chiefly with achlorhydria as, from the general practitioner's point of view it is not so rare a condition as achylia, and it is more readily recognised from the simple tests which are at his disposal.

Etiology: There is no doubt that many cases of achlorhydria are quite benign in character: they may be discovered accidentally, the patient having no gastric symptoms, or it may

be a transitory abnormality in the gastric secretion. On the other hand, a persistent achlorhydria may be the accompaniment of a gastric carcinoma, or, if the case is one of pernicious (Addison's) anaemia, a true achylia is generally recognised as being a constant feature of this blood disease. It will therefore be seen that the mere presence of achlorhydria may be a symptom of little significance, or it may point to some serious pathological condition: hence the importance of getting a full history combined with a careful clinical examination.

In simple achlorhydria, nervous disturbances appear to play a prominent rôle. It is a common accompaniment of neurasthenia and profound nervous exhaustion. Indeed, in a neurotic patient, the morbid fear of the stomach tube may suffice to inhibit or curtail the flow of gastric juice, though once the initial dread has been overcome a subsequent gastric analysis may reveal a normal or hyper-acidity. I have satisfied myself that such may actually occur, so that it may be inadvisable to lay stress on a first gastric analysis carried out on a nervous patient, where an apparent achlorhydria has been obtained.

It may seem rational to suggest that where neurotic patients habitually show an absence of free hydrochloric acid, such cases are probably below "par" in other ways: subnormal blood pressure, etc., and therefore the achlorhydria or achylia is simply a constitutional sign of low vitality, and that we

are not dealing simply or essentially with a gastric case. Such an explanation, however, does not cover all cases of simple achlorhydria, as for example, those described by Bennett and Ryle, before quoted, where the individuals were apparently healthy and quite normal in every other way.

In recent years much literature has accumulated regarding the association of achlorhydria with anaemia, but even now a complete explanation of this phenomenon is required. No doubt the presence of free hydrochloric acid in the gastric juice is almost sufficient to negative a diagnosis of pernicious anaemia, so constant is a true achylia gastrica present in this condition. The evidence now seems definite that the achlorhydria precedes the development of the anaemia, and is not secondary to it as some writers have suggested. Faber,[48] in 1913, was the first to bring conclusive proof that the achylia is primary and not a result of the anaemia. He described 3 cases in which the blood was normal - haemoglobin percentages 100, 95, and 90 respectively - when the presence of achylia was discovered. Ten, seven, and three years later typical pernicious anaemia began to develop. The achlorhydria persists when the anaemia has almost or completely disappeared. Indeed the achylia is far more constant than any other symptom of pernicious anaemia, not excluding the anaemia. With the advent of the histamine test for true achlorhydria, the interesting suggestion has been put forward by Nye and Sippe[49] that it is highly probable that its earlier detection and treatment would have considerable

preventive value. At the same time it must be recognised that the mere prescription of dilute hydrochloric acid has no therapeutic effect on pernicious anaemia when it is established.

There is a less serious type of anaemia, called microcytic or achlorhydric, which of recent years has been found to be not uncommon in middle-aged women. Achlorhydria is present in these cases. D. T. Davies,(50) L. G. Witts,(51) and others have stressed the fact that the gastro-intestinal symptoms may be more severe in this type than in Addison's anaemia. (It is important to remember this type, as it may show a glossitis as well, so that one might be misled into considering it a case of pernicious anaemia: hence the advisability of a careful blood examination. As is now well known achlorhydric anaemia responds to iron medication).

Gastritis, through prolonged irritation of the gastric mucous membrane, may produce achlorhydria. Alcohol may thus indirectly cause anacidity through producing a primary gastritis.

Symptoms: These may be entirely absent, but I find that it is more usual for the patient to complain of some epigastric discomfort during the day-time, though rare to have any nocturnal symptoms: in this respect differing from hypersecretion where pain and even vomiting may occur during the night.

It is generally recognised that the stomach empties

itself more rapidly in cases of achlorhydria or achylia. Indeed, in one case I had I did not find any fluid in the organ after one hour had elapsed from taking a test-breakfast. Subsequently the case was found to be one of achlorhydria with an extremely active stomach.

Diarrhoea is a fairly frequent symptom, though it is not necessarily due to a hypermotile stomach. An explanation which seems equally possible is that intestinal putrefaction is more common in these cases, as the antiseptic action of hydrochloric acid is not present, and in consequence gastro-intestinal irritation is more likely to be present, and this may set up "gastrogenous" diarrhoea.

Paradoxical though it may seem, I have found patients complain of acid eructations and occasional pyrosis with achlorhydria so these symptoms form no criterion as to the degree of acidity present, and certainly they do not necessarily suggest a hyperchlorhydria.

Loss of appetite is often present, and thirst is a feature where there is much diarrhoea.

Gastric Analysis: This is essential for diagnostic purposes. In simple achlorhydria the findings are characteristic: there is absence of free hydrochloric acid and lactic acid, and no evidence of fermentation or putrefaction. The quantity of fluid is much reduced and it may be difficult to extract. The particles of toast may be undigested, and consequently they are apt to block the tube. The findings are

different from those of chronic gastritis or advanced gastric carcinoma, in both of which diseases there may be no free hydrochloric acid, but in early malignant disease it may be quite difficult to distinguish, as far as the findings from chemical analysis go. This will be considered later.

Prognosis: Rational treatment of a neurotic case of achlorhydria may soon result in an improvement or cure. On the other hand, in constitutional achlorhydria the anacidity will persist despite the administration of dilute HCl. In associated chronic gastritis, anaemia, or neoplasm, the prognosis of the case is dominated by the primary disease.

Diagnosis: Rests mainly on gastric analysis. In chronic gastritis there is excessive mucus present, and if the specimen is allowed to stand there may be evidence of fermentation. In malignant neoplasm there may be signs of putrefaction, occult or visible blood, and lactic acid. But in early malignancy it may be impossible to be certain of the diagnosis. The correct procedure in such a case where it is practicable is, to carry out a fractional test and have an expert radiological examination.

Treatment: At the risk of appearing tedious it must be again mentioned that here, as in other gastric derangements, it is of the first importance to try and relieve any anxiety or worry the patient may have, and it is time well spent in endeavouring to ascertain if any mental apprehension exists.

If found and removed the treatment of simple achlorhydria is more likely to result in success.

Dietetic measures are important, and best results seem to be obtained where the intake of animal protein is restricted, at the same time allowing a liberal quantity of carbohydrates. Where butcher meat is taken it should be well masticated before swallowing. I usually give a diet sheet to the patient based on the following:-

Breakfast: Orange juice or grape fruit, withoug sugar; 4 oz. fine oatmeal or Cream of Wheat, well cooked, with cream or milk, but without sugar. One egg cooked any way except fried. (If this upsets the liver, white fish may be substituted). Toast and butter. One cup of tea.

Dinner. Small quantity of lamb or mutton chop, or 4 oz. of boiled chicken; one or two oz. of potato or vegetable purée. Baked apple or tinned pear. A glass of water.

6 p.m. Egg or fish if not taken at breakfast. Toast and butter. One cup of tea, or preferably cocoa or coffee, with milk.

10 p.m. A glass of "orzone". (Emulsion of olive oil and Horlick's malted milk).

Where the patient is not out working, I usually suggest a glass of water about 11 a.m., and in the afternoon a

cup of weak tea and a plain biscuit.

Condiments are allowed in moderation with the object of stimulating the gastric secretion, and consequently they may have a beneficial effect in cases of simple achlorhydria.

I find milk is usually well tolerated, so that milk puddings may be given with benefit as a change from fruit at dinner-time.

Medicinal treatment: On the whole I have not satisfied myself that the administration of dilute hydrochloric acid is of much benefit in effecting an increase in the gastric acidity, even when doses of a drachm or over are prescribed. In spite of this, however, it is my practice to give it as a routine in such cases to begin with, as it sometimes appears to afford relief from the indefinite symptoms, though I place greater reliance on dietetic measures. Where there is diarrhoea, dilute acid may produce a symptomatic cure when nothing else will.

The treatment of an associated anaemia or subacute combined degeneration of the spinal cord is outside the scope of this paper, but there is an interesting practical point brought out by Wilkinson(52) regarding the modern treatment of Addison's anaemia by desiccated hog's stomach as compared with liver diet combined with hydrochloric acid and pepsin mixture. (As this has to do with the dyspepsia found in these cases of anaemia I suggest it is appropriate to mention it here). He found that in a series of 208 cases examined by

fractional gastric analysis, there were definite peptic activities in all the samples after a desiccated stomach gruel meal, until the stomach had emptied. No peptic activities were obtained after the gruel plus liver extract. This observation is of considerable importance in its bearing on the deficient digestion and gastric disturbances in pernicious anaemia, and when the frequent occurrence of flatulent dyspepsia in this condition which is relieved by a liver diet and HCl and pepsin mixture is recalled (Wilkinson and Brockbank).(53) It is seen, therefore, that the strong peptic activity of an active hog's stomach used in the treatment of pernicious anaemia should serve as a useful adjunct to the haemopoietic properties of the product - this has been confirmed clinically, since the pepsin-acid mixture can usually be discontinued with this form of therapy.

Case History.

M.B., female, aged 35; married; one child; highly neurotic temperament. Family history negative. Past history: measles in childhood, otherwise had been remarkably well. Had one confinement four years previously, after which she had a uterine prolapse. Operation for this in October, 1931. When seen some months after this she complained of intermittent discomfort in the epigastric region, the pain sometimes going through to the back. The pain was uninfluenced by the taking of food. Her weight had been stationary for the past three

years. Her appetite was capricious, but usually not very good. Bowels regular.

Physical examination revealed a medium-sized woman of healthy appearance but with an "anxious" expression. She admitted she was inclined to worry over trifles. Examination of chest negative. Pulse 100, but she was somewhat excited when it was counted. Blood pressure 130/70. No evidence of anaemia. Abdomen on inspection and palpation failed to reveal any abnormality beyond indefinite epigastric tenderness, which was slight and unlocalised. Gastric analysis after test-breakfast showed absence of free hydrochloric acid and a total acidity of 4. No lactic acid or blood present. No excess of mucus. An examination a week later gave a similar result. She did not submit to X-ray investigation as she wished to try the effect of treatment.

The case was regarded as one of simple achlorhydria, and she was put on a diet similar to that already mentioned, and acid. hydrochlor. dil. was prescribed in drachm doses to be taken in half a tumbler of water with meals. After a holiday she improved and up to eighteen months after treatment she had secured symptomatic relief.

It will be noted her symptoms originated or coincided with her operation, and she admitted she had been worried at the prospect of this, though anxious to get the prolapse cured. It may be suggested, therefore, that a functional gastric derangement was caused through the fear of operation. Whether

the gastric juice is now being secreted normally I cannot say, but she felt so much better when last seen that she was unwilling to undergo the slight discomfort of the stomach tube again.

(c) SENSORY NEUROSIS.

General Considerations.

It is the cases which comprise this group which often give the practitioner a considerable amount of trouble, both from the diagnostic and therapeutic point of view. In no section of the classification I have adopted is its arbitrary nature more apparent than in this one, as in reality many of the patients suffer from some derangement of the motor and secretory functions in addition to sensory disturbances. These cases rarely reach a hospital ward, and, except perhaps for an X-ray examination they may not attend the out-patient department, so that there is little opportunity for the medical student to carry out a clinical study of a group of cases with which, as a general practitioner, he will frequently come into contact. If he is able by careful examination, clinical history, and gastric analysis to exclude organic disease, there is much he can do to alleviate the suffering of a considerable percentage of those gastric cases which help to comprise this group.

At the outset it is admitted that there may be an organic basis for the symptoms, and every endeavour must be made to ascertain whether or not such is the case, as otherwise treatment directed to the functional disturbance only, will be unproductive of a cure.

Various names have been given to nervous dysfunction of

the stomach: gastric neurasthenia, nervous indigestion, nervous dyspepsia, etc., and provided such terms are not used to cloak an insufficiently established diagnosis they are all equally good, for, in spite of the statement of certain writers that there is no such condition as nervous indigestion, I feel convinced that there is a group of functional dyspepsias which can be justifiably so classified. It is the privilege of the general practitioner to be able to follow up particular cases over a number of years, so that he is able from personal observation to test the result of diagnosis and consequent treatment, and many times the writer has been able to prove, at least to his own satisfaction, that conditions which in some ways simulated organic disease of the gastro-intestinal tract were in reality simply manifestations of that protean functional derangement called nervous indigestion.

Just as gastric secretion can be induced by pleasurable emotions, so also it can be stopped by unpleasant or painful ones. Cannon, from whom I have before quoted, speaks of the ancient ordeal used in India to pick a thief out of a group of suspected persons. Each was given a bowl of rice, and the criminal was detected by his dry mouth, and his slowness in salivating and swallowing the food. Without presuming any such criminal intent, it would seem, therefore, that it may be appropriate for the physician to warn his patient against eating at those times when the mind is so distracted that there is no possibility of the development of psychic juice. Alvarez,(54)

quoting Pavlov, states that the soup at the beginning of the meal with its strong tendency to excite secretion in the stomach saves many an inattentive and uninterested diner from indigestion. As Brillat-Savarin has said "Animals feed, man eats, the man of intellect alone knows how to eat."

The physician who would successfully treat functional gastric neuroses must not omit to regard his case from the psychological aspect, as it is largely by gauging the mental status of his patient, and noting how he reacts to the everyday occurrences of life that a satisfactory basis for treatment can be elaborated. The type of individual who is habitually overwhelmed with worry, real or imaginary, who never seems just able to cope with the wear and tear of life, is the type of patient who, sooner or later, is apt to suffer from gastric dysfunction, and the most likely form is a sensory neurosis.

Just because there is so often no organic basis for the symptoms it does not follow that the patient's sensations are imaginary and therefore of no consequence. Many of these cases suffer intensely and if they can be cured the benefit to themselves is just as great as if they had had an acute appendicitis which had been successfully treated by surgical procedure.

Before considering gastric neurasthenia in some detail there is one point regarding the physiology of the stomach which has some bearing on the origin of sensory disturbances. It is a physiological fact that the mucous membrane of the stomach is insensitive to tactile stimuli, and also to heat and

cold, and yet one may have gastric pain, which must be produced by some stimulus. It is recognised that tension is this stimulus, and pain is produced when there is an undue increase in the intra-gastric tension. This has some bearing on dietetic treatment, as care should be exercised not to overload the organ with too large a quantity of sloppy food.

Gastric Neurasthenia.

Etiology: Usually there is a constitutional or hereditary factor, and careful inquiry should be made regarding the family history. To find out the actual cause, some time may require to be spent in order to ascertain whether the origin of the symptoms coincide with, or followed upon, some private anxiety, worry, or depression. Not infrequently a first consultation fails to bring out a predisposing cause, but subsequently, when the physician has the patient's complete confidence some such condition as masturbation, sex jealousy, financial dilemma, etc., may be found to have an important etiological bearing on the gastric symptoms. As to why the stomach should be the particular site of discomfort it is unnecessary to surmise, as it is common knowledge that where there is any mental worry it is nearly always productive of "indigestion" sooner or later. Hurried meals, eaten under strain or whilst mentally preoccupied, may be sufficient to produce symptoms.

After any debilitating disease, and particularly after

influenza, it is not uncommon to find the patient developing into a state of neurasthenia, and in many such cases it is of the gastric type. (It is hardly necessary to add that I am not referring to so-called gastric influenza, which of course is simply a manifestation of the acute disease). I have usually found definite secretory abnormalities in these cases, generally hypochlorhydria, but owing to the more pronounced nervous symptoms I prefer to include them in the group of sensory neuroses. A further point I would stress in support of this grouping is, that even when the gastric acidity has returned to normal proportions, the nervous symptoms may remain in spite of no organic abnormality being present. Such cases are, I suggest, really types of nervous exhaustion, and the ultimate prognosis is good.

Just as nervous symptoms may become grafted on to a true organic condition and persist after the latter has disappeared, so the converse may hold, and a primary nervous disorder of the stomach may, through loss of appetite and secretory abnormality, give rise to an organic catarrh of the viscus. It will thus be evident that it is often a matter of extreme difficulty to ascertain whether or not we are dealing simply with a case of gastric neurasthenia or whether there is not a structural lesion of the stomach present as well. Such cases may require several examinations before the matter can be definitely settled.

Hurst,[55] who is always sceptical regarding functional

dyspepsias, separates neurasthenic dyspepsia into two varieties, which he says can only be recognised with certainty with the aid of X-rays and gastric analysis: the asthenic, occurring in an individual with a stomach with less than the average tone and secretion, and the hypersthenic, occurring in one with a stomach with more than the average tone and secretion.

I submit that it is hardly possible or indeed necessary to have every suspected case of gastric neurasthenia X-rayed in the first instance, and, as many cases clear up satisfactorily without this investigation, the classification as advised by Hurst is not quite practicable from the ordinary physician's point of view.

Symptoms: I feel there is little to be gained by enumerating the symptoms of gastric neurasthenia, as not only are no two cases the same, but the symptoms vary in a particular case. Indeed this latter point may be of diagnostic value: the very variability of the symptoms from day to day tends to arouse one's suspicions that there is at least a neurasthenic element in such a patient. Regarding pain: there are a few points which I have found helpful. It is often described as being very severe, but on palpating the region complained of, it is remarkable the amount of manipulation tolerated in spite of it being so "severe". The physician is then inclined to discount some of the patient's history in so far as it relates to gross discomfort. (Genuine severe pain is so rare that

Hutchison (56) states it almost negatives a diagnosis of nervous indigestion). Further, it is unusual for the patient to be wakened by the pain, though he may state he cannot sleep at night, but this should not be regarded as a case of post hoc ergo propter hoc.

Vomiting is not usually a feature though nausea is common; but even when the former symptom persists, however, it does not necessarily rule out a diagnosis of nervous dyspepsia. I have a patient, W.A., male, aged 30, who has been under my care for a number of years. His appendix was removed five years ago, and the following year his abdomen was again opened and a gastro-enterostomy performed for a duodenal ulcer. He remained well for a year and then complained of daily vomiting which has continued more or less since. He has been X-rayed and examined with test meals with negative result as regards the presence of adhesions or another ulcer. In spite of his history of daily vomiting he has kept his weight and looks fit. After months of observation he is regarded as a chronic neurasthenic case, and the surgeon considers a further laparotomy quite inadvisable. I am convinced that this patient will continue to have gastric symptoms, and that no surgical treatment will cure him: he has a fixed idea which is almost unchangeable, and one is almost inclined to agree with Ross (57) that such a case is a type showing a true delusion. Certainly the patient in question would always be able to state that he has had organic disease; but it is the considered opinion of

those who have examined him since, that it is a super-added gastric neurasthenia which has been left behind.

Loss of weight is not necessarily a sign of organic disease, as I have had definite cases of nervous dyspepsia which showed this symptom. Where it is marked, however, it is advisable to regard the case as one of structural disease until a full examination has been carried out.

Gastric Analysis: is not of considerable value as far as establishing the diagnosis goes, but it is of value in showing any gross secretory abnormality. Where the result varies markedly in the same case within a few days it is suggestive of nervous dyspepsia. On the whole, my cases of gastric neurasthenia tend to show a hypochlorhydria rather than excessive acid concentration.

Physical Signs: These may be absent, and in any case there is no pathognomonic sign. If definite pain is complained of over a spot in the epigastrium I endeavour to locate it accurately in order to see whether it is in a similar position when a subsequent examination is made. Usually the site of the pain has inexplicably altered its position. This tendency to have a variable pain-site may be helpful in arriving at a diagnosis when taken in conjunction with other symptoms pointing to neurasthenia.

Diagnosis: The information gained by a thorough study of the history of the case may be of great value, and it should

be emphasised that the patient's mentality demands almost as much consideration as his physical condition. Often the physical examination and the result of gastric analysis are negative: hence the importance of the history.

The main points on which I would base a diagnosis of gastric neurasthenia are: the variability of the subjective symptoms from day to day; the inability to find on physical examination any gross signs of disease in spite of the series of complaints made by the patient; the presence of a neurotic disposition. I realise that frequently the patient is conversant with a particular disease, such as appendicitis, or cholecystitis, and that he or she may give a description which may and often does simulate a particular organic condition, but the physical examination does not usually bear this out. The real difficulty arises where there is a degree of nervous instability superimposed on an organic disease, and in such a case it may be impossible to reach a final diagnosis without utilising all the modern aids at our disposal.

Not content with diagnosing a case as one of nervous indigestion, there is nowadays a tendency to subdivide this group into three divisions: the hypersthenic, due to vagus stimulation; the asthenic, due to sympathetic stimulation; and the psychogenic, where there is supposed to be a central origin, i.e., where the digestive organs are apparently quite normal in structure and function, but where indigestion is constantly complained of. Such a classification is adopted by

Hunt, (58) but while theoretically a case may be made out for such a subdivision, I submit it is of little practical significance, as in actual practice it is rare indeed for a case to exhibit the characteristics of one type only, and from a diagnostic point of view the functional condition of nervous dyspepsia is too variable in its manifestations to attempt artificially to subdivide it.

Prognosis: Some improvement can usually be looked for, but relapses are not uncommon, as we are generally dealing with an unstable nervous system. When the condition follows an acute illness, such as influenza, a cure may result which is permanent.

Treatment: The essential of treatment is neither diet nor drugs, but psychotherapy, and the method I adopt, having established the diagnosis, is (i) to reassure the patient that he has got no serious organic disease; (ii) to tell him that his condition is quite a common one, and very amenable to treatment provided that he gives his willing co-operation.

If the patient has worries I endeavour to get him or her to discuss them as the mere unburdening of the patient to one who is sympathetic is of some value in assuaging any mental anxiety which may be present. Where it is simply a case of the dread of organic disease, it is necessary for the patient to have full confidence in the medical adviser, and then a categorical denial of any growth or ulcer being present should tend to bring about an improvement. Where there is financial or

other embarrassment causing a nervous derangement, it is my custom to advise a light, easily digested but nutritious diet and give a bromide mixture. Here, of course, it is not possible to remove the cause, but by prescribing sedatives some improvement may be looked for, and there is always the hope that the depressing factor may be removed later.

Where there is no sign of improvement in spite of suggestion, etc., I have sometimes found gastric lavage to be of benefit, even when there was no sign of secretory abnormality or gastritis. I can only think that in these cases the psychological impression that "something is being done" unconsciously produces a feeling that a cure is being effected. I admit that such treatment is unorthodox, but I can plead that in certain cases the result has justified the procedure. Needless to say this is not a method of routine, but rather an empirical attempt to effect an improvement where other forms of treatment have failed.

If possible, a change of environment is most helpful. I have found this of use particularly in the case of working-class mothers who are exhausted with home cares and worries. A short stay at a convalescent home sometimes yields good results.

Diet: This is not usually altered unless there are any gross errors noted. At the same time it is often tactful to give some specific advice as to meals, as many patients of

the type under consideration seem to expect definite guidance in this matter. Where there are secretory abnormalities I usually give a diet-sheet for the type present. The administration of an emulsion of Horlick's malted milk and olive oil at bedtime is generally well tolerated, and it seems to help to lessen the tendency to insomnia so often present.

Drugs: It is usually my practice to give a bitter tonic in these cases, as apart from its intrinsic value the average neurotic patient feels that some form of medicinal treatment is necessary, and I have found it advisable to act accordingly. A mixture of nux vomica, bromide and gentian is frequently all that is necessary. On the whole I have found alkalies to be disappointing. Indeed, where they result in marked improvement, the question of organic disease being present should be reconsidered. Where there is a subacidity I include in the bitter mixture, dilute hydrochloric acid (dosage up to one drachm), with pepsin, and this is taken in half a glass of water with meals.

I now propose to give a few representative case histories, as by this means it is perhaps easier to show the difficulties one encounters in actual practice. The first one, I think, is interesting from the diagnostic point of view, and although not perhaps a true gastric neurasthenia, I feel it is worth recording if only to show how one may be misled by taking a history at its face value.

Case Histories.

CASE 1. Mrs M., aged 38; housewife; married 8 years; no family. Family history of no significance except that the patient and one of her three sisters showed evidence of simple goitre. I saw this case for the first time on 28.11.32. She complained of epigastric discomfort and occasional vomiting of three weeks' duration. She was quite definite as to the origin of the sickness: she was in the enclosed cabin of a motor boat on Loch Lomond and there was an objectionable escape of paraffin or petrol fumes which she endured as long as possible, until finally she had to give up and request to be put ashore, where she vomited. Since then she had had almost constant nausea and occasional vomiting. No previous history of stomach trouble, and her health had been satisfactory apart from some menstrual irregularity. Her previous two periods, however, had been at normal intervals. Regarding the goitre, she said it had been the same size for many years. She admitted she had always been of a nervous disposition.

Physical examination revealed a tall gaunt woman somewhat muscularly developed and of sallow complexion. Her weight-height ratio was satisfactory, and there was no evidence that she was losing weight in spite of the vomiting and nausea. No tendency to exophthalmos. Abdominal examination was negative apart from slight epigastric tenderness not localised to one spot. The respiratory and circulatory systems

appeared to be functioning normally. The pulse rate was 100, but she was somewhat excited and nervous at the time, so no significance was attached to this. She stated that she felt that the paraffin was still in her system, and though I tried to dispel that impression, I fear she was scarcely convinced. Urine and faeces were normal. No sign of organic disease of the nervous system.

A tentative diagnosis of nervous dyspepsia was made, a bitter sedative was prescribed, and she was asked to report within a week. This she did, and stated there was no improvement in her condition. A test meal was given the next day with the following result: free hydrochloric acid 0.0365 per cent; total acidity 0.09125 per cent; a definite subacidity. The Gunzberg test for free HCl was positive. There was no lactic acid or occult blood, and no evidence of gross food retention. With dilute acid and pepsin added to her mixture she appeared to improve for some days, but she gradually drifted back to the old symptoms. I said that had she been recently married and missed a menstrual period, her symptoms would have strongly resembled those of early pregnancy. She admitted she had never even thought of such a possibility. However, as she was not improving, I examined her breasts and was surprised to find fluid in them on applying digital pressure. The areolae were dark, but she insisted they were always that colour. Also, she had not missed any period, and in fact had menstruated since the

origin of the gastric symptoms. The result of a vaginal examination was indefinite.

As the case was certainly not a normal one I sent her to the Samaritan Hospital for Women. The uterine examination was inconclusive, and she was kept in for a chloroform examination. This was done and the surgeon thought the uterus was enlarged and probably gravid. He told her candidly that though he thought she was pregnant, he was not positive, and he suggested that I should arrange to have the Zondek-Ascheim test performed. I therefore sent the urine away for this purpose, and the result was positive for pregnancy, and later she proceeded to term without further abnormality.

Discussion: It may be argued that an examination of the breasts at the outset would have rendered much of the investigation unnecessary, or at any rate would have suggested the possibility of pregnancy. That is admitted, but when a woman is approaching middle-age, who has been married for several years without conception having occurred, and who has had no recent amenorrhoea, consults her medical adviser regarding gastric pain and nausea, I am of opinion that most physicians would not give the question of the possibility of pregnancy a thought to begin with. Again, the patient was so emphatic regarding the cause of the primary indisposition that this served as a further misleading factor.

In any case, it was not a typical "morning sickness" of pregnancy, and the fact that there was both a secretory derangement and definite epigastric discomfort, leaves me with the impression that there was a functional dyspepsia present which may or may not have been entirely dependent on the patient's pregnant condition.

CASE 2. Mrs. E., aged 40; married; 4 children. Family history negative. Past history: no serious illnesses. She had always been of a nervous, excitable nature, and had occasionally had attacks of "indigestion," but for some months past attacks of epigastric discomfort and nausea were becoming more frequent. No vomiting. The discomfort had no definite relation to the intake of food, but she thought it was somewhat worse within half an hour of taking a meal. Flatulence was troublesome. She admitted she was inclined to worry over trifles.

Physical Examination: She was a woman 5 ft. 6 in. in height and 8 st. 10 lbs. weight and of healthy appearance. (Her nervous nature was known to me as I have attended the family for several years.) Tongue was moist and clean, and gums in good condition. False teeth. Respiratory and circulatory systems negative. During the abdominal examination the patient was troubled with belching of flatulence, which I considered to be largely due to aerophagy. No tenderness found on palpating the abdomen. On 16.3.32 she had a test

breakfast with the following result: free hydrochloric acid 35; total acidity 52. No lactic acid, no excess of mucus, and no occult blood. Urine and faeces negative.

A diagnosis of gastric neurasthenia was made, and the patient was told that there was no evidence of any organic disease to worry about, and that with rest and a plain nourishing diet she would soon be all right. Orzone was suggested as a bed-time drink. It is now over a year since she consulted me, and she has remained well in the interval. She said that "ever since having the tube passed" she had felt much better, and the orzone at night had been very helpful at night in inducing sleep.

This case is more typical of nervous indigestion than Case 1. The patient was of an impressionable type, and I am of opinion that the mere reassuring her there was no evidence of organic disease present did her more good than the gastric analysis or the malted milk and olive oil at night, though her own explanation of her cure was different.

CASE 3. E.M., female aged 50; housewife; married; three children. Family history of no significance. Past history: apart from occasional attacks of "bilious vomiting" the patient had enjoyed good health. These attacks had never been so severe as to necessitate her seeking medical advice. On the evening of 27.4.32 I was called in to see her, and found her in bed in an emotional state, and

complaining of epigastric pain and vomiting. She appeared to be excited, and was weeping, but whether this was due to the pain or to some external cause, I could not at the time ascertain. Her pulse was 76 per minute and of good quality and she did not "look" ill in spite of the lamentations. As there was no indication of an acute surgical condition of the abdomen, I gave her morphia gr. $\frac{1}{4}$ by mouth as the sudden onset of the symptoms suggested the possibility of biliary colic, and in any case it would serve to allay her apprehensive state. I saw her the following morning and she appeared easier though she still had some indefinite epigastric discomfort. The respiratory and circulatory systems showed no abnormality and careful abdominal palpation failed to elicit hepatic or gall-bladder tenderness. There was no previous history of jaundice. Urine and faeces normal. Two days later a test breakfast was given with this result: Free HCl 6; total acidity 30; no lactic acid; no occult blood. The meal was difficult to recover owing to a slight excess of mucus. Gastric lavage was carried out on two successive mornings, the stomach being washed out with hydrogen peroxide (one drachm to the pint of warm water), as there was evidence of a catarrhal condition of the organ. The next week another test meal was given. Result: Free HCl 16; total acidity 40. There was less mucus, the meal being evacuated with ease. Her general condition had improved.

I later elicited the information from her husband that

some days before the acute attack she had been worried through her daughter aged 21 suddenly leaving home for London, without notifying her people as to her exact whereabouts. This had upset a nervous system which was always somewhat unstable, and I regarded the gastric catarrh and hypoacidity as indirectly due to a functional derangement of the stomach brought about by an upset of the nervous mechanism.

The patient was kept in bed for a week on light diet and a sedative mixture and she has done well since. She was not anxious to submit to an X-ray investigation, and in view of her continued improvement over a period of several months, I saw no reason to insist. Her weight has been maintained, and her appetite has returned. I may add that the domestic cause of her anxiety has since been satisfactorily removed, and I suggest that this fact has contributed in no small degree to her sense of well-being.

(d) DYSPEPSIA OF INFANCY AND CHILDHOOD DUE TO FAULTY DIET.

i. Infancy.

Although this is the last section concerned with the functional dyspepsias in some ways it is the most important, from the general practitioner's point of view, as the problems involved are of almost daily occurrence, and their satisfactory solution is of such moment. There is no need to labour the point that the training in the medical curriculum affords little real opportunity for the study of minor cases of dyspepsia in the infant and child, and yet it is just these cases which may cause a mother considerable anxiety, and, if the symptoms are allowed to progress in the case of a young infant, serious wasting may result.

Where mother and child are healthy, and where breast-feeding can be carried out, in my experience it is rare to find the infant suffering from any intractable form of indigestion, and, providing the feeds are given at regular intervals and sufficient time allowed at each feed - say twenty minutes - the infant should thrive without undue difficulty. It is when artificial feeding has to be adopted that trouble frequently begins. After several years in an industrial practice I find that it is only by giving simple instructions to the mother that there is any real hope of specific directions being carried out, and I have been struck with the remarkable frequency of rapid improvement in the infant's condition which results

when the correct quantity of milk has been ordered.

A very considerable literature has accumulated on the subject of infant feeding, and the more one reads about it, the more confusing it all seems. I propose, therefore, to describe the method I have found most suitable, though I recognise that many other systems may serve equally well, but most of them have the practical disadvantage that, from the mother's point of view, they are not so simple to carry out.

Gastro-intestinal disturbances are the most common cause of malnutrition in infancy, and it is in these cases that modification of a faulty diet is especially necessary.

In the infant, subjective symptoms of course can only be surmised, and objective signs are chiefly confined to vomiting and consequent loss of weight. With regard to vomiting: it is paradoxical but true that this sometimes occurs when the only error in the diet is that the infant is not getting enough food. Probably the child in sucking vigorously at the empty bottle swallows too much air, and this causes eructations which tend to bring up some of the milk recently swallowed. I propose to give details of one of my cases showing this feature later (page 136). The question arises then, what is the correct quantity for a child of a given age? I have found the method for calculating this recommended by Fleming (59) to be of great service. His method presupposes that infants should be given quantities of food suitable for their age rather than their weight: i.e., their expected weight rather than their

actual weight should be used in calculating their food requirements. Experience has shown that on an average the total daily caloric requirements per kilo. (2.2 lbs.) of body weight of normal infants is made up as follows:

The basal metabolism requirement	55	calories per kilo.
Growth.....	5	" " "
Loss in excreta.....	5	" " "
Muscular exertion.....	35	" " "
Total.....	100	" " "

The aim should be, therefore, to provide a normal infant with a daily ration of a caloric value of 100 for every kilo. of its body weight. It is, perhaps, advisable to enlarge briefly on the factors which combine to give the total of 100 calories:

Basal requirement of under-nourished infants: It has been found that until one-third of the expected or normal weight for the age has been lost, the basal metabolism remains constant. When the infant becomes more than 35 per cent. below its normal weight, the basal metabolism gradually diminishes as emaciation advances. The rate of diminution is about 1 calorie for every 1 per cent. the infant is below 65 per cent. of its expected weight. (Thus an infant only 35 per cent. of its expected weight will have a basal metabolism of 25 calories per kilo. of its expected weight (55 - 30 = 25)).

Amount required for growth: A slightly larger amount should be made for this in severely under-nourished children.

Loss in excreta: Same allowance always made here.

Muscular exertion: Under-nourished infants are less active than the well-nourished. When one-third of the weight has been lost 25 calories per kilo. of expected weight should be sufficient.

The total requirements per day per kilo. of expected weight are 90 calories when one-third of the expected weight has been lost. As emaciation proceeds beyond this, one calorie per kilo. of expected weight should be deducted for every one per cent. the infant is below 65 per cent. of its expected weight. Thus, when only 45 per cent. of its expected weight it requires 90 - 20 = 70 calories per kilo. of expected weight.

To estimate the expected weight of an infant at any age it is assumed that at birth it should weigh 3.2 kilo. or 7 lbs. It should add 600 gm. or 1¼ lbs. every month till it is 6 months old, and thereafter 500 gm. or 1 lb. till it is a year old.

It is now simple to calculate the caloric requirements of infants in all stages of nutrition. First ascertain the actual weight, then its expected weight, then calculate the percentage it is of its expected weight. If it was 65 per cent. of its expected weight it would require 90 calories per kilo. It is, of course, necessary to convert calories into terms of food and for this purpose it is assumed that an ounce of milk has a caloric value of 17, and a drachm of sugar has a similar caloric value of 17. By dividing the caloric requirement by

17 the figure obtained gives the number of ounces of milk necessary per day, and this is further divided by the number of feeds per 24 hours to obtain the dose of milk per feed. It is usually advisable to allow a teaspoonful (ʒ i) per feed, and for this purpose one ounce less of milk is given each time according to the calculated amount.

It is, I think, a matter for regret that so many mathematical figures have of necessity crept into this section. But it will be noticed that the actual calculation is simple, and in any case it is the physician who does it, the mother being instructed in the quantity and time of each feed.

I propose to give details now of a case I had which I think serves to demonstrate how the method outlined above works out in actual practice.

Baby H. was born in the Maternity Hospital as the mother had a contracted pelvis. For two weeks the mother breast-fed it, but her milk was insufficient and finally it was put on the bottle only. I was called in when the infant was three months old, as it was not thriving and was vomiting frequently. I formed the opinion that it was actually being under-fed, and I decided to instruct the mother to give it whole milk and sugar, the quantity being based on the method outlined above. At 3 months it should have weighed 7 lbs. + $1\frac{1}{4}$ x 3 lbs. = $10\frac{3}{4}$ lbs. 1 lb. = 450 gm. (approx.); therefore $10\frac{3}{4}$ lbs. = 4,800 gm. (approx.). Now the actual weight

of this infant was $8\frac{1}{2}$ lbs. or 3,800 gm. Thus it was only 78 per cent. of the weight it should be. ($\frac{3,800}{4,800} \times \frac{100}{1} = 78$). As this was within 35 per cent. of its expected weight it should receive about 90 calories per kilo. of its expected weight per day. Its expected weight was $10\frac{3}{4}$ lbs. or 4.9 kilo.; therefore its total caloric requirement per day should be 4.9 x 90 = 441 calories. $\frac{441}{17}$ = 26 ounces of milk per day. Five feeds a day were suggested and a teaspoonful of sugar to be added to each feed. The directions given to the mother, then, were: (i) to feed the infant at 6 a.m., 10 a.m., 2 p.m., 6 p.m. and 10 p.m.; and (ii) to give at each feed $4\frac{1}{4}$ ounces of whole milk and one teaspoonful of ordinary cane sugar. These quantities were later revised as the child grew older. The vomiting ceased and regular gain in weight was the result.

No doubt in the homes of the well-to-do elaborate procedures may be welcomed, but at the same time essentials may be overlooked. In a working-class practice such as this is, I have found simple directions based on a rational method of calculation to be of very great value in correcting dyspepsia and loss of weight due to erroneous quantities in the milk-dieting of infants.

Vomiting may also occur where there is no gross dietetic error, and where there is no apparent sign of organic disease. Such cases sometimes respond to a <u>diminution in the fat-content</u> of the milk. In this type of dyspepsia I have found the Pirquet diet to be of benefit. This method also has

the advantage of simplicity as far as giving the parent directions is concerned. Briefly it consists in diluting the milk with equal quantities of water and adding a teaspoonful of sugar for every ounce of water. It will be noted that this brings the mixture to the same caloric value as whole milk.

Casein indigestion is sometimes the factor producing gastro-intestinal symptoms in the infant, and in such cases I have found it often helpful to use whey as a diluent of the cow's milk, instead of water. To prepare whey a pint of milk is taken, warmed to 100°F. and then a teaspoonful of rennet is added. The mixture is allowed to stand in a warm place till a firm curd has formed. The curd is then broken up with a fork, and strained through a sieve lined with muslin. (If whey is used as a diluent for fresh milk it should be boiled to destroy any ferments present). The particular advantage of this preparation is, that it increases the quantity of lact-albumin (digestible protein) while diminishing by dilution the percentage of casein in the milk to which it is added.

I readily admit that all these methods of modifying cow's milk may fail to correct the dyspeptic symptoms of the infant: in such a case recourse may be had to condensed or dry milk. While realising, too, that in many clinics this latter method of artificial feeding is the one of choice, I hold that where breast feeding is impracticable the use of fresh cow's

milk obtained from a reliable source is the next best food for the infant. In any event, this is not the place to discuss the relative merits of the many proprietary foods on the market, and it is only by the empirical use of a particular brand that its claims can be established or otherwise in a particular case.

There is another point which I have found of practical value where there has been considerable gastro-intestinal upset due to faulty milk-feeding. By stopping the administration in any form temporarily and substituting for it raw-meat juice and cod-liver oil, there has resulted a rapid improvement in the gastric symptoms and in the appearance of the stools. In such cases the return to milk must necessarily be gradual.

The possibility of constitutional disease - especially congenital syphilis - must always be considered where the infant continues to fail, or where there is a history of abortions or still-births. Again, it is important to realise that the symptoms presented by the little patient may be so atypical as to obscure the underlying cause. I recently had a case which presented some difficulty:

Baby A., a female infant, was born after a normal confinement, in December, 1932. A week after birth it developed icterus neonatorum in a very persistent form. The mother was unable to feed it owing to early loss of milk, so it was put on cow's milk in what was considered a proper quantity for its

age. In spite of the jaundice it appeared to thrive, but after the fourth week it started to vomit, and, in spite of trying many modifications of diet, including the use of dried milks, it failed to respond. The abdomen was frequently inspected, but on no occasion was there any suggestion of visible peristalsis. (The parents were healthy, and there had been a previous pregnancy which had resulted in a healthy child). Obviously, however, the infant was rapidly failing, and it was sent to hospital, Though even in hospital the symptoms were not regarded as definite, it was decided to operate in case of pyloric stenosis. This was actually found to be the case, and the infant made a good recovery. The absence of definite projectile vomiting and visible peristalsis would have led to a diagnosis of marasmus, had not an actual lesion been found at operation, and one is inclined to wonder whether some fatal cases of "wasting" may not in fact have been due to congenital pyloric stenosis with atypical symptoms!

In ordinary cases of dyspepsia in infants I feel that frequent changes of diet are much to be deprecated. If a mixture has been prepared which we know to be correct for the weight and caloric requirements of the child, it should be given a reasonable trial to permit the previous dyspepsia to subside, and to give the infant's stomach time to accommodate itself to a rationally balanced feed. It is only in cases such as the one cited above where the infant is getting rapidly and progressively worse, that one is justified in modifying this rule.

ii. Childhood.

"Indigestion" is one of the commonest causes of indisposition in childhood, but it may be extremely difficult to recognise in many instances, as the symptoms may appear in so many guises. I propose to deal with the subject from the clinical rather than the scientific aspect as in general practice it is, I submit, the former aspect which is the chief concern of the physician.

It is often the case that a mother brings her child to the consulting room with the ready-made diagnosis of "bilious attacks": this may be simply a cloak for a serious disease such as appendicitis, or it may be a gastro-intestinal upset due to faulty diet. In any case, I suggest it is of great importance to get a careful history of the onset of the symptoms and then to make full inquiry as to the child's diet during the preceding few days. The old axiom that one man's meat is another man's poison is particularly applicable when applied to the diet of children. To take but one instance: eggs are regarded as a useful food for most growing children, yet it is common knowledge that with certain children they act on the gastro-intestinal system in a manner which must be described as toxic. Hence the difficulty in being dogmatic in giving generalised dietetic advice where children are concerned. I always attach much importance to a mother's statement when she says a particular food "never agrees" with her child.

Broadly speaking, the main type of dyspepsia in later childhood appears to be of carbohydrate origin, and even in cases where there is definite evidence of mal-assimilation of certain other classes of food - such as fat - there is frequently an associated defective carbohydrate metabolism. Certainly in the district in which I practise the average child appears to receive too much food of the carbohydrate category. This seems to have an economic cause to some extent, in that bread and potatoes are cheap, and the child is allowed to over-indulge himself in these articles of diet. At the same time it is no doubt true that children of the more well-to-do classes at times suffer from the same form of indigestion, but due here to an excess of cakes, sweets, biscuits, etc.

Apart from faulty diet there are certain accessory factors which may contribute to the dyspepsia, viz., defective mastication, faulty habits and methods of feeding, and constipation: these will be briefly dealt with in considering treatment.

The Symptoms of carbohydrate dyspepsia are not usually sufficiently definite to be pathognomonic of the condition. Very often I find the mother comes with her child stating it is "getting so thin", and that it has a "dry irritating cough", and nothing may be mentioned pointing to a gastric derangement except that, when questioned, there is a history that the child has lost its appetite. It is just this type of case

where a full dietetic history must be obtained, and often information is elicited that the child is having "pieces" (of bread) in between meals. No doubt the clinical picture of the child has made the mother anxious in case it is developing into a case of pulmonary tuberculosis, and certainly the cough and loss of weight are so suggestive that one may be excused for considering the possibility of a pulmonary lesion. In such a case as this I inspect the stools, and if an excess of mucus is present, the chances are that the origin of the trouble is dietetic and not organic.

Pain in the abdomen is variable, and, if present, is usually indefinite in character. The abdomen may be somewhat distended - here again superficially simulating a tuberculous tendency - but no glands are palpable. The mother may have noticed that the child "went pale" at times. This may be due to spasms of colic, and may precede the passage of mucus-coated stools. It is admitted that these symptoms may occur where more serious disturbance is present than mere indigestion, but the point I would emphasise is, that quite frequently the whole cause is one of functional dyspepsia. Six months ago, however, I had a case which appeared to point to carbohydrate indigestion, but which was found to have an organic basis:

Edith M., aged 7, was brought to me owing to loss of weight and appetite, and with indefinite colicky abdominal pains. The mother, who was an observant woman, noticed that the symp-

toms were more common during the school days than at the week-end. The child was nervous, and did not "get on" with her teacher. Mrs. M. not unnaturally thought that the child was worrying about school and that this was causing the loss of appetite and "indigestion", but she was anxious to obtain medical advice. The child's daily diet appeared to be satisfactory, except for the fact that she was inclined to have too much carbohydrate in the form of bread. Abdominal palpation did not yield any definite result beyond a slightly spongy sensation: no pain was produced on pressure. Specific directions were given as to diet, similar to that which will be mentioned later under treatment, and the mother was told to return with the child if continuous improvement did not result. The girl was brought back during the Easter vacation (a month after the first examination). Apparently some improvement appeared to take place for a time but the colicky pains had returned, in spite of the fact that the girl was now on holiday. This time on abdominal examination there appeared to be relative tenderness in the right iliac region, and I suggested there may be a subacute appendicitis, which was causing a recurrence of the symptoms. Edith was sent to the Children's Hospital where she was kept under observation for a week. Laparotomy was then performed and an inflamed appendix was removed. She made a good recovery and is now putting on weight steadily. At no time had she vomited, and, as far as I am aware, her temperature had been normal.

The occurrence of night-terrors is another feature of dyspepsia in children, but it is also not uncommon where threadworms are present: hence the importance of inspecting the stools.

Nausea and vomiting may be present and it is not infrequent for acute symptoms to usher in an attack of indigestion. Thus Ian M., aged $2\frac{1}{2}$, suddenly took ill one evening with a temperature of 100^{o} and severe vomiting. Nothing was retained in the stomach, the breath was heavy and the tongue coated. Abdominal examination did not suggest any surgical condition, and the child was starved for 24 hours, only sips of water being allowed. Calomel in fractional doses was prescribed and on the second day improvement set in which was maintained. Prior to this acute attack the child had been having too great a carbohydrate intake: biscuits and sweets between meals, with rather too liberal a supply of potatoes at the mid-day meal. This case is typical of many.

Diagnosis: The one condition which prolonged dyspepsia due to faulty diet is most apt to simulate is tuberculosis in some form or another. In this matter I agree with Hutcheson (60) who states that phthisis is a rare disease in children. A useful way to differentiate the two conditions, where the clinical signs are in doubt, is to have the temperature taken night and morning for a week. If there is a regular nocturnal rise, this would favour a tuberculous basis for the symptoms. Abdominal tuberculosis, on the other hand, may provide more difficulty as it is commoner than the respiratory form in children. Radio-

logical examination by an expert for the presence or otherwise of enlarged mesenteric glands may yield useful results but even with care the diagnosis may be in doubt for some time. Where the disease is advanced, of course, the doughy feeling obtained on abdominal palpation in cases of tuberculous peritonitis is sufficiently characteristic to point to organic disease. Mere enlargement of the abdomen in an otherwise emaciated child is quite compatible with simple functional derangement, hence it is that careful abdominal palpation is of much greater clinical value than inspection in these cases.

Treatment: There is no doubt that defective mastication is frequently present in these cases, so that the gums and teeth should be examined with a view to the correction of any gross defect. Further, the common tendency amongst children to "bolt" their meals must be combated. This is not always an easy thing to ensure in a working man's household, where there may be a large family, but I always endeavour to impress on the mother the urgency of giving the child a reasonable time for the meal, and advising her to see that it rests ten minutes afterwards. Where the school child gets up late in the morning I feel it is better for it to have a slight breakfast rather than bolt down a good meal and then rush off to school: no amount of dietetic instructions will avail if the pernicious habit of food bolting is allowed to prevail.

Diet: The basis of this is to strictly control the carbohydrate intake. Sweets are forbidden, as also are excess of pastry, biscuits and jams. Bread is only allowed in small quantities, and similarly potatoes, porridge, and cereals are curtailed in amount. I give a diet sheet based on the following:

Breakfast: Small quantity of fine oatmeal well-cooked, with milk but no sugar. Soft boiled egg or small amount of bacon. Half a slice of thin toast. Cup of weak tea or milk.

At 11 a.m. if the child has an interval at school, one plain biscuit may be allowed.

Dinner: Where financial considerations do not permit of roast beef or mutton, a small quantity of mince, rabbit, tripe, or white fish may be allowed, with small portion of mashed potatoes or green vegetables in similar amount. For pudding, custard, jelly, or blancmange with stewed fruit juice, or baked apples. Milk may be given as a beverage, but if the child is having milk at breakfast and tea, it may be advisable to give water at the mid-day meal.

All seasoned foods, pickles, condiments, and pastry are forbidden. Soups, broths, etc., in small quantity are good for small children.

Tea: A cup of milk, bread and butter in moderate quantity; jam, plain madeira cake.

Supper (about 7 p.m.): a cup of milk or Ovaltine and a plain biscuit.

No food is allowed between the above meals, and the mother is instructed to see that the child gets no sweets.

Medicinal Treatment: Where the onset is acute I find the prescribing of calomel in fractional doses four hourly for twelve hours of benefit. In the morning a saline or hourly doses of Eno's fruit salts till purgation results. Sometimes the salts are better tolerated if the effervescence is allowed to subside before swallowing. Also, during the acute stage no food is allowed, but fluid in the form of sips of water is given as required. The return to a solid diet is gradual. Bismuth carbonate and sodium carbonate in a carminative mixture are helpful in settling an irritated stomach due to a faulty diet.

With regard to the associated constipation, rather than upset the digestion with excessive quantities of fruit juice, I have found an astringent tonic mixture, continued over a period of weeks if necessary, to be an aid in correcting the tendency to bowel sluggishness. Such a mixture contains tincture of nux vomica minims iii, compound rhubarb tincture, minims xv, sodium bicarbonate grains viii, and infusion of gentian to two drachms. This is given to a child of 6-8 years, thrice daily before meals.

Before concluding this section I feel it is advisable to mention briefly two conditions as they are associated more or less intimately with carbohydrate tolerance, viz., ketosis

and cyclical vomiting.

It is now generally held that the common factor underlying most ketotic states is a failure of carbohydrate metabolism, or carbohydrate starvation. Where this takes place there is an increased and incomplete combustion of fat, the products of which - acetone and diacetic acid - appear in the blood and urine.

As Bellingham Smith and Feiling[61] state, it is doubtful whether any diet at any period of life is entirely deficient in carbohydrate. Yet a child may suffer from carbohydrate starvation because it has an inherent difficulty in digesting, absorbing, and metabolising starchy food. It is starches which form the bulk of the carbohydrate diet of older children, and there is an increased tendency to ketotic conditions as the result of a deficiency of easily digestible carbohydrate.

The treatment consists in the correction of the faulty diet. In young infants on weaning, the addition of an adequate quantity of sugar to the milk may be all that is necessary. In older children the difficulty some of them have in digesting the more complicated carbohydrate foods may be overcome to some extent by giving a good malt extract after food.

Glucose is most useful where there is a tendency to ketosis, and if vomiting is not a prominent symptom it may be given by the mouth as lemonade.

Cyclical vomiting, where there are recurrent attacks of vomiting, is regarded as a form of ketosis, and I think the tendency nowadays is to look upon it as another example of carbohydrate starvation, but at the same time there appears to be a neurotic element present. Some years ago I had a case of this recurrent vomiting in a girl Matilda M., aged 6. During one bout of violent vomiting she brought up a considerable quantity of red blood. She was removed to hospital and was thoroughly examined both clinically and radiologically, but no organic disease of stomach or chest was found to be present. After discharge from hospital she was kept on glucose for some months - a dessert spoonful twice daily with meals. Her diet was regulated, and she has had no relapse since. Prior to the attack with the haemorrhage she had had a few milder bouts of vomiting, and her case was regarded as one of cyclical vomiting, though the bleeding was never satisfactorily explained. This child was of a neurotic type: very emotional and subject to headaches.

Many of these children present an idiosyncracy towards animal fat, so that care should be taken not to advise cod liver oil, cream, or other fatty foods, in cases which appear to suggest a tendency to cyclical vomiting or ketosis.

Prophylaxis, then, in these cases consists not only in adding glucose to the diet, but some restriction should be placed on the fatty constituents. Alkalies seem particularly beneficial in some of these cases, and I have found even ordinary bicarbonate of soda given alone, a useful adjuvant to the dietetic régime.

CONSTIPATION IN RELATION TO THE FUNCTIONAL DYSPEPSIAS.

It may be appropriate to discuss here that most common symptom associated with functional gastric disease, constipation, before dealing with the organic dyspepsias.

I have already deprecated the tendency amongst many patients of stressing the importance of sluggish action of the bowels (pp. 13-14), but where bowel movements occur less frequently than once in three days, it may be advisable to regard the condition as pathological. It follows that the significance of the word constipation is so variable with patients that to be of any value in a case-history specific enquiry should be made as to the average frequency of bowel movements. Indeed, I have ceased putting the question to a patient "Are you constipated?", and instead prefer to ask, "Do the bowels move regularly each day?", and if the answer is in the negative, further details are requested.

Etiology: Having established the fact that constipation is really present an attempt is made to find the cause. No doubt faulty habits are sometimes contributory factors, particularly the common tendency not to go to stool at a particular time each day. Certainly the average working-man has little time in the morning to devote to this purpose, but really this is no excuse, as a regular habit may be incul-

cated say after the evening meal.

Dietetic Errors: As a general rule, the more refined the food, the less residue occurs, and constipation is a probable sequel. Further, and no less important, is the fact that diminished intake of fluids is often a contributory factor which must be attended to in the treatment. A less obvious aggravating cause of constipation is an excessive consumption of sugar, through, as Morgan (62) suggests, its inhibiting effect upon the hepatic function. (It may be noted that in most of the diet sheets I have suggested for the various forms of functional dyspepsia, the amount of carbohydrate in the form of sugar is curtailed with this end in view).

A third group of cases owes its origin to defects in the neuro-muscular control of the colon, tending either to an atonic condition of this part of the large bowel, or, and probably more commonly, to a spastic state of the colon.

Certain cases of chronic constipation are undoubtedly due to a loss of power to expel the contents of the pelvic colon and rectum: this type has been given the name of dyschezia by Hurst. It frequently has its origin in the faulty habits already referred to. I have vivid recollections of a pronounced case of this type which I met with several years ago:

Mrs A., aged 60, had been complaining of abdominal discomfort, chiefly below the umbilicus, of some weeks' duration.

On inquiry she said she had been having small bowel evacuations fairly regularly about every two days. Five years previously she had had a cholecystectomy performed for gall-stones, and had been fairly well since. She had, however, an abdominal hernia since the operation. She was always of a sallow complexion, and was of opinion that she was losing weight, though in this matter I could obtain no definite figures. Abdominal palpation did not reveal any abnormality. I decided to make a digital rectal examination. She had had castor oil and an enema prior to this, and I anticipated finding the rectum clear of faecal material. On examining there was a hard mass to be felt about two inches past the anus. My first impression was that it was an inoperable neoplasm, but on applying firm pressure it appeared to yield somewhat, and on withdrawing the finger there was definite evidence of a loaded lower bowel. The result of the enema and castor oil had not been satisfactory, and I literally had to use a spoon to scoop out the impacted faeces. There had been a small passage through the mass, through which the bowels were at times evacuated. Her symptoms later on cleared up entirely, and it is now over five years since and she has been reasonably well during that period. Such a case suggests that the mere history of regular bowel action does not necessarily rule out the possibility of constipation.

The indiscriminate use of cathartics, if continued over a considerable period, may produce constipation by rendering

the bowel insensitive to the normal stimuli. The unjustifiable fear of auto-intoxication may thus be an indirect causative factor, as it all too often leads to the use of purgative drugs. This question of toxic absorption has already been discussed. (p. 13).

Symptoms: Certain of the symptoms met with in the various functional dyspepsias are no doubt due to the associated constipation. But in many cases there is constipation present which is, per se, devoid of symptoms. On the other hand, colicky pains may develop after some days of bowel inactivity and diarrhoea may occur to be followed by constipation again: this is more common with the spastic type than with the atonic. Where such indefinite symptoms as mild vertigo, headache, pyrosis, and slight nausea are present, these may be partly due to the associated gastric dysfunction, and no useful purpose, I feel, would be served by endeavouring to attribute such subjective disturbances to the stomach on the one hand, or to the bowel on the other. From the practical point of view it is enough where definite constipation is present to treat it along with the other symptoms of the particular type of functional dyspepsia.

TREATMENT: Each case must be considered individually, but there are certain basic principles which I find it useful to insist on in all of them:

An attempt must be made to secure a daily evacuation of

the bowels, preferably at a fixed time, but the patient is advised not to worry unduly if he fails to secure a passage, as no harm is done if a couple of days elapse without a bowel movement. If much difficulty is experienced, a glass of water taken before going to stool may help, and water drunk between meals is a further prophylactic aid.

Diet: This is important, but where there is a particular form of functional gastric derangement present, the meals must necessarily be influenced by the stomach condition. It would seem to be a mistake to be dogmatic in advising "bland" or "rough" diets in all cases of constipation, though as a general rule, where atonic constipation is presumed to be present, a coarse diet is permissible, and such a vitamin food as Bemax has in my experience proved a useful article of food in helping to correct a sluggish bowel. On the other hand, where in the course of an X-ray investigation, a spastic colon is found, a rough diet may aggravate the condition, and is therefore contra-indicated, so that bland, bulky meals should be advised - white bread, rice, fresh fish, stewed apples, pears, or rhubarb, etc. Butter, cream, olive oil, and cream cheese are all well tolerated by a spastic colon.

At the present time there is perhaps a tendency, amounting almost to a craze, not entirely confined to the laity, to increase the vitamins and roughage of the diet beyond all reasonable proportions, and I suggest that considerable abdominal discomfort is produced in many cases through a too concentrated

coarse diet. If constipation is not corrected by the moderate use of a diet rich in roughage, then other means should be adopted to secure more frequent evacuation of the bowels, and these will now be considered.

Abdominal gymnastics: It will perhaps have been noted that occasional references have already been made to abdominal gymnastics, and I have found them in many instances to be of distinct value, though to get satisfactory results the intelligent co-operation of the patient must be obtained. I realised, of course, that it would be futile simply to tell the patient what to do, so each receives a typed sheet with the various exercises detailed concisely. These groups of exercises, eight in number, are based on the series recommended by W. G. Morgan,(63) and in my view, they are a very considerable help in overcoming functional constipation in a patient who is otherwise in sound health. If, however, there is associated organic disease of the viscera or other organs, it may be inadvisable to permit these domestic calisthenics. I append a sheet of the exercises as given to suitable patients. They are advised to carry them out cheerfully and with enthusiasm, and to continue them daily, if necessary, for many months. The groups are not carried out in toto to begin with, but, commencing with the first one, an extra group is added every two weeks, though in some cases a more rapid increase may be considered advisable. Where the bed mattress is inclined to sag, and this is usually the case, it is advisable for the patient to carry

GROUP 1. Lying flat upon the back, without a pillow, with arms folded on chest:
Slowly raise right thigh to a right angle, with leg extended, while counting 10, and lower to floor while counting 10.
Repeat with left limb.
Repeat with both limbs at the same time.

GROUP 2. Position as in Group 1:
Slowly raise right thigh to right angle with the body, while counting 10, and then carry it slowly over to the left as far as possible, while counting 10, then slowly return it to the floor by the same route.
Repeat with left limb.

GROUP 3. Position as in Group 1.
Flex right leg upon right thigh.
Flex thigh upon abdomen 5 times rapidly.
Repeat with left limb.
Repeat with both limbs.

GROUP 4. Assume standing posture, with hands on hips:
Bend trunk forward as far as possible.
Resume erect position and bend as far backward as possible, always keeping the legs rigidly extended; perform these movements from 5 to 10 times.
Position same, carry trunk over to right side from 8 to 10
Repeat, carrying trunk over to left side. times.
Position same, rotate the trunk to right side upon the spine 5 to 10 times.
Repeat rotating trunk to left side.

GROUP 5. Position as in Group 4:
Carry trunk forward.
Without assuming upright position, carry trunk to left and backward to upright position.
Repeat in opposite direction.

GROUP 6. Position as in Groups 1 and 2:
Slowly raise the head from the floor, while counting 5, and lower it in the same manner.
With arms folded over chest, raise the trunk from the hips slowly to a sitting posture, then return to recumbent position.
With arms folded over chest, shoulders kept squarely upon the floor, roll the legs and trunk over to the left so that their weight is on the left side, the shoulders still being flat on the floor. Repeat with the opposite side.

GROUP 7. Position as in Groups 4 and 5:
Bring thigh to right angle with trunk, with the leg flexed.
Extend leg upon the thigh slowly. Slowly return to original position.
Repeat with left limb.
Carry right limb as far backward as possible. Then carry leg backward until the heel touches the buttocks.
Slowly return to original position.
Repeat with left limb.
Carry right thigh up until it is strongly flexed upon the abdomen.
Repeat with left limb.

GROUP 8. Position as in Groups 5, 6, and 7:
Rise on toes from 10 to 20 times.

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out the exercises on a couple of blankets folded over, on the floor. The exercises should, of course, be performed in the morning before breakfast.

I have had no practical experience in the administration of electricity in the treatment of constipation, and in any case such a method should only be carried out by an expert. This is rarely practicable where a man is working, and certainly it should be unnecessary for patients to sacrifice work for habitual constipation.

In atonic constipation expert abdominal massage may be of benefit, but auto-massage is not without risk, though recommended by certain writers. I prefer to rely on exercises as they are much safer where no expert assistance is available.

I place the administration of drugs last in the aids to treatment as I am reluctant to prescribe them in chronic constipation. At the same time one is not so obsessed with the other methods at hand not to realize that in certain cases it is really necessary to prescribe cathartics. I feel, however, that drastic purgatives have no place in the treatment of habitual constipation, and I impress on the patient that chronic complaints call for perseverance in treatment, and that a cure should not be expected in a short time.

Those preparations which (a) increase the bulk of the faeces without causing bowel irritation, or (b) lubricate the faeces through the mineral oil they contain should first be

tried as they are often sufficient to relieve the constipation. In the former category some form of agar-agar is helpful, and in my private practice I have found the proprietary preparation Normacol satisfactory. In the lubricant category Agarol Brand Compound with or without phenolphthalein serves a useful purpose. In panel practice liquid paraffin in suitable regulated doses is helpful where dyschezia is present.

Belladonna is most useful where the constipation is associated with a spastic colon, and I have used it empirically with success, where no X-ray examination was carried out, but where the colicky pains of intestinal spasm suggested the colon to be the seat of nervous contractions. For this purpose 10 minims of the tincture given before meals rapidly relieves the discomfort.

Where there is an associated congestion of the haemorrhoidal veins with some proctitis, a proprietary suppository Anusol (iodoresorcinsulphonate of bismuth) inserted at bedtime and one before breakfast seems to both relieve the discomfort of the piles, and also to contribute to a soft evacuation of the bowels. Presumably it has an astringent action on the inflamed parts.

It is usual to find that cascara has often been taken by the patient before coming for advice or treatment, but when it is taken as a single dose at bedtime it rarely is of use in curing the constipation. On the whole, I have found that the best result is obtained when the liquid extract is prescribed

thrice daily before meals, the dose being 5-15 drops, and it should be taken in about half a wineglass of water. The dose is gradually reduced as the treatment progresses along the lines suggested above.

Regarding enemas, apart from their use in an emergency, I do not advocate their regular administration in chronic constipation, but the most satisfactory vehicle to use in this connection in my experience is olive oil. The oil should be well warmed before using and it should be allowed to run in slowly.

I do not propose to give case histories of constipation per se as it is really outside the scope of this paper, and the interpolation of this section is only justified when it is regarded as a fuller description of one of the commonest symptoms of the functional dyspepsias than was found possible when dealing with the individual types of gastric dysfunction.

II. THE ORGANIC DYSPEPSIAS.

(a) GASTRIC IN ORIGIN.

General Considerations.

It is, no doubt, a controversial matter as to whether organic disease of the stomach or functional disorder is commoner as a cause of dyspepsia, but in my own experience, by far the greater number of patients who seek medical advice about their "stomachs" suffer from a derangement of function. I am aware that this is not the view of many writers, and I can only infer that their statistics are (i) either secured from say hospital sources, where the more intractable cases are dealt with, or (ii) cases with organic disease elsewhere but which possess gastric symptoms are included in the group of organic gastric disease, or, (iii) their experience is simply at variance with my own. The point I would make, however, is, that ulcer, cancer, and gastritis are relatively uncommon amongst the cases of so-called indigestion which the general practitioner is called upon to treat. This is not to assume, however, that because the probability is that a particular case is one of functional disease, therefore a full examination is unnecessary. It is, I submit, a safe plan to regard each gastric case as one of potential organic disease, and if this is kept in mind, one is less likely to "miss" early

cases of structural disease. It cannot be over-emphasised that the onset of early malignant disease of the stomach may be so insidious as to develop to an inoperable extent without producing any marked degree of gastric discomfort, so that the family physician must be ever on the alert for these apparently latent cases, for it is only by their early recognition that there is any hope of arresting the disease.

In this section I propose to deal more particularly with the diagnosis of cases of organic disease producing dyspepsia. Treatment will be considered in so far as it can be carried out in the patient's home, though it must be admitted that in an industrial practice such as the one I am engaged in, the question of surgical treatment comes up for consideration more frequently in connection with peptic ulcer than it would do in a middle-class district, and for this reason more attention is paid to medical post-operative treatment than to the purely medical treatment of gastro-duodenal ulceration. Such a state of affairs cannot be regarded as satisfactory, as it seems to me basically wrong to regard peptic ulceration as necessarily and inevitably a surgical condition. But the alternative is no better, and this for economic reasons, When an ulcer has been definitely located, the slow and painstaking treatment required if medically dealt with presupposes domestic conditions which rarely exist in a working-man's house, particul-

arly in these days of unemployment. The result is the patient is admitted to hospital, where the tendency appears to be towards surgical treatment unless there are definite contra-indications. The constant demand for beds in the voluntary hospitals under existing conditions appears to be unavoidable, and a gastroenterostomy operation enables the patient to get home probably in a considerably shorter time than could be safely effected simply by treating the ulcer case by medical means. In any event, I am of opinion that institutional accommodation for the medical treatment of peptic ulcer is advisable, as without expert radiological examination from time to time it is scarcely possible to say whether the ulcer is healing satisfactorily: the mere cessation of subjective symptoms when on a "diet" is really no criterion.

Regarding the importance of test-meal findings in organic dyspepsias of gastric origin, the results may be of considerable utility as an aid to diagnosis. Their significance must, of course, be interpreted in conjunction with the clinical and radiological data obtained in the particular case, and when thus considered the result of chemical analysis may be sufficient to establish the diagnosis in an otherwise doubtful case.

It would be difficult to over estimate the value of X-ray examination in the organic dyspepsias - particularly in ulcer and cancer cases - but such an examination, when it

yields a negative result must not be interpreted as necessarily ruling out organic disease, especially when the clinical evidence is strongly in favour of such being present. I had practical experience of the limitations of radiography some little time ago, and although it was not a gastric case I feel the details are pertinent, as it concerns the use of radiography for diagnostic purposes in pathological conditions of the gastro-intestinal tract.

In September 1932 W.A., a male aged 26 consulted me. His sole complaint was persistent diarrhoea. He had no pain, his temperature was normal and his appetite was good. Duration of symptoms about four weeks. He thought he was losing weight but was not sure. No history of passing blood or mucus per rectum. Physical examination of the chest and abdomen was entirely negative. No history or signs of tuberculosis. He was put on an astringent mixture and was told to return within a week if not better, and he was advised to take castor oil the night before reporting so that a rectal examination could be more satisfactorily carried out. His condition was unchanged at the end of a week. On digitally examining the rectum I was of opinion there was a hard mass to be felt with the tip of the index finger. He was sent to the infirmary where he had a sigmoidoscopic examination, a barium enema, and X-ray plates were taken. The whole of this examination was negative, and he was advised as to his diet, and was told that his symptoms

should clear up. He appeared to improve somewhat on a bland and restricted diet, but in March 1933 he was obviously worse, and was definitely losing weight. A further rectal examination produced the same sensation as before, and he was sent back to the infirmary. An inoperable rectal carcinoma was found, and deep radium therapy having failed to benefit him, he is to have a colostomy performed to afford him temporary relief.

This case is not mentioned with the specific object of pointing to the futility of radiography, but merely to show that too much weight should not be invariably given to the interpretation of plates or films when the clinical findings at least suggest an alternative diagnosis.

In conclusion, no amount of time spent on "special" examinations should be allowed to replace that devoted to securing a careful history followed by a thorough clinical investigation.

Gastritis.

Gastritis, in the strict sense, implies a condition in which there is a definite inflammatory lesion in the mucous membrane of the stomach which ultimately results in atrophic changes. The term, however, is used probably more loosely, and with less justification in many cases, than perhaps any other dealing with gastro-intestinal derangements. There is still a prevailing tendency for the

physician, confronted with a patient complaining of indefinite gastric symptoms to label it "gastritis" without carrying out a gastric analysis, which, I submit, is the one way of satisfactorily determining the presence or otherwise of chronic gastric catarrh. Assuming my contention regarding the laxity in diagnostic method is correct, the inference is that true gastritis is by no means a common disease. These remarks apply particularly to chronic gastritis: the acute form is less likely to cause diagnostic errors.

ACUTE GASTRITIS. Etiology: It is logical to expect that any irritant swallowed in food or as a poison is liable to set up an acute inflammatory condition of the gastric mucosa, and, depending on the nature and quantity of the irritant, rests the severity of the subjective symptoms. A less obvious cause is exposure to cold, and I have seen an acute attack more than once apparently brought about by a youth standing on a cold day watching a football match. In young children it is more commonly due to dietetic indiscretions, and the onset may be alarmingly sudden, though it may subside equally rapidly. Again, as a prodromal sign of one of the acute infective fevers, the vomiting associated with the accompanying gastric derangement is presumably due to the bacterial toxin affecting the gastric mucosa.

I propose to confine myself here to the consideration

of the acute simple or catarrhal gastritis, as other types such as phlegmonous and suppurative are so rare as to be more of academic than practical interest from the point of view of the general practitioner.

Symptoms: The onset is usually sudden: epigastric discomfort which may amount to actual pain, and perhaps generalised tenderness to palpation. Nausea and vomiting are frequently present. In severe cases the vomit may be streaked with blood, in addition to the presence of altered food and mucus. Constitutional symptoms are usual to begin with - febrile temperature, headache, great thirst, and foul breath. There may be actual rigidity of the recti muscles, and in such a case there may be serious diagnostic difficulty. Details will be given of a case which simulated an acute gastritis as it is just these borderline types which may cause a practitioner considerable worry and anxiety.

Diagnosis: The diagnosis of acute simple gastritis without fever, particularly in children, does not usually present much difficulty. Children are more liable to disorders of digestion than to any other afebrile complaint, and thus when indigestion develops suddenly with vomiting of bile-stained mucus, the probability is that there is an acute gastritis present. While it is true to say the exception proves the rule, it is advisable to keep possible

exceptions in mind: thus appendicitis in an infant is a practical possibility, and it may be confused with the more common gastric catarrh. The infant daughter of a medical colleague, Shiela G., aged 18 months took ill about two years ago with what appeared to be abdominal discomfort and vomiting. There was no muscular rigidity and the vomiting was slight. The temperature was 99°. Acute gastric catarrh was suspected. In two days from the onset of the illness there was a suggestion of localised tenderness in the right iliac region, but it was not definite. She was examined by a consulting surgeon who considered there was sufficient likelihood of appendicular disease to justify a laparotomy. An acutely inflamed and elongated appendix was removed and the child made a perfect recovery.

Febrile gastritis especially in children may give rise to difficulty owing to the possibility of it being secondary to an infectious disease. If, however, after 24 hours there are no signs of a rash or other infectious symptoms, the probability is it is a simple catarrhal gastritis.

Diagnosis: It is unlikely that a perforation of a gastric or duodenal ulcer may be mistaken for acute gastritis, especially where the existence of such an ulcer is known. It is different, however, where a slight perforation occurs in an apparently healthy subject. Here real diffi-

culty may be encountered. Three years ago I had such a case. I was called in about midnight to see a male patient, G.M., aged 20. He had been in good health until that evening, when he had a hot mutton pie for supper. About 10 p.m. he developed acute epigastric pain with vomiting. When seen by me there was definite generalised tenderness over the epigastrium and a slight suggestion of rigidity. The vomit appeared to contain altered food and a fair quantity of mucus. His pulse was 100 and temperature normal. My first impression was that he was suffering from acute gastric catarrh, but owing to the slight rigidity I did not feel satisfied in leaving him, and yet I did not wish to have him removed to hospital at such an hour unless the diagnosis pointed more definitely to a surgical condition. I decided to examine him again in half an hour. This time the objective symptoms were unaltered, but he stated the pain was worse, and he was inclined to writhe about the bed (a point against perforation). However more by intuition than logical reasoning I felt it was advisable to send him into hospital as soon as possible, and this was done. He was kept under observation till about 10 a.m. and then a laparotomy was performed. A pin-point perforation in the first part of the duodenum was discovered, and his convalescence was uneventful. After his dismissal from hospital he admitted to me that he had had attacks of what he took to be indigestion on previous occasions but never severe enough for him to seek medical advice. When first interrogated he was presumably too ill to give me this information.

Treatment: Rest in bed is essential in severe cases and advisable in milder ones. This may be all that is necessary, as the vomiting which ushers in an attack may be sufficient to dispel the irritant, and the return to normal health may occur without interference though I think it is advisable to "rest" the stomach for 12-24 hours. In children fractional doses of calomel followed next morning by an effervescing saline are very helpful where the vomiting is not too severe. While gastric lavage is advised by certain writers I am reluctant to adopt this procedure in acute catarrhal gastritis, especially in children, and the majority of cases respond to starvation, combined with a purgative or enema to ensure a satisfactory bowel evacuation. There is no doubt that the technique of lavage, simple though it is, is alarming to the child patient, and it therefore tends to aggravate the distress which is already present. If the stomach can tolerate fluids I find that glucose drinks are satisfying and beneficial, and latterly I have been using glucose-D (glucose to which an adequate quantity of vitamin D has been added) with quite good results.

After the stomach has been emptied of its irritating contents, a gastric sedative consisting of bismuth and opium may be given: this I have found particularly useful in adult cases.

The return to a normal diet must be gradual. All

strong alcoholic fluids, condiments and rich foods must be rigidly excluded.

I do not propose to give details of "straight" cases as I have considered it of more interest to give instances of cases which simulated acute gastric catarrh, as when the diagnosis is established the treatment is essentially palliative.

CHRONIC GASTRITIS. This condition implies a definite inflammatory lesion in the mucous membrane of the stomach which ultimately produces atrophic changes in the secretory mechanism of the organ. There is no doubt that as a clinical entity it is difficult to diagnose the disease with certainty, and even when the gastric analysis has revealed an excess of mucus, pointing to inflammation of the mucus coat, it is still necessary and important to ascertain whether the gastritis is secondary to a more serious lesion.

Etiology: The commonest toxic causes are probably over-indulgence in alcohol, and strong tea drinking. Excessive tobacco-smoking over a long period is no doubt a contributory factor in certain cases. A vicious circle may be established, particularly by the chronic alcoholic, who indulges in the frequent use of strong condiments to stimulate his flagging appetite, with the result that further gastric irritation is produced, and this in turn causes more damage to the inflamed mucous membrane.

Certain drugs seem to be particularly prone to upset digestion and cause a toxic gastritis in time: amongst these, the salicylates, arsenic, quinine and mercury are the more common, so that the physician should be on guard when treating a chronic complaint, and vary the therapeutic measures he may adopt when he observes symptoms of gastric intolerance supervening.

Finally, chronic gastric catarrh may be secondary to some other disease of the stomach: ulcer or carcinoma; or, it may be associated with organic disease elsewhere: phthisis, renal or cardiac disease etc.

It will thus be noted that even when the diagnosis is established it is still necessary to find out the etiological factor at work before attempting a prognosis and adopting a line of treatment.

Symptoms: An insidious onset is the rule, and even when the disease is advanced there is no pathognomonic symptom to point to the diagnosis. Pain is not a feature, but epigastric discomfort is usually present. As a general rule there is loss ofappetite, and even when there is a desire for food, a few mouthfuls are usually sufficient to produce a sensation of satiety. The commonest symptom I find is nausea or vomiting, particularly in the morning. When the latter is present it usually consists of an excessive quantity of thick tenacious mucus. Such a symptom when of frequent occurrence, and associated with an alcoholic

history (not always readily obtained) strongly points to a toxic gastritis.

Flatulence is common especially in the late stage when there is considerable diminution of free hydrochloric acid, as then there is decomposition of the food particles with the formation of carbon dioxide. Intestinal flatulence with the passage of foul-smelling flatus may also be present. Thirst may be a distressing symptom, particularly in alcoholic gastritis, and it may serve as an excuse for satisfying the craving for more of the intoxicant.

When the disease is allowed to progress, constitutional symptoms occur such as tachycardia, arrhythmia, emaciation and general debility. The patient wakens in the morning quite unrefreshed, lassitude is present, and he feels generally unfit to cope with his work.

Abdominal examination may reveal some epigastric fulness with more or less general tenderness, but the physical signs in an uncomplicated case are practically negative.

Gastric analysis: This is essential for a diagnosis to be made with any degree of certainty. Typically, free hydrochloric acid is markedly diminished, if present at all. Indeed, I find free hydrochloric acid is frequently absent, though the total acidity may be normal on account of excessive fermentation with the consequent presence of organic acids. Excess of mucus is almost invariably present, though in a very late stage even this may be nearly absent. Care should

be taken to distinguish mucus swallowed from the naso-pharyngeal passage, from gastric mucus. The former is recognised from true gastric by its clear lumpy appearance. Owing to the mucus the filtering of the specimen is characteristically slow, and the coarse particles of the toast are seen surrounded by mucus.

Differential diagnosis: It is necessary to exclude the following: (a) peptic ulcer and malignant disease; (b) chronic cholecystitis; (c) chronic appendicitis; and (d) nervous dyspepsia.

(a) Pain is usually more a feature of the symptoms, and, in the case of ulcer, the periodicity of the discomfort is an aid to differentiation. Radiographic examination should be carried out in suspicious cases. Loss of weight in malignant cases is usually much more rapid than in chronic gastric catarrh.

(b) The differentiation of the late stages of chronic gastritis and old-standing cholecystitis is often difficult, because in each the gastric juice shows a subacidity, and there may be no excess of mucus in either condition. A careful history supplemented by X-ray studies of the gall-bladder and stomach may be of considerable assistance.

(c) Chronic appendicitis may be more readily distinguished: a history of a previous acute attack and discomfort after defaecation, with the abdominal pain situated more in

the right iliac region: these point to an unhealthy appendix, irrespective of the findings on gastric analysis.

(d) In my view nervous dyspepsia usually constitutes the greatest difficulty. Indeed the two conditions may be co-existent; and the chronic gastric catarrh may cause secondary nervous manifestations. Where the initial symptoms are purely digestive, the case is probably a primary gastritis. However, an error in diagnosis here may not be of much significance, as in either case the dietetic and general treatment are sufficiently similar to prove or benefit.

Prognosis: This should be guarded, as even when an apparent cure has been effected, relapses may occur. But in all cases, except when associated with serious organic disease, some improvement can be looked for where a regimen such as I propose to give, is conscientiously carried out by the patient.

Treatment: In mild cases it is my practice to rely on dietetic measures combined with such alterations in the personal habits of the patient which may seem advisable.

As regards diet, the aim is to suggest one which is at once nutritious, bland, and non-irritating. Where possible, small meals are recommended at short intervals, rather than three larger ones per day. Alcohol and tobacco must be forbidden to begin with, and their permanent abandonment

is advisable in patients subject to chronic gastritis. The following is the type of diet sheet recommended to begin with, subject to individual modifications:

7.a.m. Glass of warm water with pinch of salt added. Rest lying down 15 minutes.

7.30 a.m. 6-8 ounces of strained oatmeal, well-cooked. Take with milk or cream. Soft boiled egg. Cold toast with butter. Cup of weak tea with milk. No sugar.

10.30 a.m. Glass of warm milk with plain biscuit.

12-1 p.m. 2 ounces of steak well-cooked; mashed potato. White bread (2 ounces). Baked apple. Glass of water.

4 p.m. As at 10.30 a.m.

6.30 p.m. Eggs (one or two) cooked any way except fried; bread (white) and butter. Cup of weak tea with milk. No sugar.

9.30 p.m. Cup of Ovaltine or cocoa and plain biscuit if desired.

Such a diet should be taken for about two weeks, and then it may be supplemented both in quantity and variety until the usual number of daily meals are being taken. Lamb, chicken, and fresh fish may be added to the menu, and in about two months, in a case which is progressing satisfact-

orily, return to a full diet may be allowed, though the following should be excluded for many months: fried foods, pickles, sausages, pork, veal, cucumber, cabbage, and coarse cereals.

Slow eating and thorough mastication of the food before swallowing should be enjoined, as many of these patients are inclined to bolt their meals more from habit than necessity. Rest in the recumbent position after food may be helpful, and, where so many of one's patients are unemployed, there is usually no difficulty in the individual finding time for this. Where constipation is a feature, and where the gastric symptoms are not severe, abdominal gymnastics in the morning are sometimes advised. Tepid baths and exercise falling short of actual fatigue are general measures which indirectly serve to benefit the gastric condition.

In the majority of cases of chronic gastritis, the regimen outlines is sufficient to effect a cure, but more intractable cases require further measures, including lavage and medication.

Lavage: Where, following gastric analysis, the stomach is found to contain a considerable quantity of mucus, washing out the organ in the morning before breakfast may be very beneficial. For this purpose I have used sodium bicarbonate (one drachm to the pint of warm water), but latterly I have substituted dilute hydrogen peroxide

(one drachm to the pint), as recommended by Hurst,[64] with satisfactory results. Indeed where an apparent achlorhydria is present, a normal secretion of gastric juice may sometimes be restored by this method of lavage. I employ Senoran's evacuator as a rule, but where the rather large stomach tube is resented by the patient, the wash-out may be effected by the smaller Ryle tube and syringe. Lavage is essentially a temporary form of treatment, and in no way supplants careful attention to diet and the general health.

Regarding <u>therapeutic</u> aids to treatment, I have found these to be somewhat disappointing. The indiscriminate prescription of dilute HCl in chronic gastritis is to be deprecated. I agree with Hurst who states that only in those cases in which there is no mucus from the beginning, or in which the acid secretion does not return after lavage, should large doses of dilute HCl - up to two drachms in orangeade - be given as a beverage with each meal. Einhorn,[65] who was in the habit of employing dilute HCl alone or with pepsin, has of late years entirely abandoned pepsin, and generally restricted the HCl, basing his decision on the belief that the means which serve for the digestion and utilisation of food by the organism are certainly not limited to the stomach, but that the principal part of the process takes place in the intestine. Predigested foods, or medicaments which contain the active principles of the gastric

juice and serve to replace the work done by the stomach will, in course of time, have a deteriorating effect upon the gastric functions. This may well be the case, and certainly in practice I have not found the administration of dilute HCl or pepsin to be of any real benefit in chronic gastritis. On the other hand, mixtures containing tonic bitters such as quassia, gentian, calumba, or nux vomica, may produce some improvement, presumably by acting as gastric secretory stimulants indirectly through their bitter taste.

Case History.

J.D., male aged 30, married; compositor. Of nervous temperament, but had had no illnesses of any consequence, though he was not of robust physique. Family history negative. His habits were temperate apart from rather excessive cigarette smoking: 20-30 per day. For some months had been having attacks of "indigestion," with feeling of nausea especially in the morning. No history of vomiting and no acute pain, though he had frequent discomfort over the epigastrium. Sometimes he thought the taking of food relieved the pain or discomfort, but this was not always the case. Appetite fair though breakfast was not usually enjoyed. Morning headache present. Bowels acted regularly without purgatives. Belching was annoying and there were frequent eructations of acid fluid. Flatulence was marked especially in the early morning.

Physical examination showed a man of normal height

and weight ratio. He was of a sallow complexion, but had always been somewhat pale. Examination of oral cavity was negative. Superficial reflexes were active. Lungs showed no abnormality. Heart sounds rather soft, and tachycardia was a feature: pulse rate 120 per minute. Liver and gall bladder appeared normal to percussion and palpation. Abdomen was negative to inspection, and the epigastric tenderness to palpation was slight and not localised to any one spot. Gastric analysis was carried out on 15.4.32 with the following result: free HCl 0.08 per cent; total acidity 0.13 per cent. Gunzberg test was positive for free HCl. The meal was difficult to extract owing to excess of mucus. No occult blood present. Urine and faeces showed no abnormality. X-ray examination of the stomach did not reveal any pathological condition, and a diagnosis of chronic gastritis was made.

Treatment: He was kept in bed two weeks, partly on account of the cardiac debility, and partly to rest the stomach. Frequent bland meals were advised, and smoking was stopped to begin with, and rigidly curtailed afterwards. Lavage was carried out on one occasion only, as patient wished to see the effects of palliative treatment, and as a matter of fact he readily responded to the dietetic regime. After a month's absence from work he was able to resume, feeling considerably better. I saw him six months later, and found the improvement had been maintained, but he was

still careful of his diet, and cigarette smoking had been reduced to about five per day.

In this case the only irritating factor to account for the gastritis appeared to be the tobacco. He was, however, inclined to bolt his food, and this probably contributed to the persistence of the symptoms.

Many similar cases of chronic gastritis could be cited, differing of course in details, but I feel no useful purpose would be served in describing further representative histories, as it would inevitably involve a certain amount of recapitulation.

Peptic Ulcer.

Although theoretically it should not be difficult to distinguish between gastric and duodenal ulceration I think it will be conceded that in many cases where an ulcer is definitely regarded as being present, it may be impossible for the practitioner to be sure of the locus of the ulcer, and in any case from the point of view of medical treatment the matter is not of considerable moment. Of course, where satisfactory radiographs are available it is of much help, having located the ulcer, to observe the results of treatment. But as far as the subject of gastro-duodenal ulceration concerns the general practitioner I propose to regard it as one disease, though it will be necessary occasionally to discriminate between stomach and duodenum where the localisation is of particular importance.

Etiology: A considerable amount of literature has accumulated regarding this, and even now authorities offer conflicting views as to the causation of peptic ulcer. The matter is of far more than academic interest, as naturally a satisfactory prophylaxis cannot be established where the etiological factors are in doubt. The theory that peptic ulcer is due to microbic emboli seems to be worthy of serious consideration, and in this connection the experimental investigations of Rosenow(66) must be briefly mentioned. This worker claims to have isolated streptococci which have tendencies to attack specific portions of the alimentary tract. Thus he states he has cultivated from root-abscesses of the teeth of patients with gastric ulcer and from those ulcers themselves, streptococci which, when injected into the circulatory system of animals, produced gastric ulcers. As far as I know this experimental evidence of bacteriological infection as a cause of gastro-intestinal ulceration has not been confirmed by other writers, but it seems a subject demanding substantiation of refutation by research workers in this country. It is, of course, a recognised fact that local sources of sepsis and peptic ulcer frequently co-exist, and the association of chronic appendicitis, cholecystitis, and gastro-duodenal ulceration has often been referred to in surgical literature of the digestive tract. So that superficially at any rate there is much to suggest that the microbic theory of ulcer causation is correct in at least a proportion of these cases.

One factor in the etiology of peptic ulcer about which there is general agreement is that it is usually associated with the presence of highly-acid gastric juice, though this does not necessarily mean that there is a pathological concentration of acid in the stomach. It may be that there is an absence of the normal alkaline duodenal regurgitation which causes the "climbing-curve" of acid in the fractional meal. The deleterious action of the hydrochloric acid must be preceded, however, by some local injury or diminished vitality in the area that is ultimately to form an ulcer, and it is the nature and cause of this injury which has created divergent views.

While it is fair to state, then, that ulcer is liable to occur in individuals who suffer from hyperacidity, it is equally true to state that many people have hyperchlorhydria who never develop ulcer. Probably there is some kind of diathesis which predisposes to ulcer in certain persons, and in this matter A. F. Hurst[67] has for a number of years emphasised this tendency in certain types of cases. It has been shown that a large proportion of healthy men with hyperchlorhydria have also hypertonic stomachs. These are the individuals who have what Hurst calls the <u>hypersthenic</u> gastric diathesis: an inborn variation from the average normal which manifests itself in hypertonus of the stomach, with rapid peristalsis and rapid evacuation, plus hyperchlorhydria with

digestive hypersecretion. This type is more common in the male than in the female. It is compatible with perfect health, but it renders the individual liable under certain conditions to develop duodenal ulcer. The other type of diathesis is the hyposthenic. Here the patient has a long, low, slowly emptying stomach, and if hyperchlorhydria exists symptoms may occur suggestive of gastric ulcer. (If, however hypochlorhydria is present the result is totally different: an achlorhydric gastritis may result, but this type never goes on to ulcer formation, as for this the presence of free hydrochloric acid is essential. It may cause vague epigastric discomfort and nausea, but usually it is symptomless. It is, however, an important cause of disease in other parts of the body, as the achlorhydria not only leads to absence of peptic digestion in the stomach, but it is the chief cause of intestinal infection owing to loss of the acid barrier.)

I have considered it advisable to enter this parenthesis to make it clear that the hyposthenic gastric diathesis has necessarily no connection with hypoacidity.

It would be incorrect to assume that Hurst's theory of diatheses has been accepted by all authorities - Bennett, from whose work I have frequently quoted, does not agree that the evidence advanced is conclusive proof - but I venture to suggest that it meets the facts in so

many cases that any exceptions only tend to substantiate the basic truth of the hypothesis.

Such obvious irritants as excessive tobacco-smoking and the abuse of alcohol, and the habitual consumption of highly spiced, hard, and irritating foods may all tend to contribute to the formation of a peptic ulcer where the predisposition to such exists.

It may be surmised from what has been said regarding the etiology of peptic ulcer that much can be done by way of prophylaxis, and it is the general practitioner who is in a privileged position to give timely warning. This important practical point will be elaborated when considering treatment.

Symptoms: Pain: This is perhaps the most important and constant symptom irrespective of the exact site of the ulcer; and the time of onset with regard to the intake of food may provide a very rough indication as to the position of the ulcer. The writer has had to refer on innumerable occasions, when discussing the functional dyspepsias, to "epigastric discomfort," which is at the best an unsatisfactory expression, and as an aid to a specific diagnosis it is of little use, and yet it had to be mentioned almost ad nauseam as it is one of the symptoms of gastric dysfunction. With ulcer, however, actual pain is usually experienced, and it is of a boring, aching type, sometimes radiating through to the back. In gastric

ulcer the pain usually comes on within half an hour of taking food and as a rule disappears before the next meal is due, whereas in duodenal ulcer it occurs three to four hours after a meal, and it may even waken the patient by recurring in the early hours of the morning.

As Moynihan(68) states, we have a quadruple rhythm in gastric ulcer: food, comfort, pain, comfort, and then again, food, comfort, pain, comfort; whereas in cases of duodenal ulcer it is typically a triple rhythm: food, comfort, pain, and then again, food, comfort, pain.

In actual practice, however, it is often by no means easy to get a typical history of pain in relation to the onset of meals, even when a gastric or duodenal ulcer is found to be present, and I find this opinion is upheld by Hunt(69) who quotes from a paper by Emery and Monroe in which these workers found no apparent difference, as regards food and pain, between gastric and duodenal ulcers in 556 cases. On the other hand <u>for the particular patient</u> the pain is usually constant: i.e., it occurs at the same interval after a meal, and in this respect it differs from the pain and discomfort which is complained of by patients with nervous dyspepsia.

Regarding the cause of the pain there has been much controversy. Hyperacidity <u>per se</u> cannot be regarded as the cause, as one frequently finds that acid concentration is no real criterion as regards severity of the pain, but

it may be that excess of hydrochloric acid causes a sluggish action of the pyloric sphincter, apart from spasm, and when active peristalsis occurs, instead of the gastric contents being shot into the duodenum there is delay thus causing a rise of tension in the vestibule - and it is this increased tension which causes the pain. This increased tension as a cause of pain has been experimentally produced by D. Macleod,(70) who produced typical attacks of pain in some 20 cases of duodenal ulcer by blowing air into the duodenum through an Einhorn tube.

Vomiting: Though commoner in gastric than in duodenal ulcer it may be absent in both conditions. It may occur when the pain is very severe, and if this is the case it rapidly relieves the discomfort. In duodenal ulcer the patient may voluntarily induce vomiting to obtain relief, as experience has taught him that voiding of the gastric contents is unlikely without artificial aid.

Haemorrhage: This again is not a constant feature: its frequency is variously assessed at from 15 to 25 per cent. of all ulcers. All degrees of haematemesis may occur, from profuse and alarming haemorrhage to slight oozing which can only be discovered by chemical analysis. Where the blood has been in contact with the acid juice for some time before vomiting occurs it presents a coffee-ground appearance, having been converted into brown acid haematin. Melaena

may be present in the faeces a few hours after, or occult blood may be found after the appropriate chemical test. A point of practical importance is that sudden haemorrhage may occur in a chronic ulcer when the patient has been free from symptoms for a long period. On two occasions I have seen this occur - both in the same patient and at an interval of over a year. This case was that of a man, W.R., aged 35, a ship-yard labourer. He did not give a typical ulcer history, but admitted to occasional attacks of "indigestion." One evening after returning from his work he had a severe haematemesis without any warning. He was given an injection of morphia and removed to the infirmary where he was kept for a month. Gastric ulcer was definitely diagnosed, and he appeared to make a satisfactory recovery under medical treatment. Within two years he had another haematemesis, though in the interval his health had been good. Again he was treated medically in hospital, and he has been well since, and that is fully five years ago. (The question of surgical procedure during a quiescent period was considered, but the patient preferred to risk a recurrence of the bleeding rather than undergo a laparotomy.)

Anaemia and malnutrition may occur as secondary symptoms in chronic gastric ulcer, as the patient may be afraid to eat owing to the pain. On the other hand, the duodenal ulcer patient may appear to be in good health as he usually takes his food well, because he finds his pain

is relieved by the intake of food.

Constipation is usually present in cases of peptic ulcer.

Nervous symptoms: I think there is no doubt that worry and anxiety aggravate the symptoms of ulcer, and it is important to keep this fact in mind in order to prevent one from regarding a nervous patient as necessarily only suffering from gastric neurasthenia when he complains of stomach symptoms. In other words, the predominance of apparently purely nervous symptoms should not be allowed to exclude the possibility of a peptic ulcer being present. I had an interesting case of an organic nervous disorder which to some extent obscured the presence of a duodenal ulcer, details of which I propose to give later (p.209).

Physical Signs: During an attack of pain due to ulcer it is usual to find a small circumscribed area where tenderness is most pronounced on applying digital pressure. Such a site should be marked with a skin pencil, with a view to testing this point at a subsequent examination. If the pain recurs at the same place it suggests at least the advisability of a radiological examination, as an organic lesion may be present. In the intervals between attacks of acute pain there may still be relative tenderness over the ulcer site. Where the area of pain on pal-

pation is in the mid-epigastric line, I submit its significance is not necessarily of such importance as if it was localised to either side of this line. I am aware that some authorities, such as A. J. Walton,[71] hold that pain on pressure at the mid-epigastric point is remarkably constant with ulcer, but I have many times elicited tenderness here in patients who, after exhaustive examination, have been found to be suffering from functional dyspepsia only. These individuals are usually somewhat thin and neurotic, and the pain appears to be due to pressure over the abdominal aorta.

Muscular tenderness and rigidity: In uncomplicated cases of peptic ulcer the most I have found as regards these signs is a slight resistance of the upper recti. It is reflex in origin, and unlike the ulcer tenderness it does not vary with change of position of the patient.

Cases are recorded of a palpable tumour being present due to a large chronic ulcer, but I have not had such a case. Where a mass can be felt the possibility of neoplasm must of course be suspected until its presence has been definitely disproved.

Where a chronic ulcer is juxta-pyloric in situation there is a tendency sooner or later for stenosis of the pylorus to occur, and then the physical signs and symptoms of dilatation of the stomach may develop and complicate the clinical picture.

Investigations. (a) X-rays: I suppose there are only two unequivocal methods of diagnosing gastric ulcer: that of the radiologist and that of the surgeon. We may suspect its presence from the history and clinical examination, but I venture to think that no surgeon would ordinarily operate nowadays without radiological confirmation of the diagnosis. In the case of duodenal ulcer the condition may be surmised with greater accuracy from the history and chemical analysis, but here too, the expert radiographer may be of great service.

I have in an earlier section (p. 29) emphasised the advisability of obtaining the assistance of an experienced radiologist both to take the film and supply an interpretation as to the findings. Deformity of the duodenal cap may be just as suggestive of an ulcer in this region, as the ulcer niche or crater is characteristic of an ulcer of the stomach itself.

(b) Gastric Analysis: It must be admitted that the one-hour meal is of less value than the fractional test, as regards the information to be derived therefrom. But in gastric ulcer even the results of the latter method are so variable that a diagnosis could hardly be based on the findings. In suspected duodenal ulcer, however, where a climbing curve is obtained, so that after perhaps two and a half to three hours the height of the curve is at its

maximum: such a result would strongly point to an ulcer, especially where the clinical symptoms supported such a diagnosis.

(c) Occult Blood: This should always be tested for in suspected cases of ulceration: a positive result is of material importance, though when negative it does not necessarily rule out the possibility of gastro-duodenal ulceration.

Differential Diagnosis:

I. Neurasthenic dyspepsia may simulate peptic ulcer - both gastric and duodenal - depending on the type of individual. Thus in the gastric ulcer type, a middle-aged woman of poor physique who has become constitutionally exhausted through worry or overwork may develop symptoms analagous to those of gastric ulcer. The stomach becomes deficient in tone and ptotic. But with rest in bed a few days the symptoms clear up without therapeutic measures, though a relapse may occur after another period of excessive fatigue. When, on the other hand, the duodenal ulcer type, or, as Hurst[72] calls it, the individual with the hypersthenic gastric diathesis, becomes neurasthenic, the hypersensitiveness of his nervous system makes him aware of digestive processes which would otherwise pass unperceived, and it may result in symptoms which are almost indistinguishable from those of a true duodenal ulcer. It

is, therefore, unjustifiable to operate unless the biochemical and radiological evidence confirm the diagnosis made from a consideration of the subjective symptoms. If septic foci are present an ulcer may develop sooner or later. It follows that in these cases the cause of the pain complained of is not due to an ulcer, but rather to the disturbed motor functions of the stomach.

The following are the salient features of a recent case of "pseudo" duodenal ulcer:

T.D., an unemployed labourer, aged 45, had had periodic attacks of pain in the epigastrium for some months. The pain occurred regularly about two and a half hours after meals. There was tenderness on palpating one inch to right of mid-epigastric line. No vomiting; alcoholic history; had had good health apart from being subject to "indigestion." He was well-nourished in appearance, and, although examined on several occasions he never seemed to be suffering from excessive pain. Dieting, rest, and antacid medication helped him for a time, but he returned later with the old complaint. A single-hour test-meal was given with the following result: free HCl 30; total acidity 67. No occult blood was found, and there was no evidence of gross food retention. No excess of mucus in spite of alcoholic history. He was sent to the dispensary and a radiological examination was carried out. The surgeon admitted that the history was suggestive of duodenal ulcer, but the "X-ray after the barium meal shows

no abnormality of any sort in stomach or duodenum." The man was of a somewhat nervous disposition, and he told me later that an intimate friend of his had been attending the infirmary with a duodenal ulcer: it is possible that this knowledge produced a psychological effect on the patient, causing him to subconsciously correlate his own gastric symptoms with those of his friend. At any rate, when he was definitely informed that there was no evidence of ulcer, his improvement was sufficiently remarkable to suggest the above hypothesis. The question of malingering does not arise in this case as the man was unemployed, and he did not stand to gain by incapacity for work through sickness.

2. Gastric Crises. As a general rule abdominal symptoms in a tabetic case should only be diagnosed as of tabetic origin if they have the characteristic features of crises with none of the symptoms of ulcer. But at the same time it must be realised that in rare cases haematemesis may occur in a true crisis, though no ulcer may be found after exhaustive examination. I have described a case of this nature (p. 59).

3. Reflex Dyspepsias. As the epigastric pain in reflex dyspepsias is produced in the stomach as a result of reflexes from the diseased organ, it may simulate gastro-duodenal ulceration. But in the majority of cases the time of onset, the exciting causes, and the means of obtaining

relief are sufficiently distinctive to prevent confusion. Further, the local signs and symptoms of the primary disease help to make the diagnosis clear.

The two commonest forms of reflex dyspepsia are chronic appendicitis and crhonic cholecystitis.

Chronic appendicitis: It is only when this condition produces pain in the epigastrium that the term appendix dyspepsia can be correctly applied. Ordinarily, the pain is more or less continuous, and is not affected by meals to the same extent as is peptic ulceration. The effects of an "ulcer diet" fail to produce amelioration of the symptoms.

Chronic cholecystitis: This condition may closely simulate duodenal ulcer, though again the symptoms are more irregular, and the sensation of a feeling of distension, which often leads to aerophagy is not common in duodenal ulcer. Gastric analysis may not help in these cases, as there may be hyperchlorhydria with a rising curve, though in the more usual type achlorhydria is common. Radiography is very advisable, so that examination in an institution should be recommended, where the Graham technique (using pills of tetra-iodo-phenolphthalein) can be carried out. A mere X-ray photograph may not be sufficient, as, providing there are no gall stones present, the gall-bladder may appear normal, filling and emptying at the usual rate. In

doubtful cases, therefore, it is advisable for the radiologist to give a small opaque meal after locating the gall bladder, and then, by observing the effect of palpating over the duodenal bulb, he can determine whether there are any adhesions between the gall bladder and the duodenum. The general practitioner should realise that cholecystography affords a very exact anatomical means of making a diagnosis in these admittedly difficult cases.

4. Carcinoma of the Stomach. If there is pain in this disease it is usually constant, and does not show the rhythmical tendency so usual in ulcer. I say "if" advisedly, as it is quite possible for pain to be practically absent throughout the greater course of the disease: reference will be made to this important fact when dealing particularly with gastric cancer. Gastric analysis may give useful results: where there is an absence of free hydrochloric acid, and where lactic acid is present in a patient over 40 with a history of indigestion which has been persistent for a few months only, it is advisable to suspect malignancy until the contrary has been proved by exhaustive examination. (The question of achlorhydria in cancer of the stomach is a most important one in connection with diagnosis, and is discussed later.)

No doubt it is possible for a gastric ulcer to take on malignant characteristics, but on the whole this is rare,

in spite of much loose writing on this subject. The statistics furnished by MacLean[73] should do much to show that it is the exception rather than the rule for this serious complication to arise. Too much reliance should not be placed on the presence or otherwise of a palpable tumour. An advanced growth may be present which cannot be palpated, and on the other hand, a thickened pylorus due to old ulceration in this locality may give the sensation of a tumour.

Haematemesis: Where a copious haemorrhage occurs, but when once stopped does not recur for a long time - this favours an ulcer diagnosis. On the other hand, a relatively small haemorrhage which recurs at short intervals is perhaps more suggestive of malignancy.

Radiography may be of inestimable assistance in differentiation.

5. Gastritis. The indefinite character of the pain, and the morning vomiting of excess of mucus when present should suggest a catarrhal rather than an ulcerative condition. But sometimes the symptoms of gastritis are sufficiently atypical to render a radiographic examination advisable. A history of chronic alcoholism would suggest a gastritis, but this may be accompanied by an ulcer, so that such a history should not necessarily rule out the possibility of a peptic ulcer. Indeed, the case last referred to (p.193)

gave a history of excessive alcoholic consumption over a period of years, and yet he had symptoms of duodenal ulcer, though this was not confirmed after thorough examination.

6. Hyperchlorhydria. Here again the discomfort in the epigastrium - if present at all - is general rather than local. Vomiting is not common, and if present it shows neither red nor altered blood. Though the discomfort may be relieved by taking more food - as in duodenal ulcer - the so-called "hunger-pain" is not so intense. These cases however, if their symptoms tend to persist, should be X-rayed, as, after all, they are potential cases of ulcer, especially of the duodenal type.

Complications: Briefly these may be: perforation, haemorrhage, deformities due to cicatrising of the ulcer, and malignant degeneration of the ulcer.

Perforation: This is the most serious of the acute complications, and the life of the patient may depend on its early diagnosis and treatment. The latter is essentially surgical and so does not concern us here, but it is usually the general practitioner who has to establish the diagnosis in the first instance. Typically there is a sharp violent abdominal pain, with which is associated the symptoms of shock: dilated pupils, a small thready pulse, anxious expression and subnormal temperature. On examina-

tion the abdomen has a board-like rigidity, Respirations are shallow, and the patient lies almost motionless. When he is seen for the first time an hour or two later the diagnosis may be more difficult, in that the shock symptoms may be less evident owing to a temporary reaction, but the important sign of abdominal rigidity remains. Later still peritonitis supervenes, and shock again becomes pronounced. A fatal termination is the result unless surgical intervention is resorted to, but in any case the prognosis is necessarily grave. No doubt where operative procedure is carried out just after the onset of the acute symptoms the chances of recovery are correspondingly greater. It may be impossible to locate the site of the perforation from abdominal palpation, but where a definite history of ulcer is obtainable it is natural to assume that this is the locus of the acute lesion.

Haemorrhage: This has been mentioned when discussing symptoms, but where the bleeding is very profuse, it is more in the nature of a complication. A point worthy of note, I think, is that even with a large haemorrhage it is remarkable how, in the great majority of cases, the condition will subside with rest and medical treatment. The fact that it may start quite insidiously - as mentioned on p.188 - must be borne in mind when giving a prognosis in a case of peptic ulcer.

Deformities due to cicatrising of ulcer: Varying degrees of deformity may result, depending on the extent and site of the healing ulcer, but only two call for more detailed description, viz., pyloric stenosis and the so-called "hour-glass" stomach.

Pyloric stenosis: Although I consider it is correct to state that malignant disease is the commonest cause of pyloric stenosis, the cicatrising of a simple peptic ulcer may produce the same result. The clinical picture of stenosis is characteristic: visible peristalsis associated with the vomiting of large quantities of liquid containing food. Several pints of fluid may be voided every few days. Radiographic examination reveals a stomach showing gross delay in the passage of the barium meal. The treatment is essentially surgical.

Hour-glass contraction of the stomach: There are no pathognomonic clinical signs of this condition, and the diagnosis rests on X-ray examination. It is due to cicatrisation of an ulcer on the lesser curvature, fibrous tissue forming, and pulling on the posterior wall of the stomach to a greater extent than on the anterior, which is naturally more fixed in position. (There is also a functional or pseudo-hour-glass stomach, but this may be differentiated by the change in its appearance when plates are taken with the patient in the erect and horizontal positions - when the

patient is in the horizontal position the hour-glass shape disappears.) Extraordinarily good results are sometimes obtained by the surgical treatment of organic hour-glass stomachs.

Malignant degeneration of peptic ulcer: This is the gravest complication, but I have already emphasised (p.196) that it is fortunately an uncommon one. I refer, of course to gastric ulcers in this connection: the question of the possibility of malignant degeneration of duodenal ulcers is so rare that it need not be considered here. The practical importance of malignant degeneration lies in its bearing on gastric surgery. If malignancy following ulceration were as common as some investigators make out the advisability of gastrectomy in cases of chronic ulcer would have to be considered much more frequently. The fact that simple gastro-enterostomy is so often completely successful in cases of chronic peptic ulcer would at least serve to discount the malignant degeneration theory. But undoubtedly cancer of the stomach may follow an ulcer, and this realisation must be kept in mind in cases which have failed to respond satis-factorily to prolonged medical treatment.

TREATMENT: Prophylaxis: If the practitioner could persuade all his patients to lead a healthy life from youth upwards, to have dental examination at regular intervals, and all septic foci such as infected tonsils or sinuses

radically dealt with at an early stage, it is very probable that gastro-duodenitis as well as other forms of indigestion would be infinitely less common than they are at present. It follows, therefore, that when a patient consults his medical attendant because of some digestive complaint, and in the routine examination a focus of infection is discovered it is very advisable to have it attended to, as by this means subsequent and more serious disease may be prevented.

The question of surgical treatment: A decision on this point must be arrived at once a peptic ulcer has been definitely diagnosed, and the practitioner should be able to give the patient an idea of the relative prospects of the two forms of treatment. Certain cases, of course, present no difficulty: where there is perforation, or severe deformity of the stomach, or pyloric obstruction, operative procedure is definitely indicated. Again, where time of treatment is of importance, and if the patient's general condition is otherwise satisfactory, an operation would probably give the better result.

Where domestic facilities are lacking, in an uncomplicated case, it may be advisable to suggest radical treatment if there is not a quick response to medical methods. On the whole, I am of opinion that in a working-class practice, cases of chronic peptic ulcer give better results from surgical than from medical treatment, and this I would at-

tribute not to any inherent fault in medical methods, but to the patient, through force of circumstances, being unable to carry out the régime over a sufficiently long period.

It is pertinent to note the results of surgical treatment. The British Medical Association enquiry carried out by A. P. Luff[74] on over 2,000 post-operation cases showed that 90 per cent. of these patients were well 4 to 8 years after operation. It is only fair to add, however, that much criticism has been brought to bear on these figures. Moynihan[75] recommends gastrectomy in gastric ulcer, and gastroenterostomy with destruction of the ulcer in duodenal ulcer, and has had an operative mortality of under 2 per cent. in the former and only one death in 1,000 cases of the latter.

The possibility of the formation of an anastomotic ulcer after a short-circuiting operation is of some practical importance. Thus in a series of 181 cases recorded by Scotson[76] five developed anastomotic ulcers, but as these cases had all perforated before the primary operation one may assume that the patients were a more serious operative risk than the typical peptic ulcer case.

Medical Treatment: Where the patient is unfit or unwilling to submit to surgical interference or institutional treatment, the method I adopt is as follows: He is told that rest in bed for a few weeks is advisable and that he must

coöperate in carrying out the arduous dietetic treatment. I have not found it practicable to give the rather complicated routine of "ulcer diet" as recommended by Hurst: in an institution it could be carried out in comparative ease, but in a working-man's home experience has shown me that unless the directions are simple they will not be carried out. Usually, therefore, I advise the patient to be given 3 pints of milk per day, given in feeds of approximately 8 ounces every two hours. The milk feeds may be modified two or three times a day by substituting arrowroot, or Benger's food for the ordinary milk. Sodium citrate (grains 10) is added to each milk feed to help to neutralise the acidity of the gastric juice, and at the same time to act as an anti-coagulant. Recent observations have shown that individuals with chronic ulcer secrete large quantities of very acid gastric juice throughout the night, whereas little or none is secreted by the average normal stomach. (Hurst[77]) It is advisable, therefore, to have a glass of citrated milk at the bedside, so that the patient can take some during the night if he wakes up.

Such a diet is constipating, so that milk of magnesia is ordered, the dose depending on the severity of the constipation. Being an antacid, it is also of assistance in controlling the hyperacidity.

It is not practicable to have the patient X-rayed at regular intervals, but examination of the stools for occult

blood affords a rough means of gauging whether the ulcer is healing, though too much significance should not be attached to a negative result, as with such a bland diet a minimum of irritation is caused to the ulcer site. A positive result, of course, indicates the advisability of a prolongation of the milk diet. Later, providing the case is progressing satisfactorily, milk puddings are added to the diet - sago, tapioca, rice, curds and cream etc. Toast and fresh butter are then cautiously added to the menu. Fresh fish, tripe, and chicken may be reached by the third week provided the patient exhibits none of the ulcer symptoms, and the stools remain free from occult blood.

Concurrently with the special diet, therapeutic measures are adopted with the object of controlling the acidity, and thus providing the ulcer with a better chance of healing. I have not found it necessary or indeed advisable to prescribe alkalies in the almost heroic frequency and dosage recommended by certain writers, of whom MacLean is perhaps the chief advocate. The powder he prescribed (Sodium bicarbonate ½ ounce, heavy magnesium carbonate 1 ounce, calcium carbonate 1 ounce, bismuth oxycarbonate 2 drachms) in teaspoonful doses is certainly effective in allaying the discomfort and reducing the acidity, but I have obtained equally good results from a powder containing bismuth carbonate, prepared chalk, and the light oxide of magnesia. The prepared chalk has the advantage of being insoluble, so that

after complete neutralisation has taken place in the stomach, the excess of the alkali is not absorbed, and there is therefore less tendency for alkalosis to develop than with the more soluble alkaline salts. A teaspoonful of the alkaline powder is ordered between meals, and also if there is any suggestion of "indigestion" or heartburn. Instead of continuing this alkaline medication for weeks at a time, the administration orally, before meals, of $\frac{1}{200}$ grain of atropin sulphate dissolved in water is a useful alternative method of diminishing the acidity, by lessening the secretion of gastric juice. The dose may be increased till dryness of the mouth occurs. Certain writers suggest the adding of the dry extract of belladonna to the alkaline powder, but I have found it more satisfactory to keep this drug separate, as it then provides an alternative to the alkali, thus rendering alkalosis less likely.

Regarding the duodenal tube for feeding purposes, as recommended by Einhorn, I have had no practical experience of this method, but it is noteworthy that in America, where it was at one time popular, its use is gradually being discontinued. It is not a form of treatment to be undertaken in an artisan household, necessitating, as it does, the tube remaining <u>in situ</u> for days at a time, and besides it seems in itself an unsatisfactory method, as there is nothing in the stomach to neutralise the free acid each time a feed is introduced into the upper bowel via the tube.

Where olive oil can be tolerated by the patient I have found it to be of value. Presumably it acts by causing some pyloric relaxation, thus reducing the pressure in the vestibule. I have not given it in the large quantities suggested by some writers - J. H. Anderson(78) prescribes as much as six ounces a day - but a tablespoonful given twice or thrice daily before a meal is a distinct aid to the medical treatment of a case.

Post operation and post ulcer régime: It is here where the general practitioner can be of service to the patient, by advising him as to his future modus vivendi, with special reference to diet. One often hears the remark after a successful gastroenterostomy that the patient "can eat anything" without experiencing the old pain and discomfort. But I suggest that when a patient has recovered from the operation he should be given a sheet outlining the type of diet he should adhere to more or less permanently. Unfortunately this is not done as a matter of routine, so when the patient returns to his physician it is very advisable for the latter to give the necessary instructions. The following represents the main articles to avoid, and it enables the patient to select a diet for himself with more freedom than if a stereotyped diet is given to him.

Avoid: All raw vegetables. Green vegetables may be taken only if well cooked and thoroughly mashed with butter.

Avoid: The skins and seeds of all fruits; raisins, currants, and candied peel in cakes and

Avoid: porridge, unless cooked from the finest oatmeal.

Avoid: pepper, mustard, excess of salt; salted fish or meat; pork; twice-cooked dishes; curry, chutney, vinegar, sour fruit.

Avoid: alcohol, and tobacco should be strictly curtailed, and completely stopped if there are any symptoms of indigestion.

Avoid: effervescing drinks.

Take: Plenty of vegetable fats: butter and olive oil are beneficial.

Meals should be eaten slowly and thoroughly masticated; and, if possible rest for at least ¼ hour after meals.

If practicable, a glass of citrated milk should be taken in the middle of the morning and afternoon, but where the patient is working, a biscuit may be consumed in place of the milk.

CONCLUSIONS:

1. Medical treatment of chronic gastro-duodenal ulceration is rarely a practical proposition in an artisan household, but where possible it should be given a trial, preferably in an institution, where radiographic progress can be recorded.

2. The failure of medical treatment is chiefly due to inability of the patient to carry out the rigorous and protracted regime.

3. Where medical treatment is unsuccessful or inadvisable, surgical treatment should not be delayed.

4. After operation, dietetic care should always be exercised, as there is the possibility of an anastomotic ulcer developing, whether from the ulcer diathesis of the patient, or from the nature of the operation.

5. By observing the dietetic rules outlined above governing after-treatment, there is a strong probability that a permanent cure has been effected.

Case History.

G.K., male aged 52; iron-dresser in factory; married; of nervous temperament and poor physique, Family history negative. For several years this man had been subject to attacks of indigestion: epigastric pain coming on two to three hours after a meal, the pain being relieved temporarily by the intake of more food. He was not anxious to attend at the infirmary and preferred to have empirical medical treatment, as the administration of antacid mixtures and a bland diet usually allayed his symptoms for some months at a time.

In May 1932 he consulted me, complaining of increasing general weakness, with no particular reference to dyspeptic symptoms. His height was 5 ft. 7 in. and his weight 9 st. 2 lbs., and he stated he was getting thinner. Physical examination revealed a man of emaciated appearance, with evidence of muscular wasting: not only were the trunk and shoulder muscles affected, but the thenar and hypothenar eminences were distinctly atrophied. There was evidence of fibrillary tremors in the larger muscles. Pulmonary examination did not reveal any gross abnormality, and the circulatory system was negative apart from rather soft cardiac sounds. The superficial and deep reflexes were active. The

abdomen did not reveal any abnormality at the time of this examination, and he was free from symptoms of indigestion on this occasion. Blood pressure 140/60; Wasserman test negative. Urine and faeces showed no abnormality.

The case indeed suggested to me an incipient progressive muscular atrophy, and he was sent to the dispensary for examination. The physician there, however, was of opinion that the patient had chronic phthisis, and that this accounted for the wasting. He was X-rayed there in May, and the report stated that "though inconclusive, the plates show increased fibrosis and dullness at the left apex." This diagnosis appeared unsatisfactory, however, as the man had never had any pulmonary symptoms, and he was referred by me to the Medical Officer of Health, who could find no ausculatory evidence of tuberculosis, but the latter decided to have an independent X-ray examination, and on 27th May the following report was received: "There is no evidence of pulmonary tuberculosis. The hilar shadows are somewhat increased, and there is perhaps some increased atriation radiating from there, but as the man is an iron-dresser and works in a dusty atmosphere the condition shown is probably occupational."

After three months, and in spite of tonics and fresh air, the patient showed no improvement, and he was admitted to a medical ward for investigation. Whilst there attention was paid to his history of gastric disturbances, and he was

examined by fractional test-meal and X-ray. I append a photo of one of the films which shows an ulcer niche in the first part of the duodenum. (No gastric abnormality was noted). He was then transferred to a surgical ward, and a gastro-enterostomy was performed. The patient was discharged in February, 1933, when I received a report from the physician, from which the following is an extract: "The patient was admitted to Ward -- on account of wasting and weakness, and the question of progressive muscular atrophy was raised. The progress was not typical and neither were the electrical reactions. Our final view is that the wasting is more likely to be due to the effects of the ulcer."

It is now several months since the operation, and while he has had no gastric symptoms (on the post-ulcer régime), and is taking his food well, there has been no improvement in his weight or muscular development. Indeed when last weighed he was only 7 st. 8 lbs. He has recently been in the medical ward again, where he is now regarded as an intractable case of atypical progressive muscular atrophy. When discharged on 31.10.33 he had had a course of protein shocks with unfortunately no real benefit.

DISCUSSION: I have thought it advisable to record this case rather than one of simple gastro-duodenal ulceration, as it is of interest in that, in this case it must be admitted, I suggest, that the duodenal ulcer, though present, had little

G.K. Ulcer 1st part Duodenum: confirmed at Operation.

if any bearing on the constitutional weakness of the patient, in spite of the opinion expressed on his first discharge from hospital. That there was definitely an ulcer cannot be disputed as it was seen at operation, and while it may be as well that the patient had a gastro-enterostomy performed with destruction of the ulcer, it is still open to question whether the risk of operation was justified when (a) he was not complaining of gastric symptoms at the time or for some months prior to admission, and (b) he had clinical symptoms which at least strongly pointed to a derangement of neuro-muscular function. Indeed I regard this case as one of the few where a prolonged course of medical treatment for the duodenal ulcer would have been justified, my reasons being (i) he was in an institution and in bed for a period of some weeks in any case, and (ii) during that time the effect of dieting with X-ray control could have been readily carried out.

Carcinoma of the Stomach.

Probably nowhere in the whole field of Medicine is it more important to secure an <u>early</u> diagnosis of the condition, if a cure is to be effected, than in gastric carcinoma. And thus it is that there rests with the general practitioner a tremendous responsibility when he is called upon to investigate a potentially malignant case. Even when he realises this it is often far from easy to establish the diagnosis at a suf-

ficiently early stage to ensure a real hope of a surgical cure. I realise that this statement would not be admitted by certain authorities. Thus Bennett[79] states that "in any particular individual the question of whether or not cancer of the stomach is present can be answered accurately in ninety-nine out of every hundred cases." No doubt if every case of indigestion in a middle-aged individual was investigated by the fractional meal, radiography, blood counts, etc., the cases of gastric malignant disease which have been "missed" at an early stage would be very considerably reduced, but I submit that such a routine is impracticable. But what can be done is to keep in touch with the patient until he is entirely free from symptoms. Even when this precaution is taken, however, it is still possible that an undetected neoplasm may exist, as I propose to show in recording details of a case which has provided me with an invaluable lesson as to how tragically insidious gastric cancer may be.

ETIOLOGY: There are still conflicting theories as to the cause of cancer of the stomach. It may be that chronic gastritis is an important factor in some cases - as Hurst suggests - and yet it is a fact that many severe cases of chronic gastritis do not develop malignant manifestations, in spite of the prolonged abuse of alcohol, over-smoking, irregular and hurried meals, etc. Similarly the supporters of the ulcer-cancer theory cannot explain why the great majority of ulcers do not become cancerous, and if they do not admit that this is

so, abundant statistical evidence can be quoted to support it. Indeed no useful purpose would be served by discussing this difficult question further beyond saying that the writer feels an open mind should be kept on this subject until research workers have discovered something more substantial than the present theories.

SYMPTOMS: Anorexia: In my experience the symptom which is most common in all forms of gastric carcinoma is loss of appetite, and it may be present in the earliest stage. It is not, of course, pathognomonic, but if it is associated with anaemia and epigastric discomfort it is extremely suggestive of malignancy. But it may be the only symptom for some weeks - hence the necessity for a thorough clinical examination where anorexia persists in a middle-aged individual, accompanied by some loss of weight.

Pain: I cannot emphasise too strongly that pain may be absent throughout the whole course of the illness until a fatal termination is imminent, though I admit it is more usual for the patient sooner or later to experience a dull, gnawing sensation in the epigastrium which is not relieved, but instead may be aggravated, by the intake of food. The administration of ant-acids does not produce the temporary relief so readily obtained in cases of peptic ulcer. Naturally the site of the growth has some influence on the character of the pain. Thus where the lower part of the oesophagus or the cardia is

involved, an early symptom is dysphagia and pain at the lower end of the sternum: I propose to give details of such a case. Where the body of the stomach is involved pain may not be a troublesome feature until late on in the disease, and even in pyloric cancer it is remarkable how little discomfort may be experienced, though here again it is more usual to get early discomfort than where the growth is not "orificial".

An atypical type of pain may be described as "pseudo-anginal": I have not noticed it referred to in the literature of gastric cancer, but about five years ago I had such a case. The patient, J.G., aged 50, a baker, came to consult me complaining of pain in the praecordial region often quite severe and at times shooting down the left arm. He had lost some weight and had anorexia. I was inclined at first to regard it as a true angina though the circulatory system did not reveal any gross abnormality, and the systolic and diastolic pressures were normal for his age. I could find no symptoms definitely attributable to a stomach lesion, but he was sent to the infirmary where the gastro-intestinal tract was X-rayed, and a gastric tumour was located, later found to be malignant. At the subsequent operation two-thirds of the stomach was resected and he made a satisfactory recovery. It is now over five years since the operation and the patient has enjoyed good health, and he has had no more thoracic pain. We must presume there were no secondary metastases in the chest to account for the pain in the thorax, as the operation would not have re-

lieved him of all symptoms for so many years; and I can only suggest that the gastric lesion was responsible for a rather unusual type of referred pain.

Nausea and vomiting: These symptoms occur sooner or later, being earlier and more distressing as a rule in pyloric cancer. The vomit may contain excess of mucus due to an accompanying gastritis, and the products of fermentation may give the material brought up a foul odour. Visible or occult blood may be present. Nausea is a more common and an earlier symptom than vomiting, which indeed may be absent during the greater part of the illness. Where there is pyloric stenosis, of course, the vomiting of large quantities of fluid mixed with foul-smelling fermenting food characteristic of dilatation of the stomach is a common symptom.

Extra-gastric symptoms such as hepatic pain, etc., may occur in the later stages where there is secondary involvement of other organs.

PHYSICAL SIGNS: In the early stages there may be no abnormal physical signs even after most careful abdominal palpation. Sometimes up till the end of the illness tenderness and pain may not be elicited on palpating the epigastrium, and no tumour may be felt even on deep palpation.

Note should be taken of the weight of the patient, and enquiry made as to whether he has been getting thinner of recent months. It would be misleading to suggest, however, that con-

tinuous loss of weight is a necessary accompaniment of gastric cancer. Unfortunately there may be a temporary improvement in the patient's nutrition even when a neoplasm is present. I say "unfortunately" advisedly, as it may lead one to regard the case in a less serious light because of this increase in body-weight. But later, of course, a progressive emaciation sets in leaving the diagnosis in little doubt. This temporary improvement in certain cases under medical treatment may be attributed to the alleviation of an accompanying gastritis.

Anaemia may be an early sign, and if associated with loss of weight, even in the absence of digestive symptoms, the case should be regarded as potentially one of gastric carcinoma.

Some time ago, however, I had a case which on the above supposition suggested a gastric neoplasm but which fortunately turned out to be otherwise, and it may be of interest to give the salient features:

Mrs I., aged 45, housewife, had an acute tonsillitis in January, 1933. Early in March I was called in to see her, and she stated she had been troubled with indigestion since the tonsillitis, and she felt she was getting thinner. There was loss of appetite, and obvious signs of anaemia. On examination she certainly appeared to be in failing health, and abdominal palpation showed the epigastrium to be uniformly tender though quite soft. The circulatory system showed a definite tachycardia (pulse 120-130) with the sounds weak. At first I was inclined to regard the nausea and weakness as

being secondary to a myocardial condition, but the epigastric discomfort was so much in evidence that I gave her a test breakfast, with the following result: Free HCL 0.0511 per cent.; total acidity 0.1387 per cent. There was no evidence of gross retention of food, and no occult blood. The Gunzberg test was also positive for free hydrochloric acid.

I considered it advisable to carry out a blood examination, with the following result: Red cell count 3,931,250; white cells 6,560; percentage haemoglobin 65; colour index 0.83. The blood film suggested a secondary anaemia.

I was frankly suspicious of a neoplasm, though the cardiac condition rather complicated the clinical picture. She was admitted to a medical ward and was discharged on 25.5.33 very much improved. She was found to be a case of microcytic anaemia with myocarditis. The report stated "No abnormality was discovered in the X-ray findings. Three gastric analyses were carried out; in the first there was a trace of free HCl; in the last two there was complete achlorhydria. Rate of emptying the stomach was normal. In all the blood counts there was slight lowering of the red cell count with a corresponding lowering of the haemoglobin percentage. There is some enlargement of the liver and spleen. She was treated with ferri et ammon. cit. grains 5 thrice daily with ac. hydrochlor. dil. minims 10. Nativelle's digitalin gr. 1/600 was prescribed for the myocarditis." When seen six months after discharge from hospital her improvement had been maintained.

Such a case, I submit, is instructive, in that although it was regarded in a graver light than was justified, it points to the necessity of utilising both radiological and laboratory methods in order to establish a correct diagnosis, where from a purely clinical examination of the patient a prognosis may be given which is later found to require drastic revision.

X-ray Examination and Gastric Analysis: Early malignant disease of the stomach cannot be diagnosed definitely as a rule from simply a clinical examination, but fortunately much help can be derived from radiography and analysis of the gastric contents.

In expert hands radiography may yield results of very considerable value: thus where a "filling defect" is shown to be present in a case which clinically suggests a tumour, the diagnosis is practically confirmed. Indeed the usefulness of X-ray examinations has long passed the controversial stage as regards gastro-intestinal investigations. Critics of gastric analysis are not wanting, though I submit that much useful corroborative evidence may be supplied with the aid of the stomach tube. If gastric analysis were regarded more as a clinical and less as a laboratory procedure a further step would be taken in the earlier diagnosis of cancer of the stomach.

Information of value may be obtained by evacuating the fasting stomach content. This may be done by giving the patient finely divided charcoal in milk overnight, and then passing

a Ryle tube in the morning. If charcoal is found to be present in the material evacuated, along with a relatively high concentration of acid other than hydrochloric, there is definite evidence of at least partial gastric obstruction, resulting in stagnation of the gastric contents.

If, after test-meal examination, free hydrochloric acid is found to be present, it does not rule out the possibility of carcinoma, though at one time achlorhydria was regarded as almost a sine qua non in such cases. A considerable amount of work has been carried out on the subject of anacidity in recent years, and in this connection the researches of Bloomfield and Pollard[80] in America merit some consideration. These workers used histamine as being a powerful stimulus of the gastric secretion. In 56 cases of verified cancer of the stomach anacidity was present in 69 per cent. Furthermore, acid, if present, was found to be greatly diminished. On the other hand, they admit that in a large consecutive series of cases of cancer of the stomach, every degree of secretion up to practically normal will be found.

I have already briefly referred (p. 22) to controversies which have occurred over the question of lactic acid in the gastric contents. The question has been ably dealt with by Davidson and Calder,[81] who state that while lactic acid has no specific relation to cancer of the stomach, it occurs in 50-70 per cent. of cases of gastric cancer and in only approximately 5 per cent. of cases suffering from all other diseases

combined; that lactic acid production depends on the simultaneous occurrence in the stomach, of a suitable hydrogen ion concentration, and a delay in the gastric emptying time; that a combination of these two factors is found ten times more frequently in cancer of the stomach than in all other gastric diseases; and that a positive lactic acid test, while not pathognomonic of cancer, is unquestionably a helpful diagnostic sign. This seems to be a very fair statement of the weight one should attach to the finding of lactic acid in the gastric contents.

When occult blood is found to be present in a case presenting signs of gastric stagnation on gastric analysis, with diminished or absent free hydrochloric acid, the possibility of a neoplasm being the cause should be seriously considered.

To summarise then, so much information may be obtained from stomach analysis when carried out by the clinician, that it should not be neglected in a suspicious malignant case, and when combined with radiography it should be nearly always possible to arrive at a positive diagnosis.

Differential Diagnosis: It is impossible to avoid a certain amount of repetition when discussing this section, but it is probably the most important one as far as the practitioner and patient are concerned. I would suggest that the main conditions from which cancer of the stomach may have to be distinguished are: nervous dyspepsia, peptic ulcer, chronic gastritis, and pernicious anaemia.

Nervous Dyspepsia: While it may be true to state that a patient with this neurosis may have all the subjective symptoms of gastric carcinoma he will usually have other signs of a neurosis. Further, the physical examination will yield a negative result (though this may be the case in early carcinoma). No occult blood in the stools and the absence of gastric fermentation combined with a negative radiological examination should go far to suggest a functional reason for the patient's symptoms. It should be unnecessary to add that persistent nervous indigestion in a middle-aged patient should only be diagnosed as such after the most thorough investigation.

Peptic Ulcer: Typically the differentiation is not difficult, but sometimes it may be almost impossible to distinguish the two conditions. When the pain is periodic in relation to the intake of food, when the appetite is good and where vomiting gives immediate relief from painful symptoms: these factors favour an ulcer diagnosis. But where there is cicatricial thickening in the pyloric region from old ulceration, with consequent stasis and fermentation of the gastric contents, it may not be easy to differentiate, especially where there is occult blood in the stools and slight anaemia. A history of chronic indigestion, however, would tend to suggest the less serious condition as being present. Laparotomy, indeed, may be necessary before a positive diagnosis can be made.

Chronic Gastritis: Here again chronicity may be an aid. With morning vomiting of material containing excess of mucus in a person with an alcoholic history, it may be reasonable to consider gastritis as the cause. But realising that malignancy may follow in certain cases, any aggravation of the symptoms would render a complete examination advisable. Achlorhydria may be present in both conditions, but testing for occult blood at times with negative result would rather discount the presence of a growth.

Pernicious Anaemia: The clinical picture here may simulate a neoplasm of the stomach in many ways: indigestion, anorexia, and anaemia may all be present, but the blood picture - a megalocystosis and high colour index - is helpful in a doubtful case. In addition, if there is evidence of subacute combined degeneration of the cord - paraesthesias, loss of knee jerks, etc., - the diagnosis would point rather to the blood disease. Achlorhydria, of course, is of no help in differentiating, but investigation should be made to find if there is an excessive amount of lactic acid and the products of fermentation, in which case a neoplasm may be the cause of the anaemia.

Of course in all these four conditions gastric cancer may be present as well, but with the aid of gastric analysis and radiography it should be possible in most cases to be reasonably certain, though I admit there will always be an occasional obscure case which requires a laparotomy to elucid-

ate the problem.

There is one further point regarding diagnosis: viz. the advisability of having a blood Wassermann test carried out. I recollect vividly seeing an X-ray photograph of a stomach at a post-graduate lecture-demonstration. The lesion looked like a typical neoplasm, but in reality it was a gumma, and plates taken after anti-specific treatment resulted in the stomach resuming a normal appearance. The patient was present and he stated he had been in excellent health for several years after the appropriate medical treatment.

Prognosis: This is very unfavourable, and I have not had a case which lived for more than eighteen months from the onset of definite signs of malignancy, with the exception of the patient I have referred to with the pseudo-angina pain.

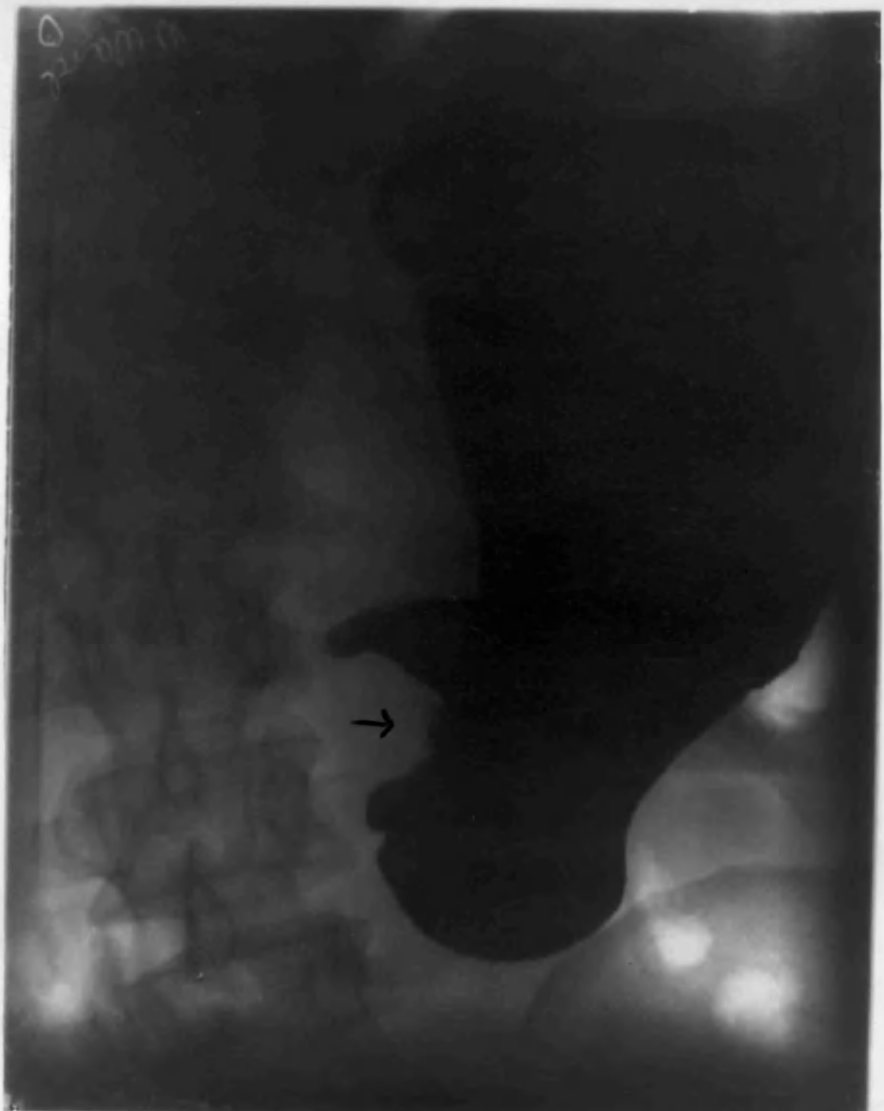
Treatment: Unless the growth is at a very early stage it is doubtful whether the discomfort of a surgical operation is justified. Where there is pyloric obstruction, however, the latter days of the patient may be made easier by a short-circuiting operation. Medical treatment is necessarily palliative, and it is in such a disease as cancer of the stomach where the administration of opium is increasing doses is undoubtedly indicated. As a rule such analgesics as aspirin are unable to afford the necessary relief from the more or less constant pain. The diet should, of course, be bland. Where vomiting is a distressing feature iced

champagne given orally may be tolerated when other forms of alcohol only serve to aggravate the symptoms. Economic considerations, however, sometimes unfortunately preclude this form of palliative treatment.

Case Histories.

CASE 1. J.E., male, aged 52, railway clerk. Family history negative. Since reaching adolescence he had been very healthy. When a boy he contracted some form of "blood-poisoning" in the left leg, which necessitated amputation of the limb at the thigh. He had various scars on the trunk which, he stated were due to the septic condition which caused him to lose a limb. He had enjoyed good health until three months prior to consulting me. At that time he had some domestic worry, and since then he had complained of intermittent "indigestion," with some anorexia. He did not consider he was losing weight. He was an abstainer and a non-smoker.

Physical examination revealed a well-nourished and healthy-looking man 5 ft. 8 inches in height, and 11 st. 6 lbs. in weight. I had known him for a number of years, and certainly from his appearance there was no suggestion of failing health. Heart and circulatory system revealed no abnormality, and the same applied to the respiratory and excretory organs. Abdominal palpation was entirely negative.

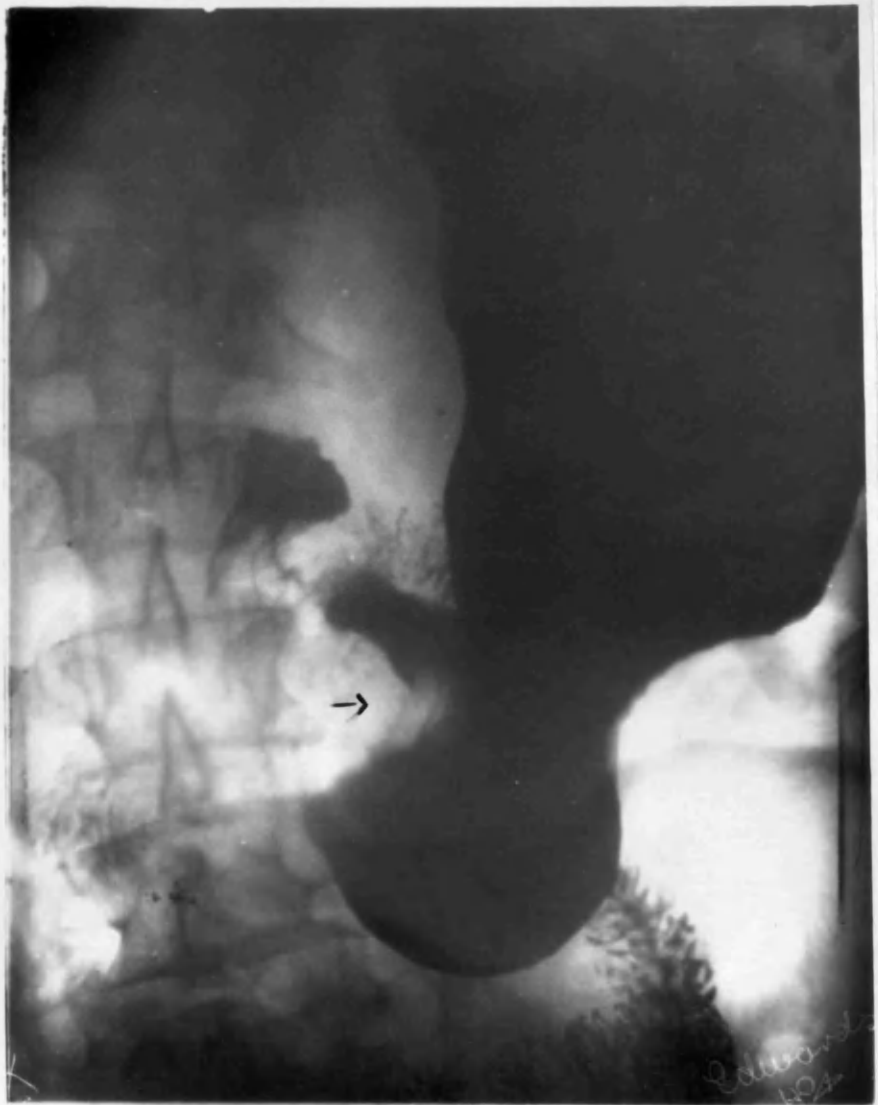


J.E. Filling defect due to pyloric carcinoma.
Taken immediately p.c.

There was no evidence of increased liver dullness; the spleen was not palpable.

In view of the duration of the indigestion I carried out a test meal examination (single-hour method) on 12.5.32 with the following result: Amount withdrawn 40 cc. Appearance normal. Free HCl 0.073 per cent; total acidity 0.146 per cent. No lactic acid; no occult blood. Gunzberg test positive for free HCl. In fact the findings were those one might expect from a simple hypochlorhydria. (There was no sign to suggest delay in the emptying of the organ.) He was put on a mixture containing dilute hydrochloric acid minims xxx and pepsin to be taken with meals and he seemed to improve. His diet was modified to consist of bland non-irritating and nutritious food. Within a month his weight had increased two pounds and he expressed himself as feeling quite fit again.

I did not see him again until 12.9.32, when there was a marked change in his appearance: he was now definitely anaemic, and complained of feeling extremely weak on the slightest exertion. He had no abdominal pain, but loss of appetite was again a feature of the case. A blood examination was carried out, and I found the red cell count to be 3,200,000 per c.mm; white cells 10,000; percentage haemoglobin 65. The blood film suggested a secondary anaemia. He was sent to the infirmary with the request for a gastro-intestinal X-ray examination. The patient was admitted to



J.E. Filling defect due to pyloric carcinoma.
Taken 40" P.C.

a medical ward for observation and on 19.9.32 the digestive tract was X-rayed.

The radiologist's report stated "a filling defect is present at the pyloric quarter of the stomach. The appearances are those of tumour." I append two photos of the films as they serve to show the lesion very clearly. The patient was later transferred to a surgical ward and a laparotomy was performed when "the stomach was found to be the seat of an inoperable carcinoma, and many secondary glands were found in the omentum. The wound was closed without any operation being done. During the convalescence, however, E. felt much better and is under the impression that something has been done to relieve the condition." (This quotation is from the surgeon's report.) The patient was brought home and died early in January 1933. After returning home he was troubled with vomiting but absolutely denied having any abdominal pain though I frequently queried him about this. Also, the abdomen remained soft and palpable and nothing could be felt on digital examination. It was only a few hours before he died that he complained of any epigastric pain.

DISCUSSION: I venture to think that this case presents certain features which are of interest and importance from the diagnostic point of view.

1. The complete absence of epigastric pain throughout

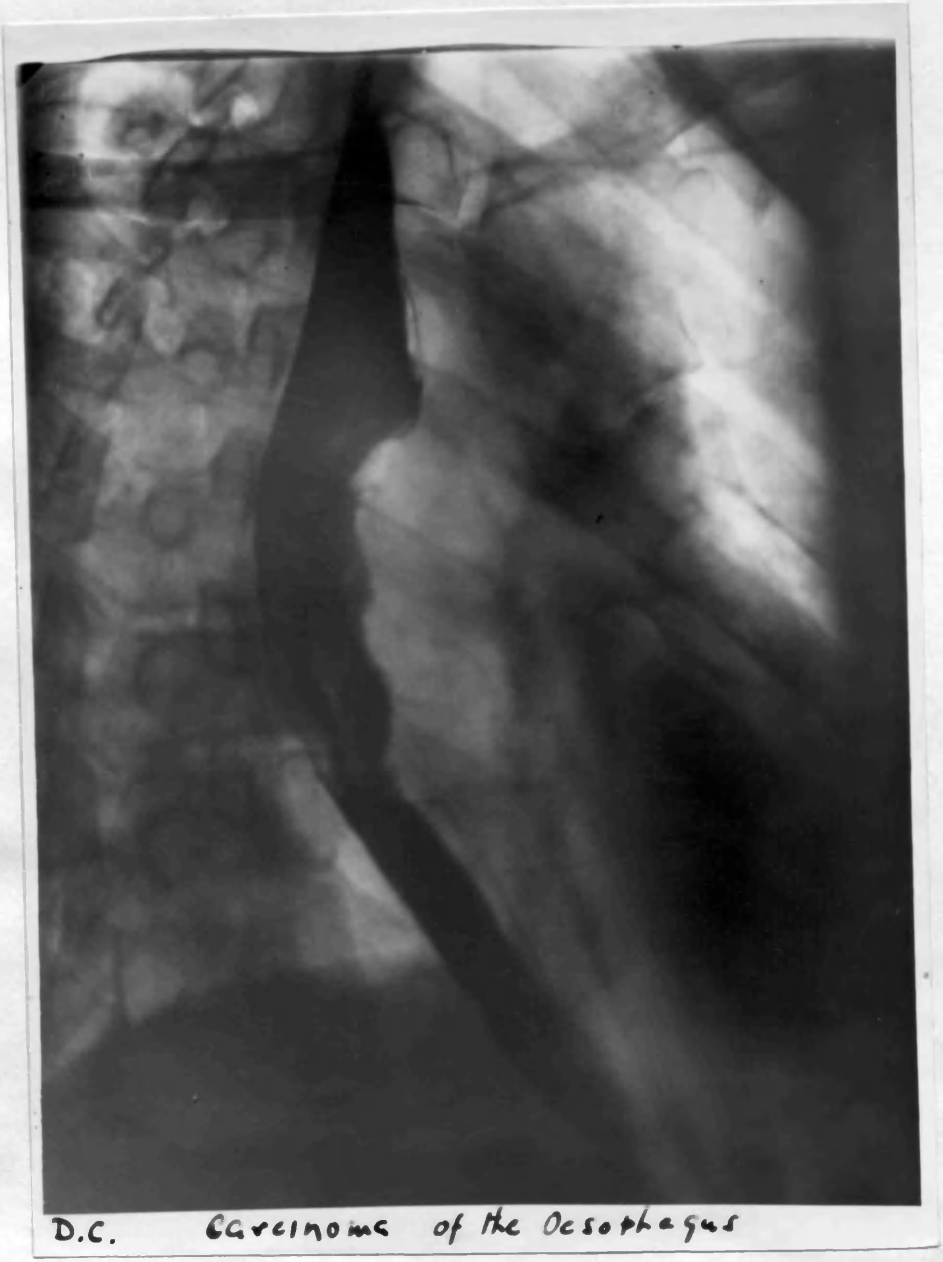
the whole course of the illness, with the exception of the last few hours. (I knew the patient well and have no reason to suspect he was trying to hide any subjective symptoms he may have had.) Indeed, were it not for the blood film, his appearance and the absence of localising symptoms were more suggestive of Addison's anaemia than a pyloric neoplasm. The text-books rightly lay emphasis that where the cancer is "orificial" the signs and symptoms are usually more acute. But in this case, even when the growth was large and inoperable - as the photos show - the patient did not have the copious vomiting so often associated with pyloric stenosis and consequent stagnation of the gastric contents.

2. The temporary improvement and gain in weight were misleading factors after the patient first reported at the surgery and received medical treatment. This, of course, was the time when a thorough X-ray examination should have been carried out, but I confess that the patient appeared to make such progress that I was reluctant to advise what seemed then to be unnecessary. But this case has had the effect of making the writer suspicious of all patients complaining of persistent anorexia associated with a diminished secretion of free hydrochloric acid, where the individual is middle-aged and gives no history of previous dyspeptic symptoms.

CASE 2. D.C., male aged 51, shipyard engineer; married. Family history negative. He had been a healthy man "all his

days" until, about May 1933 he began to have pain in the upper part of the epigastrium and substernal region after swallowing his food. The pain, he stated, frequently went through to the back. When questioned about swallowing, he thought there was slight difficulty some seconds after the food had left the back of the throat. He did not consult me until August, i.e., three months after the onset of symptoms. He said that within that time he had lost two stones in weight. In appearance, however, he was still robust, and weighed 11 st. 10 lbs. His height was 5 ft. 10 in. Respiratory and circulatory systems were negative to examination, and his voice showed no sign of huskiness. On palpating the abdomen he felt some tenderness when pressure was applied just below the xiphisternum.

The rapid loss of weight suggested the necessity for further examination and a test-meal was arranged. On attempting to pass a Ryle tube, however, resistance was encountered after the tip of the tube had reached a point about twelve inches from the mouth. A bougie was next tried with great care, but a definite obstruction was felt, and on examining the top some blood and mucus was found to be adherent to the part though precaution had been exercised to avoid forcing the passage. Organic stricture was suspected, and the accompanying photo of an X-ray film clearly shows the presence of a neoplasm of the oesophagus well to the proximal side of the cardia of the stomach.



D.C. Carcinoma of the Oesophagus

The surgeon to whom I sent the patient did not consider X-ray or radium therapy would be of service, and the latter was sent home with the instruction that he would be readmitted when oral feeding became impracticable. Within two months, however, thoracic and cervical pain became so intense that I sent him into hospital again, and he died within three weeks of readmission of a septic pneumonia. The post-mortem revealed generalised metastases throughout the organs, pointing to a particularly virulent neoplasm, as the total duration of his symptoms was only about six months.

DISCUSSION: While this case anatomically really should be included in lesions of the oesophagus, his subjective symptom of pain was epigastric to begin with, so that it may be fair to assume that the cardia was probably involved in the lesion, though this is not brought out in the X-ray photo. But I have considered it of sufficient interest to record it, from the point of view of symptomatology, as dysphagia was <u>not</u> the most prominent feature in the early stage, but rather epigastric pain.

(b) THE ORGANIC DYSPEPSIAS OF EXTRA-GASTRIC ORIGIN.

In this, the second and last group of the organic dyspepsias, I have thought it advisable to subdivide the section into three parts, viz., (i) Reflex Dyspepsia; (ii) Associated Dyspepsia; and (iii) Simulated Dyspepsia. This division is more for descriptive purposes than practical considerations as of course it is realised that clinically a particular case may have gastric symptoms attributable to more than one of these subdivisions.

(i) Reflex Dyspepsias.

Gall-Bladder Dyspepsia.

By far the commonest form of reflex dyspepsia met with in practice is gall-bladder dysfunction: either due to gall-stones or inflammation of the organ, or both. Indeed many writers, including Alvarez and Hurst, consider that chronic cholecystitis is the commonest form of prolonged indigestion. I propose, therefore, to deal with this condition in some detail. (As cholelithiasis and acute cholecystitis are more in the nature of distinct entities and are less likely to be confused with true gastric dysfunction, the remarks which follow will be understood to refer to chronic cholecystitis, unless where otherwise stated).

Etiology: The exciting cause in all instances is probably infection. Graham and others have emphasised the importance of a primary hepatitis in the causation of gall-bladder disease. While this hepatitis may arise from the absorption of organisms from local infection in the liver-drained area, and may thus follow appendicitis and colitis, there is increasing evidence that in several cases the infection is blood-borne. The work of Wilkie[82] in Edinburgh is important in this connection. He considers that the streptococcus is the most frequent factor in cholecystitis, the B. coli playing only a minor part. The organisms enter the blood stream by some local infective focus in the body - such as septic tonsils - and they may reach the gall-bladder via the cystic artery. It is needless to stress the importance of definitely establishing the truth or otherwise of this assertion as prophylaxis and treatment must be influenced by the result. It is probable that there is some degree of biliary stagnation also present - particularly in cases of gall-stones - and thus it is commoner in individuals who are obese and who lead sedentary lives. Pregnancy may be a slightly predisposing factor, owing to its association with a raised cholesterol content of the blood.

Symptoms: In indigestion due to a chronically inflamed gall-bladder the symptoms may be quite indefinite. Sometimes they may simulate gastric or duodenal ulceration fairly closely, but as a rule the pain is more irregular in

character in that it does not bear the relationship to the intake of food into the stomach which is such a feature of the pain of peptic ulcer. Sometimes the gall-bladder is tender and palpable, in which case the diagnosis is made easier, but more often, in my experience, there is only a history of epigastric discomfort, associated perhaps with occasional bilious vomiting. Vomiting fails to afford the relief to the pain which is so characteristic a feature of ulcer, though it is admitted there is usually some slight alleviation of the epigastric discomfort following upon a copious vomit of bile.

Flatulence is perhaps a more common symptom than in a true gastric dyspepsia. This is usually attributed to aerophagy, but such an explanation does not seem to account fully for the almost constant prevalence of gaseous eructations in gall-bladder disease.

Regarding the association of cardiac disease and cholecystitis I have not been able to satisfy myself that there is a definite relationship. In the obese patient who leads a sedentary life and who has gall-bladder indigestion it is not uncommon to find some degree of myocardial weakness; but this may be due to the general state of the patient's health and not specifically the result of gall-bladder disease. But there are writers who suggest that where there is evidence of cardiac disease and cholecystitis this is an indication for operation. Thus Miller[83] is of opinion that if disease

of the gall-bladder is suspected in a patient who has cardiac pain, and if the suspicion is confirmed by a full investigation, operation should be advised. I venture to suggest, however, that before recommending a patient with cardiac disease to submit to a major operation, a very thorough trial of medical methods should be adopted in the first place, and only where there is evidence of progressive advancement of the disease should recourse be had to more radical measures. On the other hand, it must be mentioned that Hunt(84) states that auricular fibrillation and true anginal attacks are sometimes completely relieved by removal of an infected gall-bladder.

Constipation is a common associated symptom, but it is so usual to find it in ordinary gastric dyspepsia that it has no particular significance as an aid to diagnosis. Headache is fairly common, and frequently follows upon bilious vomiting.

Physical Signs: Local tenderness is very useful when found, but it may be absent. I find it easier to elicit any pain which may be present in the gall-bladder region if the part is palpated when the patient is sitting upright. Even when the patient takes a deep inspiration lying down, the gall-bladder does not descend so readily as when a sitting posture is adopted.

Epigastric tenderness may be found on palpation but

this affords no particular information pointing to cholecystitis. When there is any pain below the umbilicus some complication such as chronic appendicitis should be suspected.

Investigations: While gastric analysis does not provide any specific evidence of cholecystitis or gall-stones, radiography may yield useful results. This is particularly true since Graham and Cole in 1924 introduced their method of indirect cholecystography. At first the intravenous administration of a dye - sodium tetra-iodophenolphthalein - was used, but latterly the oral method of giving the substance was found to be equally satisfactory as regards results, with the added advantage that constitutional disturbance was less likely to follow. The maximum concentration of the dye is found in the gall-bladder 10-15 hours after administration, and the densest shadows are seen in the normal subject. It is, of course, a matter for the expert radiographer to furnish a report on the findings, but the clinical history must be taken into consideration before an adequate interpretation can be obtained. Thus a "negative shadow" in an otherwise clearly defined gall-bladder may suggest a gall-stone, and if there is a history of biliary colic this would provide valuable corroborative evidence. An ill-defined gall-bladder does not necessarily indicate chronic cholecystitis, but here again a careful history should help to bring out the true significance of the X-ray film. On the other hand, an apparently normal cholecystogram does not exclude cholecystitis, though where there is marked

inflammatory change in the organ it is unlikely that a well-marked pear-shaped opacity will be found.

Biliary drainage: The technique elaborated by Vincent Lyon in 1919 is used both as an aid to diagnosis and treatment, but from the point of view of the general practitioner its application is necessarily very restricted as the experience and time required make it unsuitable in general practice. In this respect it has the same disadvantage as the fractional test-meal.

Differential Diagnosis: I find the chief difficulty is usually to distinguish between certain functional disorders and cholecystitis, viz., nervous indigestion and migraine. From organic gastric disease, gastritis and peptic ulcer must be excluded; and finally, chronic appendicitis and spastic colon may sometimes simulate cholecystitis.

Nervous Indigestion: Here a full examination, including radiographic investigation, may be necessary, as the symptoms may exactly simulate gall-bladder disease, but certain stigmata of the neurotic patient - such as an apparent exaggeration of his or her complaints, and an obviously nervous disposition - may suggest the need for fuller examination before organic disease is diagnosed.

Migraine: Where the headache precedes the bilious attack, and where the vomiting quickly relieves the patient of

his constitutional symptoms, migraine is usually the explanation. This is particularly so where there is a history of periodic attacks of this nature.

Gastritis: It is quite possible that in cases of gastritis, especially in the alcoholic type, there may be a degree of hepatitis associated with the stomach disorder, and this in turn may lead to an accompanying gall-bladder dyspepsia. The symptoms are thus confused, though morning vomiting of mucus-coated food would suggest gastric catarrh.

Peptic Ulcer: Here the local tenderness and pain in relation to the intake of food should afford help in distinguishing, and of course X-ray examination may yield useful evidence. Cholecystitis, however, may simulate very closely duodenal ulcer though typically the two diseases are quite distinct. In cases which clinically appear to suggest the latter disease, but which on radiographic examination fail to show any deformity of the duodenal cap, it is my practice to treat them empirically for a time as if they were gall-bladder cases, and the result may justify such procedure in some individuals.

Chronic Appendicitis: The discomfort is usually below the umbilicus, and pain on defaecation and exercise are more suggestive of appendicitis.

Spastic Colon: Here again the pain is infra-umbilical, and, though colicky in character, the right hypochondriac region

is not usually affected. Inspection of the stools for mucus may be of practical help. Both chronic appendicitis and spastic colon will be more fully considered later.

Treatment: In my practice many of my cases present symptoms of gall-bladder indigestion, and though chronic are not so severe as to necessitate a cholecystogram being taken. No doubt it would be ideal to have a full investigation of each case, but hospital facilities could scarcely cope with all such examinations. The patients themselves are quite unable to afford a private radiographic examination, and the consequence is that the majority of these individuals are treated in the first instance more or less empirically and the effects of treatment noted. Where such treatment fails to afford symptomatic relief I usually recommend hospital investigation.

The medicinal treatment briefly consists of modification in the diet, drugs to act as biliary antiseptics, and the prevention of biliary stasis.

Diet: I usually advise limitation of butter, eggs, and animal fats as these are the most important food stimulants of biliary secretion. Where gall-bladder pain is a marked feature these articles are entirely cut out of the diet for a time. Carbohydrates and fruit are allowed, also vegetable purées and soups. Meat and fish are usually well tolerated.

Where biliary stasis is suspected and where fats do not appear to increase the discomfort it may be justifiable to allow them in moderation as they do tend to stimulate biliary secretion, though I prefer to prescribe magnesium sulphate for this purpose.

The biliary antiseptic I employ is hexamine, and this is given in large doses; as much as 50-100 grains with an equal quantity of potassium citrate being administered twice or thrice daily. If the urine is kept alkaline the annoyance of bladder irritation is usually avoided.

Magnesium sulphate given on an empty stomach an hour before breakfast in the largest dose which falls short of producing diarrhoea serves as an excellent promoter of biliary drainage, and as this can be better controlled than advising the taking of animal fats, etc., at meals it seems a more satisfactory method of effecting biliary drainage from the gall bladder. Occasionally, however, I find the magnesium sulphate has a nauseating tendency, and in such a case olive oil ($\frac{1}{2}$-1 oz.) taken before meals may be tolerated. Both the salts and the oil apparently act by causing reflex contractions of the biliary passages and relaxation of the sphincter of the common bile duct on entering the duodenum.

Where there is evidence of gall-stones or gross disease of the gall-bladder the advisability of surgical treatment must be seriously considered as no amount of dieting and therapeutic treatment would be likely to relieve the patient,

and the possibility of malignant disease as a later complication should not be ignored.

In addition to the medical treatment detailed above, search should be made for any primary focus of infection, and the frequent association of cholecystitis with chronic appendicitis and duodenal ulcer should be borne in mind.

Case History.

Mrs B., aged 48, housewife. Family history negative. When seen on 12.4.32 she gave a history of epigastric discomfort of several months' duration. Pain was never severe but most days she had a sensation of fulness in the epigastrium. The pain had no definite relation to the taking of food. She was of stout build, and had always led a temperate life. Constipation was a constant feature. No history of jaundice. Physical examination revealed a well-nourished and healthy-looking woman 12 st. in weight and 5 ft. 8 in. in height. Though somewhat plethoric in appearance her blood pressure was not unduly high. Circulatory and respiratory systems revealed no abnormality. On abdominal palpation there was slight tenderness over the epigastrium and in the right hypochondrium this tenderness was accentuated. The lower abdomen was free from abnormal physical signs. A single-hour test breakfast was given on 13.4.32. Free HCl was present to the extent of 0.073 per cent. and the total acidity was 0.149 per cent.: a slight degree of subacidity.

A tentative diagnosis of chronic cholecystitis was made and she was put on a diet similar to that recommended on p. 223. Hexamine and magnesium sulphate were prescribed and the patient was seen at intervals during the succeeding three months. Her subjective symptom of epigastric discomfort disappeared, and she felt so much better that her diet was later brought back to the ordinary with the exception of the yolk of eggs which appeared to disagree with her.

Discussion: This case is quite a typical one of many, and while it has no particular point of clinical interest I venture to think it is of importance as so many cases of this type are met with in general practice which fail to respond to the usual ant-acid mixtures so frequently prescribed for "stomach trouble". It is perhaps doubtful even yet whether it is sufficiently recognised that many cases of "indigestion" are of this reflex type.

In the case in question no focus of infection was discovered, but the empirical prescribing of hexamine appeared to be justified by the result, though one could not be dogmatic on this point, as the diet and the biliary stimulant magnesium sulphate probably played a considerable part in the patient's recovery.

Chronic Appendicitis.

The importance of chronic appendicitis as a reflex cause of gastric symptoms is now fairly well established,

though it was not until 1910, when Moynihan[85] introduced the term "appendix dyspepsia," that the comparative frequency of the condition became widely recognised in England and America.

Symptoms: Pain. The mere presence of slight tenderness on pressure being applied over the right iliac region indicating perhaps some slight degree of appendicular disease may not necessarily be responsible for the patient's dyspeptic symptoms. But where pressure over McBurney's point causes epigastric discomfort which reminds the patient of his chronic indigestion: this is a valuable point in suggesting the appendix as being the cause of the dyspepsia. The time of onset of the pain is irregular and does not bear a constant relationship to the intake of food. Neither food nor alkalies has the effect of appreciably lessening the discomfort: this is in contrast to ulcer pain.

Vomiting, when it occurs, is reflex in origin, and the gastric hyperchlorhydria which Bonar,[86] as quoted by Hurst,[87] has shown occurs in 88 per cent. of cases of chronic appendicitis with gastric symptoms, is doubtless of similar reflex origin.

While constipation is the rule, it is more often of the spastic type, with occasional colicky pains associated with diarrhoea. There may be pain, chiefly in the right iliac region, during defaecation. The appetite is poor, and nausea may be present even if there is no vomiting.

Physical Signs: Where there is a small localised area of definite tenderness in the right iliac fossa, one is, of course, led to suspect appendicular disease, but frequently I find on examination no such tenderness, but only vague epigastric discomfort. When the examination is carried out during a quiescent period as far as right iliac pain is concerned, it renders a correct diagnosis more difficult to obtain. A diffuse tenderness in the right iliac fossa, on the other hand, does not necessarily indicate appendicitis, as a distended caecum due to a spastic colon or severe constipation may account for the discomfort on digital pressure.

Cases are recorded where there may be pain on straightening or on raising the right leg when in the recumbent position, due to the appendix being adherent to the psoas muscle. While I have not had such a case personally, about six years ago I had one which presented just these symptoms, but due to a different cause. The patient, a boy, J.B., aged 15, had a somewhat diffuse right iliac tenderness which appeared to be slightly fluctuant, and he had the leg pain above referred to. He had no sickness or nausea, and there was no history of indigestion. He was referred to a surgeon who was suspicious of a cold abscess developing, and an X-ray examination of the pelvis and lower spine was carried out. Tuberculous sacro-iliac disease was discovered, and the abscess was the result of this condition. After eighteen months' conservative treatment in a special jacket to effect complete rest for the region

involved, the boy made a satisfactory recovery. The abscess was aspirated. While the physical signs in this case were quite in keeping with an appendicular abscess, it was the absence of typical subjective symptoms which saved the boy from the possibly serious consequences of a laparotomy.

Investigations: X-ray: Enough has perhaps been said above to suggest that I do not consider the diagnosis of chronic appendicitis always an easy matter as a cause of reflex dyspepsia. Before operation, therefore, I deem it advisable to have radiographic confirmation if possible. Screening and palpation of the appendix at the same time may provide useful direct evidence, and the presence of concretions and foreign bodies may be ascertained. Indirect evidence, such as spasmodic hour-glass contraction of the stomach, ileal and caecal stasis may also be provided from an X-ray examination. While the practitioner cannot carry out these investigations, he should provide the consultant or radiologist with the clinical data, as, after all, a conclusive X-ray diagnosis is seldom possible, but it can afford valuable confirmatory evidence.

Gastric Analysis: While it is probably more usual to find a hyperchlorhydria in chronic appendicitis, the converse may occur; and in any case it does not provide us with information pointing specifically to a diseased appendix, where such exists. But where the diagnosis rests between

duodenal ulcer and chronic appendicitis, and chemical analysis shows a subacidity to be present, it is more likely that the appendix is the cause of the symptoms.

Differential Diagnosis: The main conditions to be considered in this connection are those enumerated when discussing chronic cholecystitis, viz., peptic ulcer, colon spasm, nervous indigestion, with, of course, gall-bladder indigestion. The salient features of these diseases have been dealt with on p.236 et seq. A cholecystogram may be necessary to exclude chronic gall-bladder disease.

A history of an acute attack of appendicitis would be a most important diagnostic point, but where this cannot be obtained it should be only after thorough investigation that the indigestion should be wholly ascribed to chronic appendicitis. Indeed, I agree with Muir Dickson(88) when he states "chronic appendicitis does not, as a rule, give rise to signs and symptoms so definite that they can be considered a characteristic of the condition, and of this condition only, and it is frequently only by a process of exclusion that a diagnosis of chronic appendicitis can be entertained as the most probable cause of the signs and symptoms."

Treatment: When the diagnosis is reasonably assured, the treatment is surgical. "Reasonably assured" implies a full investigation, including X-ray examination. Where there is evidence of disease elsewhere, such as peptic ulcer, it may

not be possible to ascertain to what extent, if any, the appendix is responsible, but if a laparotomy is decided upon, the appendix would be removed as a routine procedure at the same time, assuming that it is always a possible source of focal infection.

If the patient's symptoms continue after appendicectomy, it does not follow that the operation has been unsuccessful, as I have often found that the iliac and epigastric discomfort only gradually disappears after removal of the appendix.

In doubtful cases the effect of removal of any septic focus in tonsils or teeth should be tried in the first place. When achlorhydria is present, the administration of dilute hydrochloric acid may be tried, but in my experience this has not benefited the subjective symptoms, though Hurst states it occasionally results in an apparent cure of the discomfort.

Spastic Colon.

Probably there is always an underlying neuropathic tendency in patients with this derangement. It is well known how fear and emotion may cause diarrhoea, demonstrating how stimulation of the higher centres may act upon the colon. These patients are often obsessed with the (erroneous) impression that a daily evacuation of the bowels is necessary, and to this effect they have recourse to drastic purgation which sets up an irritation of the lower bowel, and may result

in the passage of excess of mucus with the faeces.

Tobacco in certain cases is an exciting cause of spasm, and I have personal knowledge of cases where an excessive amount of smoking initiated an attack of colon spasm.

Chronic appendicitis may, through reflex action, set up a spasm. In such a case careful medical treatment of the so-called colitis may fail, owing to the primary cause - the appendix - being untreated.

Of recent years much work has been done on allergy, and it has been suggested by various writers, notably by Rowe, (89) that "food allergy" may play an important part in colon spasm. He suggests that protein sensitization from certain articles of food may lead to an attack.

Symptoms: Though epigastric pain is unusual, there is gaseous distension and discomfort in the upper abdomen, which is reflex in origin. The left iliac fossa may be the site of particular discomfort, and a generalised sensation of dragging in the lower abdomen may be present. Food does not appear to have an appreciable effect on the severity of the discomfort or pain. The symptoms are not constant, and after rather severe spasms of colic, perhaps followed by diarrhoea, the patient may feel reasonably well for some days or weeks. Constipation is always troublesome, being far more persistent than occasional transitory looseness of the stools.

The passage of excess of mucus is not always a marked feature, and the so-called casts of the colon which gave the

derangement its old name of mucous or muco-membranous colitis, is a rare sign, and not essential to establish a diagnosis. In ten years I have not seen a well-marked case.

The diagnosis is based on the neurotic type of case, the history of symptoms just enumerated, and inspection of the stools. X-ray examination may give valuable confirmatory evidence of a dilated spastic colon, but in a general practice such as mine it is not practical to obtain a radiographic examination unless the symptoms suggest some other factor as being present, such as a chronic appendicitis.

Treatment: This form of reflex dyspepsia demands general and local treatment. The constipation obsession must be eradicated if possible, and the patient told not to worry about not having a daily evacuation. Also the habit of auto-inspection of stools should not be encouraged. The actual constipation may be treated on the lines I have already suggested in an earlier section (pp. 154-160). The diet should be modified in accordance with that treatment. The tincture of belladonna in 10 minim doses thrice daily I have found beneficial as an antispasmodic.

Regarding colon lavage, this is not a method of local treatment which I have practised, as the environment of most of my cases is not adapted for such a procedure, and besides, I feel that any line of treatment which serves to concentrate the patient's attention on his alimentary system - as by regular douching - is really defeating its purpose by increasing

the tendency these patients have of becoming self-centred. The oral administration of liquid paraffin or Agarol Brand Compound is useful in helping to soften the scybalous masses in the lower bowel.

Visceroptosis.

While it is probably true that every practitioner has some patients who are obese with pendulous abdomens and ptosed viscera, and who do not complain of any digestive symptoms, I submit that a certain proportion of these cases evince reflex dyspeptic symptoms, which can only be ascribed to this physical cause.

Where the patient experiences relief from abdominal discomfort when lying down, but on assuming the erect position the aching discomfort returns, and when marked relief to the patient is obtained if the physician grips the abdomen from behind and raises the pendulous part - in such cases it seems fair to assume that visceroptosis plays an important part in the patient's discomfort.

Operative treatment has, I think, wisely gone out of fashion for these cases, and reliance is now placed more on graduated exercises to strengthen the abdominal muscles and correct the constipation, combined, in severe cases, with the wearing of an abdominal support. Such exercises as I have mentioned when dealing with constipation (p. 156) may prove helpful.

(ii) Associated Dyspepsia.

The practitioner has constantly to keep in mind that the stomach is the great sympathetic organ of the body whose functions are at once liable to be disturbed when any other viscus is attacked by disease. In the cases of specific fevers, Bright's disease, syphilis, phthisis, and diabetes, it is fair to assume that organic changes occur in the mucous membrane of the digestive tract as the result of the special toxaemias that develop in these complaints. It is obvious, therefore, that no dogmatic opinion should ever be expressed as to the cause of dyspepsia unless a comprehensive examination has been carried out.

I do not propose to deal at length with all the above-mentioned conditions as in most of them other symptoms take precedence over the gastric derangement and indicate the cause of the dyspepsia. In early phthisis, however, difficulty may be experienced, and I have already stressed the importance of a careful pulmonary examination where the patient has gastric atony associated with a slight cough and general debility (p.42).

Regarding diabetes mellitus, the modified diet patients with this disease have to take in some instances appears to upset the digestion, though I have found varying degrees of dyspepsia in diabetics prior to the commencement of treatment, suggesting that the metabolic disturbance has a disturbing effect on the stomach. Some three months ago I saw

a case which I consider of sufficient interest to record in some detail. The patient, a man, J.D., aged 45, had been attended by a colleague of mine about two weeks prior to my seeing him. I understand that the patient's only complaints had been general weakness with loss of weight and appetite. As he had dental caries and extensive pyorrhoea the physician advised the patient to have early dental treatment as a preliminary to general tonic medication. The teeth were extracted, but the patient became rapidly worse and vomiting occurred at intervals. My colleague, after a careful physical examination, could find no organic cause for the illness and intended referring the patient to hospital, but owing to the latter becoming semi-conscious rather suddenly, with sterterous breathing, it appeared as if a fatal termination was imminent. The patient was in this condition when I saw him. He was unconscious, and it was not possible to carry out a satisfactory examination. He was obviously emaciated, the pulse was 120 and thready, and abdominal palpation did not reveal any rigidity or abnormality. The arteries did not appear to be arteriosclerotic. I interrogated his wife, who stated that she thought he had been getting thinner during the preceding few months, but in spite of her advice he would not consult his doctor. The great emaciation suggested a possible malignant condition, but the coma did not "fit in" with such a supposition unless the lesion was cerebral, and the pupils and reflexes did not support this explanation. The urine had not been examined, but I asked his wife whether

he had any urinary frequency. She replied in the negative but said he had had considerable thirst lately. Fortunately he had passed some urine into a bed-pan a few hours prior to my visit, and this was examined. Fehling's solution was rapidly reduced on adding a small quantity of the urine, and the patient was admitted to the infirmary without further delay. He died within 24 hours of admission from diabetic coma.

As far as we know polyuria was not a feature of this case, and the associated dyspepsia with loss of weight suggested a possible gastric cause for his symptoms until the onset of coma. No doubt had the urine been examined as a matter of routine for sugar and albumin the diagnosis would have been made at a stage when appropriate treatment may have averted a fatal termination.

(iii) Simulated Dyspepsia.

Derangements of the respiratory and cardio-vascular systems may at times produce symptoms which not only simulate dyspepsia, but which may occasionally cause the physician considerable anxiety in deciding whether or not he is dealing with an acute abdominal condition requiring surgical interference. Rather than enter into generalisations I venture to think it may be of more practical interest to cite two cases, one respiratory and one cardio-vascular, which may be taken as typical of the occasional diagnostic problems that confront

the general practitioner:

(a) Mrs H.B., aged 48, took ill with a rigor, nausea and vomiting on 27.12.33. When examined her temperature was 100, pulse 110, and she complained of acute epigastric discomfort and slight shortness of breath. Examination of the lungs and heart was negative; there was relative tenderness on palpating over the epigastrium and right hypochondrium. The tongue was moist but furred, and she had vomited a few times. When seen the next day, the nausea and abdominal discomfort were still present, but in addition I thought there was definite diminution of the movement of the diaphragm on the right side during respiration: the left side moved freely. No pleural friction rub could be heard. Respirations were shallow (30 per minute), the temperature and pulse were practically unaltered. I suspected a diaphragmatic pleurisy, and the following day there was some dulness at the base of the right lung, and she had a "rusty" sputum. The epigastric symptoms were less in evidence though nausea continued. She was removed to hospital where the case developed on the usual lines of a lobar pneumonia, and she ultimately made a good recovery.

Discussion: I had known this patient for a number of years, and she was subject to bilious attacks at times with discomfort in the right hypochondrium. It seems fair to suppose that the diaphragmatic pleurisy may have originated

in a perihepatitis, infection of the pleura occurring through the diaphragm. While this case did not simulate an "acute abdomen", nevertheless the symptoms for the first two days were abdominal rather than thoracic, and the dyspepsia tended to obscure the real origin of the lesion.

(b) J.F., a male, aged 50, an accountant by occupation, took ill suddenly about 8 p.m. in May, 1931. He had been in his usual health that day and partook of a fairly substantial supper at 7 p.m. An hour later he developed acute epigastric pain and nausea, followed by copious vomiting. I saw him at 8.30 and he was collapsed, with small rapid pulse, great pallor, and he spoke only with obvious difficulty owing to the severe pain. The abdomen was tender to palpation in the epigastric region, and there was slight rigidity. In addition to these signs and symptoms he had a short irritating cough and a mucous sputum which I noted was definitely blood-stained. Fortunately I elicited the information that three months previously he had had acute praecordial pain which compelled him to stand in the street for a few minutes, and since then he felt he could not exert himself without a tendency to dyspnoea and cardiac discomfort. At that time he consulted another practitioner.

His blood pressure was 120/100 though the radial arteries appeared somewhat sclerosed. Râles were present at the base of the lungs, suggesting an incipient pulmonary oedema. After weighing the evidence of history and examina-

tion I concluded he was subject to angina pectoris and that he was suffering from coronary thrombosis and that the abdominal symptoms were in reality of cardiac origin. Morphia grain ¼ was given but it only afforded slight relief. By 10.30 p.m. the patient had died.

Discussion: It is well known that circulatory disorders very commonly lead to symptoms of indigestion and in certain cases the cardio-vascular disease is entirely or almost entirely masked by the digestive. It is, of course, the question of diagnosis which is of supreme importance, as the correct treatment of the case demands a recognition of the underlying cardiac derangement.

Regarding the above case, in the circumstances it was not possible to carry out a complete examination. The fact that the symptoms commenced after a hearty supper did not necessarily indicate a gastro-intestinal lesion, but rather that an already weakened myocardium had been given too much work to do. The relatively low systolic blood pressure in view of the arteriosclerotic arteries suggested a temporary collapse of the circulation and did not rule out hyperpiesis as a normal condition of this man's circulatory system. The haemorrhagic sputum suggested the possibility of a pulmonary embolism. An electrocardiogram may have provided useful information, but there was no time for fuller investigation. I may add I later communicated with the practitioner who had examined him three months previously and a diagnosis of

angina pectoris was made at that time.

Other cases of this type could be quoted, where a fatal termination did not follow, and where in consequence a fuller examination was carried out, but they are rather outside the scope of this paper, which is primarily concerned with true dyspepsia. At the same time, however, I have thought it quite relevant to discuss one case, however briefly, as I hope it will serve to show how important it is in dealing with any apparent case of gastric derangement to go into the past history. By so doing the chances of making an incorrect diagnosis may be appreciably reduced.

SUMMARY AND CONCLUSIONS.

This thesis is an attempt to deal with that group of diseases - both functional and organic - comprised in the word Dyspepsia, regarded from the point of view of a general practitioner in an industrial practice. While the basis of the material contained therein is essentially clinical, and founded on the writer's personal experience, the scientific side of the subject has not been disregarded, and modern views on etiology, etc., have been indicated where the necessity seemed to arise. Particular attention has been devoted to the functional side of dyspepsia, as this has always seemed to me a much neglected branch of the subject, and yet I would estimate that little short of half my consulting time is taken up with cases suffering from derangements of gastro-intestinal function. The following numbered paragraphs will serve to show, I hope, the salient features of each section of the thesis.

1. INTRODUCTION: Emphasis is laid on the fact that though Dyspepsia is such a common complaint in general practice - particularly the functional type of indigestion - very little of this subject is taught to the medical student, as these cases do not, as a rule, reach hospital wards. The classification of the types of dyspepsia which I have adopted is outlined. (pp. 1-5)

2. The routine procedure I adopt in investigating patients with dyspeptic symptoms is reviewed at length, emphasis being paid to the great importance of obtaining a complete history of the case. Reference is made to a suitable chart for taking down case-particulars, and a copy is enclosed. Regarding the "special" examinations in gastric cases, the importance of the practitioner doing the gastric analysis himself is discussed, and the view is expressed that this form of investigation does yield useful results, in spite of a tendency to belittle this method nowadays. The single-hour and fractional tests are dealt with and their relative advantages discussed. The importance of testing for occult blood where organic disease is suspected is mentioned. The present-day position of radiography in the field of gastro-enterogical investigation is outlined. (pp. 6-33).

3. THE FUNCTIONAL DYSPEPSIAS: Under the caption "General Considerations" I have endeavoured to show the difficulty experienced in reaching a satisfactory classification at once practical and of use for descriptive purposes. The importance of instability of the nervous system as an etiological factor in most functional dyspepsias is emphasised, and attention is drawn to the considered opinion of the writer that during the recent years of unemployment cases of functional dyspepsia have relatively increased, and this is ascribed to worry rather than to actual insufficiency of

food. Mention is made of the nerve supply to the stomach, and its association with the motor and secretory activity of the organ. This slight anatomical and physiological digression was considered necessary owing to the intimate relationship between functional dyspepsia and the nervous supply to the stomach. (pp. 34-39).

4. MOTOR NEUROSES: The various types of this form of functional dyspepsia are considered in detail:

(a) Gastric Atony: Both primary and secondary are dealt with, and in the treatment, where I consider dietetic measures of the first importance, I have stressed the advisability of giving each patient a diet-sheet with full instructions, as personal experience has shown me that no amount of talking indefinitely on dietetics is of much practical use to the average working-class patient. A specimen diet is given. Three cases are taken and discussed in some detail, the third one showing the importance of early phthisis as a cause of gastric atony.

(b) Idiopathic Achalasia of the Cardia: The difficulty the practitioner may have in diagnosing this condition from early organic disease of the lower end of the oesophagus is dealt with, and an illustrative case given.

(c) Aerophagy: This is discussed and a typical case mentioned.

(d) Pylorospasm: The importance of endeavouring to find a cause for pylorospasm is emphasised, as primary idiopathic pylorospasm is a rare disease. Two cases are given in some detail, the second one being perhaps of more interest, as exemplifying haematemesis due to pylorospasm in a tabetic crisis. (pp. 40-77).

5. SECRETORY NEUROSES:

(a) Hyperchlorhydria: The modern conception that hyperchlorhydria is not a pathological entity is discussed, and a reasoned suggestion made that at times it is a condition which per se is capable of producing symptoms. Full details are given of the writer's dietetic treatment, and the moderate administration of sodium bicarbonate (with other drugs) is advocated, in spite of the chemical fact that it gives rise to a secondary increase in the amount of free hydrochloric acid in the stomach. Two cases are discussed in detail with the result of treatment.

(b) Hypersecretion: The importance of regarding chronic hypersecretion as a manifestation of organic disease is emphasised, as also is the fact that investigation with the stomach tube is essential before reaching a positive diagnosis. Lavage as a form of treatment is discussed. Full details of a case are given in which I suggest the chief interest depends on the fact that both single-hour and X-ray examination failed to reveal what was afterwards confirmed

by fractional test and second radiographic examination, viz., hypersecretion due to juxta-pyloric ulceration.

(c) Hypochlorhydria: This is very briefly mentioned as I have not satisfied myself that, where it falls definitely short of achlorhydria, it is of much clinical significance.

(d) Achlorhydria and achylia gastrica: These terms are defined to prevent confusion, and mention is made of the fact that with the modern use of histamine injection true achylia gastrica is found to be a rarer condition than was formerly regarded as being the case. The relationship between achlorhydria and pernicious (Addison's) anaemia and microcytic anaemia is dealt with, and at the same time it is noted that achlorhydria may be perfectly simple in type, with no serious significance. Under treatment the writer's method of dieting is given in detail, and a note is added regarding the relative merits of desiccated hog's stomach and liver extract in pernicious anaemia from the point of view of the dyspepsia in these cases. Details of a case of simple achlorhydria are given. (pp. 78-112).

6. SENSORY NEUROSES: This section deals in detail with the protean manifestations of nervous indigestion and a plea is made for its recognition as a clinical entity. Emphasis is laid on regarding the cases comprised in this group from the psychological aspect, and the method I adopt is fully dealt with. The subdividing of cases of gastric

neurasthenia into several divisions is deprecated, and reasons are given. Three cases are mentioned in detail and each is discussed from the point of view of symptomatology and diagnosis. (pp. 113-131).

7. DYSPEPSIA of INFANCY and CHILDHOOD due to FAULTY DIET: Of the innumerable methods of artificial feeding in vogue, I have given full details of the one I adopt, particular regard being paid to the quantity of milk for the weight of the infant. An actual case is cited to illustrate the way the calculation is made. The carbohydrate dyspepsia of childhood is next considered, and the treatment the writer adopts is outlined. Attention is drawn to the similarity between carbohydrate dyspepsia and tuberculosis, and the differential diagnosis discussed. Reference is made to ketosis and cyclical vomiting as manifestations of metabolic disturbance in later childhood, and prophylaxis is considered. Cases are mentioned in the course of this section which have some practical bearing on the dyspepsia of childhood. (pp. 132-150).

8. CONSTIPATION in RELATION to the FUNCTIONAL DYSPEPSIAS: This complaint is so frequent in cases of dyspepsia that I have thought it advisable to devote a small section to it, detailing the method I adopt as regards treatment, with particular reference to diet and abdominal gymnastics. A chart of exercises is included. (pp. 151-160).

9. THE ORGANIC DYSPEPSIAS OF GASTRIC ORIGIN: This section deals more particularly with the diagnosis of organic disease producing dyspepsia. Treatment is considered in so far as it can be carried out in the patient's home. The usefulness and limitations of gastric analysis and X-ray investigations are briefly discussed in the introduction to this section, before considering the individual types of organic disease.

(a) Gastritis: Acute and chronic forms are discussed. The possibility of confusing a pin-point perforation and acute gastritis is mentioned, and a case illustrating such a diagnostic difficulty is cited. Full details of the writer's dietetic treatment of chronic gastritis are given, and the utility of lavage is discussed. A typical case of chronic gastritis is described.

(b) Peptic Ulcer: Modern views as to etiology are discussed, with particular reference to Hurst's theory of gastric diatheses. Under "symptomatology" the possibility of haemorrhage occurring during a quiescent period is noted and a case mentioned. Special methods of investigation are outlined. Differential diagnosis is dealt with at some length, as it is of practical importance from the general practitioner's point of view. It is emphasised that nervous indigestion may simulate peptic ulcer closely, and a case is quoted. Particular importance is attached to early malignant

disease of the stomach, and its possible confusion with peptic ulcer. Complications are considered chiefly in so far as diagnosis is concerned. Treatment is dealt with in its broad aspect and points in favour of medical or surgical methods are outlined. I hold that in the case of the average working-man surgical treatment offers the better prospect of a cure from peptic ulcer: reasons for this opinion are given. The writer's method of dealing with these cases medically is fully given. Under "case history" I have ventured to consider it of more interest to select a case of peptic ulcer which afforded some difficulty in diagnosis, rather than a "straight" case of ulcer. An X-ray photograph of the site of the lesion is given. This case is discussed from a general point of view after full details have been given.

(c) Carcinoma of the Stomach: As early diagnosis as a prelude to treatment is the only hope for cases suffering from this disease, the symptomatology has been considered in some detail. Particular note is drawn to the fact that pain is not necessarily an early symptom. A case having an atypical form of pain - pseudo-anginal - is mentioned. It is pointed out that under medical treatment there may actually be a temporary gain in weight even in cases of cancer of the stomach. While anaemia and loss of weight with or without digestive symptoms form a suspicious combination of clinical signs, it does not follow that a malignant growth exists: a case illustrating this is given. The modern view of the

position of radiography and gastric analysis is given, and regarding the latter, reference is specially paid to the significance of lactic acid from the diagnostic point of view. The differential diagnosis is considered in detail. Two cases are given, the first of which I venture to think is of some interest from the fact that pain was absent throughout the illness though the growth was pyloric, as seen in the photographs which accompany the case-history. The second case, though really an oesophageal cancer - as shown in the X-ray photograph - was somewhat atypical, in that dysphagia was not an early symptom, but rather epigastric pain. (pp. 161-230).

10. THE ORGANIC DYSPEPSIAS OF EXTRA-GASTRIC ORIGIN:

(a) Reflex Dyspepsia: Gall-bladder dyspepsia is considered first as being the commonest form of chronic indigestion. The improved method of investigating cholecystitis and cholelithiasis since the introduction of the Graham technique is mentioned, and at the same time its limitations are discussed. Medical means of promoting biliary drainage and the use of biliary antiseptics are referred to, while attention is drawn to Vincent Lyon's technique for biliary drainage and treatment. The practical difficulties of utilising Lyon's method in general practice render it rather unsuitable as a remedial measure outside an institution or hospital, in my opinion. A case of chronic

cholecystitis is described. Chronic appendicitis, visceroptosis, and spastic colon are discussed in this order, particular attention being paid to diagnosis.

(b) Associated Dyspepsia: Attention is drawn particularly to the association of gastric derangement and early pulmonary tuberculosis, and reference is made to a typical case. The connexion between dyspepsia and diabetes mellitus is mentioned, and details are given of a case of diabetic coma which earlier had manifestations of acute gastric disturbance.

(c) Simulated Dyspepsia: The statement is made that derangements of the respiratory and cardio-vascular systems may at times produce symptoms which not only simulate dyspepsia, but which may sometimes cause the practitioner considerable anxiety in deciding whether or not the case is an "acute abdomen" requiring immediate surgical interference. Two examples of simulated dyspepsia are discussed, one of diaphragmatic pleurisy followed by pneumonia, and one of angina pectoris due to coronary thrombosis. (pp. 231-256).

11. I have endeavoured to include in the Bibliography all references to papers, monographs, and books, I have made in the course of the thesis. For the clinical data and observations on cases I am alone responsible, but I have derived much help in following up cases referred to hospital from reports received from the infirmary physicians and surgeons.

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