THE DIAGNOSIS of PEPTIC ULCER in GENERAL PRACTICE

An Analysis of 183 Cases of Gastro-Intestinal Ulceration with particular reference to the Gregersen Benzidine Slide Test

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- C O N T E N T S -

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I. The primary purpose of this thesis is to demonstrate the value in General Practice of the Gregersen Benzidine Slide Test for occult blood, and to draw attention to the fact that it is as simply and rapidly performed as the usual consulting-room tests for albumin and sugar.

It is urged that as in the presence of certain clinical symptoms the urine is routinely examined for albumin and sugar, so in cases of alimentary dysfunction not quickly responding to the simplest treatment, it is an equally necessary precaution for the practitioner to test the faeces for occult blood.

II. The history and rationale of the test are described, and a review has been made of the literature reflecting authoritative medical opinion concerning its reliability.

III. Finally, by a series of cases collected in general practice over the past decade, an attempt is made to assess its value and interest in the day-to-day work of the general practitioner.

The large number of men and women rejected, or subsequently discharged from the Forces because of peptic ulcer, and the recent contributions of Tidy (1946) and Illingworth (1944), among others, have revealed the unsuspected numbers of peptic ulcer cases which existed in the general population prior to the Second World War.

While the mortality rate is known, the total incidence of peptic ulcer throughout the whole, as distinct from the insured population, is not known, and the only means by which these figures could be accurately discovered would be by a survey of their cases by all general practitioners throughout the country. This, of course, would be difficult, and in ordinary circumstances might have been almost impossible but for the fact that in 1941 regulations were introduced providing for everyone who suffered from peptic ulcer, and certain other conditions, an extra allowance of milk and eggs. This immediately drove to their doctors practically everybody suffering from indigestion of any duration. On the practitioners fell the responsibility of deciding which cases came within the letter of the regulations.

In common with other doctors throughout the country, I was immediately besieged by a rush of applicants for the extra allowance, and faced with the difficult task of conforming with

the spirit of the regulations, and at the same time dealing justly with each applicant.

Over a period of many years, in the hope of detecting early alimentary neoplasms as soon as possible, and also as an aid to the diagnosis and follow-up of active peptic ulceration, I have routinely used the Gregersen Benzidine Slide Test for occult blood. The ingredients were simple and easily available, and the test itself simply and rapidly carried out in a minute or two. Now, because of the large number and variety of dyspepsias which suddenly presented themselves to claim the extra ration, the test was to prove of the greatest value.

Many colleagues had been called to the Forces, and because of the extra work created by the filling up of numerous and necessary certificates, and other requirements created by the war, it was not at first possible to apply the test to every applicant. Many cases had, of course, been previously diagnosed, and of the others only those with symptoms of more than a few weeks' duration, or with a long and recurring history of digestive upsets, were Gregersen-tested in the first few weeks. When the pressure of the first flood of applications had eased, it was possible to go more closely into the history of the less persistent complainers, and in time to test the faeces of every patient whose dyspeptic symptoms had lasted more than about ten days on a light diet, with no medicine of any kind except an aperient when indicated.

In the hectic early days of the war, it would have been an imposition on the already overworked staff of the Out-Patient and X-Ray Departments of our Hospitals indiscriminately to have sent for X-Ray investigation every applicant for extra rations. When, however, the necessary precautions in the way of diet having been taken, occult blood was demonstrated in the faeces, it was essential to have an X-Ray confirmation of the clinical findings. By the use of the Gregersen Slide Test it was possible to come to an exact diagnosis earlier than if the patient's symptoms had been the only guide, and at the same time to relieve the Out-Patients Departments of much unnecessary work when they were least able to cope with it.

As a result of these investigations, there was revealed in my practice an incidence of peptic ulceration which, if it is in any way representative of the incidence among the general population, presents a problem of the most serious clinical and social importance.

TESTS FOR OCCULT BLOOD

Although Weber in 1893 had already introduced the Guaiac test for the chemical detection of small quantities of blood, it was not until 1901 that Boas introduced the conception of occult blood in the faeces and recognised its importance as an aid to the diagnosis of ulcerative conditions in the intestinal tract.

Occult blood is the term applied to blood in the faeces which is so changed during its passage through the alimentary canal (chiefly because the red cells are broken down and the

haemoglobin converted into haematin and haematoporphyrin) as to be unrecognisable by macroscopic or microscopic means.

Barker (1916) states that as much as 5 per cent. of blood in the stool may pass unnoticed by the naked eye.

Nearly all the chemical tests for occult blood in the intestinal contents are based on the detection of small amounts of a catalyst which is present in blood and some other organic materials. A colourless aromatic compound such as guaiacum, orthotolidin, reduced phenolphthalein, or benzidine, which become highly coloured when oxidised, is added to the material to be tested. When a peroxide is added, haemoglobin or any of its iron derivitives act as a peroxidase, thus enabling hydrogen peroxide to oxidise one of the colourless substances. In the Gregersen Slide Test the colourless substance is benzidine, an aromatic diamine which is oxidised to produce a highly coloured quinonoid product.

The use of benzidine for the detection of occult blood was first introduced by O. & R. Adler in 1904, but the method described by them was shown to be unsuitable for clinical use. The test as at present used was elaborated by Schlesinger & Holst (1906). It is extremely sensitive and gives a positive result in the presence of comparatively small amounts of ingested meat. It is pretty generally used in biochemical laboratories, and is described in the standard works of Stewart & Dunlop, Harrison and other textbooks of clinical chemistry.

The tests for occult blood in the faeces are very numerous, and their modifications even more so. For practical clinical purposes, the benzidine and guaiac tests were for a long time the two most generally used. In spite of Hurst's (1929) preference for the guaiac test, his co-worker, Bell (1923), at the New Lodge Clinic, showed the benzidine test to be the more effective, and time and experience have proved this to be so.

The first and perhaps the most thorough investigation in this country into the history and possibilities of the many tests for occult blood was made by Leech (1907) in a thesis which he submitted for his Doctorate in Medicine at Cambridge. He there surveys most exhaustively the literature on the evolution of the chemical tests, and obviously derives much of his enthusiasm and inspiration from the writings of Boas. He himself gives credit to two Americans, Steel and Butt (1905) for the first contribution in the English language on the subject. Leech used the Schumm and Westphal modification of the benzidine test. This was soon to be replaced by the method of Schlesinger & Holst, referred to above.

Passing from one reference to another in an endeavour to follow the development of these chemical tests for occult blood, I came upon the St Bartholomew's Hospital Reports for 1909, and there found another most careful and well documented study of the then known tests for occult blood

in the faeces. The author, under careful clinical conditions, had made an evaluation of Van Deen's guaiac test, the benzidine test of Schlesinger and Holst, the Aloin test, Teichman's test for haematin crystals and Weber's spectroscopic test.

Of these he found the benzidine test the most delicate of all, but occasionally, as has since been frequently confirmed, both in this country and in America, giving faint positive reactions with normal stools.

The guaiac test he thought the most reliable because it never gave a false positive except after the taking of iodides. It has been demonstrated comparatively recently by Johnson & Oliver (1941) that iron in quantities sufficient to produce a tarry stool may also give a false positive with the guaiac test, although it is much less sensitive to the ingestion of haemoglobin-containing foods.

The contribution to the St Bartholomew's Hospital Report of 1909 was entitled "The Detection of Blood Pigment in the Faeces", and the author was a young clinician, A. J. Clark, doing his first year of hospital work, who, by his industry and keenness, that year won the Senior Entrance Scholarship in Science and the Bentley Prize (for reports of cases from the wards of the hospital during the preceding year, with comments thereon). It was the first of the many valuable and outstanding contributions which the late Professor A. J. Clark was to make to medical science.

Here was already evident that combination of close scientific observation and clinical integrity which found its most complete expression in his "Applied Pharmacology", which by "bridging the gap between the science of pharmacology and the art of therapeutics" did so much to put prescribing on a more rational basis.

In 1909 Clark demonstrated in a series of cases - his own and those of others - his keen appreciation of the clinical importance of the tests for occult blood in the faeces. It was a subject which continued to hold his closest interest, as was shown in 1914 when he gained his Doctorate in Medicine at Cambridge with a thesis entitled "The Pigments present in the Faeces".

Many clinicians do not favour the Benzidine Test of Schlesinger & Holst because of its extreme sensitivity. Walter (1910) states that it gives a positive result in blood diluted to 1 in 250,000 parts of water. In view of this sensitivity, its routine use in consulting room work can be unreliable because positive results may be given, as previously suggested, when blood is not present in pathological amount.

Coopman (1921) and Gregersen (1919) after many tests state that less than 1 in 3,000 to 1 in 20,000 parts of blood in the stool cannot be considered pathological.

The personal factor also enters into the interpretation of the test, as accurate reading requires some practice in mixing the reagents and estimating the results.

For these reasons, but mostly perhaps because X-ray technique has become so reliable and so readily available as a short cut to diagnosis, the benzidine test, in hospital practice particularly, is not now so indispensable as it once was.

In general practice, however, where X-ray is not always easily available, and where there is presented the earliest opportunity for detecting disease in its incipient stages, a reliable test for occult blood is of the highest value. There are occasions, as will later be shown in Section II, when the Gregersen Slide Test has given positive results at a stage when lesions were not yet demonstrable by X-ray.

It may be contended that no test is too sensitive provided it does not give false positive results, since in many instances even a minute amount of blood in the faeces may be of serious significance. Nevertheless, a test which gives positive results with gum bleeding and after the eating of meat-containing food in moderate amounts, creates difficulties in general practice which might well dissuade general practitioners from using the test as a routine procedure.

To overcome this difficulty, Gregersen (1919) introduced his Benzidine Slide Test. He found (1916) that the catalysing

power of the normal stool did not exceed that of 0.03 per cent. of blood in boiled faeces. By reducing the routine benzidine test to one-third of its normal sensitivity, making it sensitive to not less than 0.1 per cent. of blood in the faeces, he suggested that for everyday clinical use, this modification could be used as a reliable test for pathological amounts of faecal occult blood.

Technically the test discards the use of the unstable hydrogen peroxide and, following the suggestion of Grunwald (1907) substitutes the more stable barium peroxide in a concentration of 4 per cent. Using a 50 per cent. acetic acid solution the benzidine concentration is kept at 0.5 per cent. The practical application of the test thus standardised is simple, and leaves no room for error. I have used it in the cases presently to be described, and have conformed to the technique described in "Clinical Methods" by Hutchison & Hunter (1935). It is carried out as follows:

Powders are made up containing 0.2 gram of barium peroxide and 0.025 gram of pure benzidine; if put up in wax papers they will keep indefinitely. Just before the test one powder is dissolved in 5 c.c. of freshly-prepared 50-per cent. acetic acid solution. A "button" of faeces is taken by means of a glass rod from the centre of the stool and smeared on to a clean glass slide. A few drops of the solution are then run on to the smear. A blue or blue-green colour develops within a minute if the test is positive, and the reaction is graded according to the depth of the colour and the time it takes to develop. Gregersen recognises three grades: 1 +, a pale blue or green

colour within 60 seconds; 2 +, a definite pale blue in 12 to 15 seconds; 3 +, a deep blue within 3 seconds.

The patient having been instructed to bring a small button of faeces, about the size of a bean or a pea, in a clean small jar or tin - even a matchbox will do - the test can be rapidly and easily performed during the course of the consultation. As has been suggested, it is no more complicated, and takes no longer, than testing urine for albumin or sugar. The powders, if preferred, can be made up in bulk, and kept in a dark and airtight bottle in which I have found they will keep, without deterioration, for at least six months. I have had the powder tested after that period, and found that it still gave a positive reaction in a dilution of blood in water of 1 - 70,000. For those doing the Slide Test for the first time it might be wise to have the individual powders made up separately as described in Hutchison & Hunter, till some idea is gained of the quantity appropriate for each examination. It does not take long to learn the approximate quantity required and to tip it into the test-tube, either directly from the bottle, or by using the tip of a spatula. There is no soiling of test-tubes with a faecal suspension, but a time-saving precaution is immediately to rinse out with water the test-tube containing the reagents, which do not keep in solution, and which if left in the tube, leave a brown stain on the inside, which is afterwards more difficult to remove. As only a small smear of

faeces is used, it can be easily cleaned from the slide with a piece of cotton wool. In carrying out the test, I have found the slides used by bacteriologists for hanging-drop cultures most useful. The benzidine solution is less likely to run off the slide, and a greater concentration is brought into contact with the faeces.

Although Gregersen's benzidine slide test is modified to exclude any error due to the taking of such precautions, there is still no unanimity of opinion as to whether the taking of haemoglobin- and chlorophyll-containing foods for several days previous to performing the slide test, invalidates its accuracy. Boas (1921) though he recommends the Gregersen test, nevertheless excludes meat, fish and certain vegetables from the diet. Ogilvie (1927) has tested it extensively, and is satisfied that a blue or blue-green colour appearing within thirty seconds is proof positive of the presence of occult blood, irrespective of the patient's diet. This excludes, of course, black pudding, liver, bone marrow and other foods with a high blood pigment concentration. He anticipated Johnson & Oliver by showing that iron, even in high concentration, will produce no positive reaction. Although he believes the orthotolidin test introduced by Ruttan and Hardity (1912) to be the most highly sensitive of all, Kieffer (1934) recommends the benzidine slide test for ordinary clinical use. Aaron (1924) advises it, and Bockus (1946) gives it prominence. Meulengracht and Jansen (1929) stress its importance, particularly in the diagnosis of gastro-intestinal neoplasm. It seems reasonable

to accept, therefore, that a considerable weight of authoritative opinion in Britain, America and the Continent is agreed that the benzidine slide test of Gregersen is reliable and well suited for routine clinical purposes.

Ogilvie states that the majority of British observers still hold the view that haemoglobin-containing diet may falsify the Gregersen Test, and quotes Coope (1920), Hurst (1925) and others, who rigidly exclude such foods for at least three days before the test, and affirm that without such a precaution the tests are valueless. A notable exception is Maclean (1928) who categorically maintains that no such precautions are necessary. No records of any clinical investigations supporting their view are quoted by these authorities. Nevertheless, on the Continent, where the problem has been studied on a more experimental basis, the general opinion is also in favour of a restricted diet prior to the test. It is important to make clear to the patients that not only meat but meat extracts and meat-made soup are to be included among the prohibited foods. Capt. R. M. McKay (1916) when testing a number of commercial so-called meat extracts, found that while some gave, as expected, an intense positive reaction with the ordinary benzidine test, others yielded a negative result, thus demonstrating in these preparations the absence of any animal tissue at all. Explicit instructions, if the exclusion of haemoglobin-containing food is considered necessary, must, therefore, be given. I have often found that patients fail

to realise that chicken, rabbit, tripe, and even pork, have to be excluded. The question of fish, particularly in these days, when the supplementing of meals excluding meat is so difficult, invariably arises. Capt. R. M. McKay in his investigations, using his own modification of the Schlesinger & Holst test, found that an emulsion of fish-meat in water gives a faint positive reaction; when, however, he tested the stools of military patients, whose daily ration was a liberal one of twelve ounces, including cod, plaice and hake, he invariably found the benzidine reaction to be negative. He considers the exclusion of fish an unnecessary refinement. His findings were confirmed by Ogilvie, using the benzidine slide test, which was to be expected in view of its lesser sensitivity. Ogilvie extended his investigations to fried foods, and discovered that dripping, whether beef or mutton, raw or fried, gave negative results.

Because of the absence of general agreement amongst expert opinion concerning the effect of diet on the test, I have, in my series of cases, except during the war when for obvious reasons fish could hardly be withheld, instructed the patients not to take any animal foods for three days previous to bringing the sample for testing.

VARIATION of the AMOUNTS of BLOOD REQUIRED to give a POSITIVE REACTION

The work of Kirschen (who had already worked on this problem with Boas in 1937), Sorter and Necheles (1942) has shown that the amount of blood altered in the gastro-intestinal

tract varies not only in varying pathological conditions, but also from one normal individual to another. This individual variation in the alimentary excretion of altered blood probably accounts for the varying opinion as to whether or not the taking of specimens for Gregersen testing should be preceded by the prohibition of haemoglobin-containing food. Otherwise, methodical investigation in a sufficiently large series of cases would long ago have decided the issue.

Abrahams (1920) was the first to attempt an estimation of the least quantity of human blood normally required to produce a positive reaction in the stool. He lived for a month on a diet free from all haemoglobin-containing constituents and green vegetables, during which time he swallowed measured quantities of his own blood and tested every stool. He discovered that in his own case at least 1 c.c. of blood in one dose was necessary to give a consistent positive reaction. Working in collaboration with Abrahams, Capt. R. M. McKay, by using his own elaborations of the Schlesinger and Holst's benzidine reaction, claimed that as little as 5 minims of blood, if present in the alimentary canal as a result of one haemorrhage, would be sufficient to produce a positive result. Later investigations by Bramkamp (1929) and Kieffer (1934) suggest that larger quantities may be required. In their experiments Bramkamp required 3.5 c.c. and Kieffer 5 c.c. to procure consistent positive results.

Kerchen, Sorter and Necheles fed to 146 subjects previously tested, large amounts of haemoglobin, corresponding to 9.4 c.c.

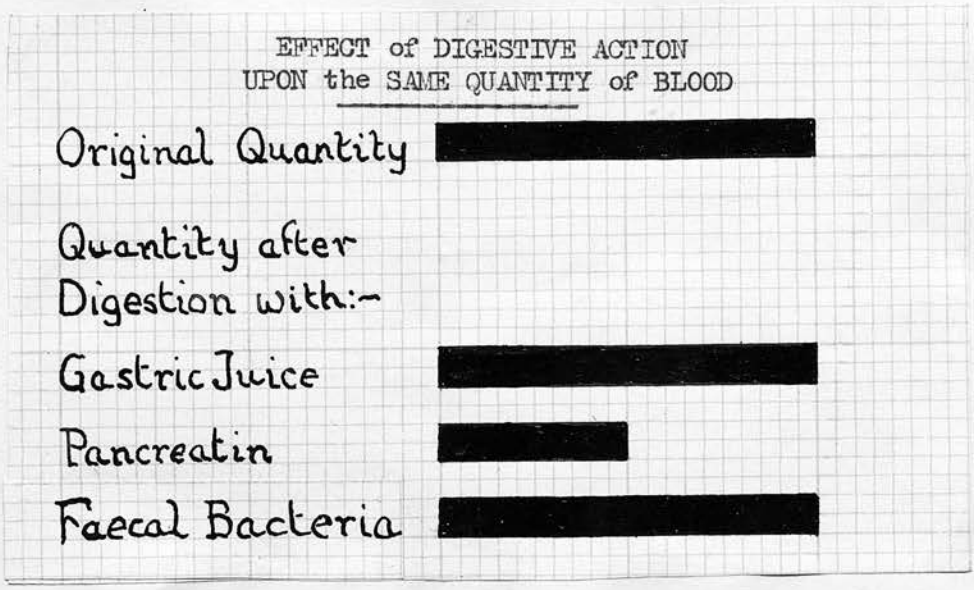
and 12.5 c.c. of blood by mouth and in small doses evenly distributed over a day. All the stools were examined during the five following days. Of the 146 subjects tested only 41 per cent. had occult blood; 59 per cent. had none. According to Kerchen, Sorter and Necheles, it can be assumed, therefore, that in certain cases blood discharged in very small amounts from lesions of the gastro-intestinal tract will undergo the same changes on its way through the stomach and bowel, and, therefore, may not be detected in the faeces by the routine benzidine method.

It is known that considerable bleeding, frequently large enough to cause severe secondary anaemia, may appear in the upper intestine or in the stomach without any visible manifestations in the faeces. This was confirmed by the experience of Necheles, who found repeated negative stool examinations immediately following a gastroscopic examination in which a freely bleeding gastric ulcer was very clearly visible. Obviously there are conditions in the bowel which can inhibit the reaction to the test for occult blood.

These observations strengthen the statement of Boas that only a positive test is of diagnostic importance, and that a negative one does not exclude the possibility of occult bleeding and, therefore, should always be related to the clinical findings.

The work of Bramkamp has given some indication of the possible conditions which influence the peroxidase reaction.

By experiment he showed that if the same amounts of blood were mixed with equal weights of faeces and water, the sensitivity of the occult blood test with the faeces mixture was reduced to a fifth of the sensitivity found with the water mixture. This seems to indicate that there may be substances in the faeces which tend to inhibit the reaction. When given in a gelatin capsule only 2 gm. of dried blood was necessary to give a positive test; whereas a negative result was obtained when the same amount was given without the capsule. By experiment he showed that the reaction of pancreatin on the blood under suitable conditions reduced its reacting powers by 50 per cent., suggesting that digestion in the intestine is important in reducing the blood to products which do not react to the benzidine test. In experimental conditions with gastric juice, simulating gastric digestion, no alteration in the sensitivity of the test could be detected. No alteration in the reaction was produced by the incubation of blood with the faeces, indicating that bacterial decomposition in the intestine was not responsible.



It has not yet been fully explained why, as Bramkamp discovered, ninety times as much blood was needed to give a positive reaction when taken by the mouth as was required when the blood was mixed with faeces. Passing through the bowel it is evident it loses much of its power to reduce peroxidase. The long alimentary tract obviously presents great opportunity for the dilution of its contents. Other possibilities were suggested by further experiments of Kirschen, Sorter and Necheles, who, by giving 10 c.c. of castor oil to subjects who had previously given a negative stool test with a given amount of ingested blood, now in several cases gave a positive reaction with the same amount of blood. It seems that the accelerated passage of haemoglobin through the bowel either diminished the possibility of a more prolonged reaction with the faeces, or diminished the possibility of haemoglobin being broken down into compounds which no longer gave a positive reaction. This assumption is supported by the findings of Ratnoff (1923) and Snapper (1927) that in small children the occult blood test is positive with much smaller amounts of blood than in adults. This change, they believe, can be related to the shorter length of and the faster passage of material through the bowel.

SPECTROSCOPIC TEST

While the spectroscopic test for occult blood is hardly within the scope of this thesis, since it is unlikely to be used in the consulting room, nevertheless it should be mentioned in view of the work of Ryffel (1928) and Boas (1935),

whose tragic death occurred in 1938. Both claim that by the spectroscopic method distinctions can be made between blood coming from malignant and benign lesions. On his spectroscopic findings, Ryffel states categorically that "provided there is some ulceration the bleeding from growths is both characteristic and persistent." He founds his contentions on the relative quantities of haematoporphyrin and haematin. Boas, on the other hand, bases his diagnosis of malignant bleeding on the separation and detection spectroscopically of what he calls "Occult haemoglobin".

Blood from below the lower colon does not show haematoporphyrin spectroscopically. The advantage of the spectroscopic test is that in association with the Gregersen Test it can be used to exclude the possibility of a positive reaction produced by blood from below that level. The spectroscopic method is much less sensitive than the chemical tests.

PREPARATION of PATIENT and TREATMENT of SPECIMEN

Although in my practice it was not feasible, it is a useful device to instruct the patient to take a dose of charcoal or carmine when starting the meat-free diet, and to wait until the charcoal or carmine has disappeared from the faeces before the specimen is collected. Kirschen, Sorter and Necheles (1942) have shown that certain preparations of carmine, when given by the mouth, induce a positive benzidine reaction. This may be related to the discovery by Macy, Reynolds and Sanders (1939) that carmine definitely affected the emptying

time of the stomach and the intestinal motility of children. Charcoal, therefore, seems to be the safer marker to use.

To avoid as far as possible the contamination of the specimen by blood from the rectum, anus and vagina, it is wise to remove the button of faeces for examination from the inside of the stool. If the stool is in the form of a scybalous mass, it can be cut with a knife and a piece removed from the interior. Where the patients have unhealthy teeth and gums, I have always instructed them to refrain from cleaning their teeth with a brush for three days previous to the test. When it is necessary to examine a stool at the earliest possible moment, a sample of faeces can almost always be immediately obtained during rectal examination with a finger stall.

It is important to test all tarry-looking stools for blood, because not only iron-containing stools simulate melaena, but according to Coope (1920) melaena is simulated by the stools of "mucous" colitis, where the mucous is intimately mixed with the faeces and coloured black with bismuth. He states also that the intense putrefaction which occurs in certain cases of colitis gives an almost black stool though no blood is present.

Kirschen, Sorter and Necheles and others have noted that on an average, blood when taken by the mouth takes three days to disappear from the stool, the test being most strongly positive the first day after ingestion, and decreasing

progressively in the next two days.

CLINICAL APPLICATION OF TEST

Much work has been done in the clinical application of the tests for occult blood. Like other tests they have their limitations, but they are sufficiently accurate to be an indispensable aid for the detection of ulcerative conditions of the alimentary canal.

Schlesinger and Holst tested the stools of 150 cases of gastric disease and found two-thirds of the cases positive; and in nearly every case further clinical evidence appeared to prove the presence of bleeding from the bowel.

Hurst states that occult blood is present in a large majority of cases of active gastric and duodenal ulcer. Barford (1928) working with Hurst at New Lodge Clinic found occult blood present in 87·6 per cent. of 30 cases of gastric ulcer and in 82·5 per cent. of 123 cases of duodenal ulcer. In only 3 per cent. of gastric ulcers and 6 per cent. of duodenal ulcers, in which signs of activity were present, was occult blood absent from the stools.

Bockus found that of ulcer cases attending his consulting room, 50 per cent. gave a positive benzidine reaction, and he suggests that in hospital practice the proportion will be greater, because hospital cases will generally be more advanced.

It seldom occurs to one in general practice to test an infant's stool for occult blood, yet H. F. Helmholtz (1909) quoted by Cammidge (1914), considers that duodenal ulcers are not uncommon among children, and that in cases of marked

anaemia, bleeding from ulcers must always be considered. He found a duodenal ulcer post mortem in eight out of sixteen cases which he examined, and in six of these the children were under three months old.

Although the findings of the Gregersen Test are not as constant in peptic ulcer as in gastro-intestinal neoplasm, because a positive reaction depends on the stage of ulcer activity at the time of the examination, nevertheless a positive result in the presence of symptoms suggestive of peptic ulcer is extremely valuable confirmatory evidence, and sometimes may reveal conditions otherwise unsuspected (e.g., cases of diverticulitis, as in my series).

It is impossible to generalise about the clinical circumstances in which occult blood may be present. Some peptic ulcers which cause great pain may show little or no occult blood (see Cases Nos.103 & 109) while others, with perhaps no pain may bleed persistently (see Cases Nos. 8 & 38). Occult blood may disappear long before the ulcer has healed radiologically. On the other hand, when radiological evidence has ceased to exist, occult blood may still be present.

I have in my records only one case of gastritis diagnosed radiologically, and this showed a "mucosa with a coarse pattern suggestive of a hypertrophic gastritis". Unfortunately, if a Gregersen Test was done on this case, the result was not noted. Apart from gastritis associated with peptic ulcer, it is a condition which apparently seldom gives rise to subjective

conditions sufficiently serious to cause an appreciable number of my patients to consult me. That inflammation of the gastric mucosa does give rise to all degrees of haemorrhage is generally accepted, and has been demonstrated by Wolff and Wolf (1943) in their patient "Tom" and by Schindler (1937) and others.

I can speak with no personal experience of occult bleeding in gastritis, but it appears to be accepted by many gastro-enterologists that in gastritis it comes and goes in spite of treatment and the improvement of symptoms. In peptic ulcer the bleeding usually stops with rest and diet, whereas in neoplasm once a positive reaction appears it most often continues indefinitely.

It is in the detection of neoplasm that the test for occult blood is not only most significant, but also, as has been said, most consistent. This was well illustrated by Meulengracht and Jansen (1929) who published their findings in 105 cases of neoplasm of the gastro-intestinal tract, in which the stools were tested by the Gregersen slide method, and except in some cases of cancer of the rectum or of the oesophagus, in which the diagnosis was otherwise shown, the diagnosis was confirmed at operation or by necropsy.

The results are seen in the following table:

Malignant Conditions Tested for Occult Blood

Diagnosis	Constant Occult Bleeding	Intermittent Occult Bleeding	Absent Occult Bleeding	Total
Cancer of the oesophagus	3	3	4	10
Cancer of the stomach	49	11	6	66
Cancer of the small intestine	3	2	-	5
Cancer of the colon	7	1	-	8
Cancer of the rectum	14	1	1	16
	76	18	11	105

It will be noticed that in 49 out of 66 cases of gastric neoplasm, persistent positive findings were made, and these findings were often of material aid in diagnosis. In 11 cases, the bleeding was intermittent and of varying intensity. In only 6 cases of gastric neoplasm was no occult blood detected. They point out that occult bleeding may not appear until the condition is long established, but in the large majority of cases it occurred sooner or later. In order of occurrence it did not take a regular place, and it was not possible to discover when occult blood is first detectable.

Occult bleeding may be the first sign localising the cancer to the gastro-intestinal tract. Meulengracht and Jansen stress the importance of repeating the test frequently because of the occasional intermittency of the bleeding. An analysis of this series of 105 cases, therefore, showed that in 89% occult blood was present. In 72% it was constantly present, and in 17% present only intermittently. In 10 out of the 11

cases which gave negative reactions, the neoplasm was situated high up in the gastro-intestinal tract. Thus, 4 out of 10 cases of oesophageal carcinoma, and 6 out of 66 cases of gastric carcinoma were negative for occult blood. These 6 cases of gastric carcinoma with negative stools were all neoplasms of the pylorus. In spite of the high percentage of positive findings, this important contribution emphasises that repeated negative tests do not exclude absolutely the presence of alimentary neoplasms, especially of the oesophagus or pyloric antrum.

After haematemesis and melaena it is valuable to examine routinely the stools for occult blood. Although in my small experience the benzidine test often remains positive for two to three weeks, Bockus states that on an average it takes seven to ten days before the benzidine test becomes consistently negative. If after that period on a haemoglobin-free diet the reaction is still positive, it is probable that oozing is still taking place, and that either the condition is malignant or the blood is coming from a walled off adherent or perforated ulcer. In these circumstances it is necessary to have an X-ray examination as soon as the patient's condition admits. In cases of malignancy certainly, and in the others possibly, operation may be required.

It is generally accepted that the most common cause of haematemesis and melaena is peptic ulceration. Natvig, Röncke, and Svaar-Seljesaeter (1943) reported 382 cases of haematemesis,

all of which were confirmed radiographically; 230 cases were of duodenal ulcer and 152 cases were gastric ulcers.

Bockus is of the opinion that peptic ulcer (gastric, duodenal and anastomatic) account for 90% of all cases of haematemesis and melaena. As has already been suggested it seems a necessary precaution in the follow-up of these cases that the stools be regularly examined for occult blood until it is clear that all bleeding has stopped.

For the sake of completeness, I include a comprehensive list of the possible conditions which may produce a positive benzidine test. It is Bockus's modification of the classification of Balfour into introgastric and extragastric causes:

CLASSIFICATION OF CAUSES

A. - Intragastric:

1. Peptic ulcer (including duodenal and postoperative ulcers)
2. Gastritis and duodenitis (erosions)
3. Gastric malignancy
4. Syphilis and tuberculosis of the stomach
5. Benign tumor of the stomach or duodenum
6. Postoperative haemorrhage
7. Rupture of sclerotic blood vessel
8. Gastric crisis of tabes dorsalis
9. Supradiaphragmatic stomach (hiatus hernia)
10. Trauma

B. - Extragastric:

1. Cirrhosis of the liver
2. Portal or mesenteric thrombosis (portal hypertension due to other causes)
3. Splenic diseases:
   Splenic anaemia, Banti's syndrome (congestive splenomegaly)
   Other types of splenomegaly

B. - Extragastric (Contd)

4. Diseases of the oesophagus:
   - Malignant or benign tumour
   - Peptic ulcer
   - Oesophagitis and erosions
   - Syphilis, tuberculosis and actinomycosis
   - Diverticula
   - Foreign body
5. Blood diseases:
   - Purpura
   - Polycythemia
   - Haemophilia
   - Pseudohaemophilia or fibrinopenia
   - Hereditary haemorrhagic angiomatosis
   - Pernicious anaemia
   - Haemolytic jaundice
   - Hodgkin's disease
   - Leukemia
6. Jaundice (prothrombin deficiency)
7. Diseases of gallbladder, appendix and pancreas (carcinoma of pancreas involving the duodenum, gallstone or malignancy eroding into stomach or duodenum)
8. Lesions of the small intestine:
   - Benign and malignant tumours
   - Carcinoma of the papilla of Vater
9. Cardiac and pulmonary disease
10. Other systemic causes (toxic, infectious and nutritional conditions and shock)
11. Aneurysm or abscess ruptured into the gastro-intestinal tract
12. Swallowed blood:
    - Epistaxis
    - Haemoptysis
    - Bleeding from mouth and pharynx
    - Malingering
13. Vicarious menstruation

In the next two Sections I give an account of 183 cases in my own practice, and an analysis showing the value and the limitations of the Gregersen Benzidine Slide Test. No attempt has been made, except where there was some particular indication, to describe the clinical history of the cases; they are set down only with a view to showing whether or not the Gregersen Slide Test was later corroborated either by X-ray, operation or subsequent history.

Where the Gregersen Slide Test has preceded the X-ray investigation, it is placed first; where it has been done during a later exacerbation of the ulcer condition, it is placed afterwards. There are included one or two cases which have given a positive Gregersen Slide Test, but are not associated with gastro-intestinal ulceration. There are not contained in this series several cases of gastro-intestinal neoplasm, particularly of the rectum and stomach, in which the diagnosis could be in no doubt.

As a rule the Gregersen Slide Test was done twice before being referred for X-ray investigation, but in some of the very obvious cases it was done only once.

Several cases with perforation previous to the period covered by this review are included, as they still have active symptoms and still attend for treatment.

ABBREVIATIONS USED: N.H.I. = National Health Insurance.
P.A. = Public Assistance.
F. = Forces, Discharged with P.U.

NOTE. - Where Benzidine Test does not appear in red, the Test was done either in Hospital or elsewhere.

DUODENAL ULCERS

(i) Cases with Gregersen Benzidine Slide Test and X-ray both POSITIVE

Case No.

1. Mrs W.- McC-: Age - 48: Occupation - Cleaner

(NHI) Typical symptoms of duodenal ulcer for a few months;
Domestic worry;
Gregersen Slide Test +++ several times;
Barium Series Aug. 1946 deformity of cap.

2. Mrs McK- : Age - 31:

(NHI) Duodenal syndrome;
Gregersen Slide Test ++;
Barium Series July 1945; deformity of cap, no crater

3. Mrs E.- A.- : Age - 53: Occupation - Housewife

Duodenal ulcer syndrome 7 years' history;
Gregersen Slide Test +++;
X-ray + 1938;
Symptoms still active;
Gregersen Slide Test +++ 20.2.47;
Gregersen Slide Test ++ 27.2.47;
Gregersen Slide Test + 1.3.47

4. Miss M.- B.- : Age - 55: Occupation - Housekeeper

(NHI) 6 weeks' history suggesting Duodenal Ulcer;
Gregersen Slide Test ++;
Barium Series Sept. 1945: duodenal ulcer
Symptoms still active

Case No.

5. Mrs M.- C.- : Age - 26: Occupation - Shop Assistant.

Long history of pain in back between the scapulae
Postural (?)
Treated in Orthopaedic Department;
(NHI) Symptoms of indigestion began to appear;
Began to develop symptoms of duodenal ulcer;
Gregersen Slide Test +++;
Barium Series 1941: duodenal ulcer;
Test Meal "Hyperchlohydria with climbing acid curve" suggestive of pyloric spasm;
Pain in back disappeared on ulcer regime.

6. Mrs H.- : Age - 65: Occupation - Housewife

Typical duodenal history, 5 months;
Gregersen Slide Test ++;
Barium Series Dec. 1945: "Deformity of cap with a diverticulum on the greater curvature side and ulcer crater at base".

7. Miss L.- P.- : Age - 22: Occupation - Clerkess

Duodenal syndrome since 15 years old;
(NHI) Associated psychological instability (kleptomania);
Gregersen Slide Test ++;
Barium Series Sept. 1941; ulcer crater demonstrated.

8. Mrs I.- P.- : Age - 31: Occupation - Winder in Bruce Peebles.

History of "stomach" trouble since 13 years old;
Gregersen Slide Test +++ several times;
Barium Series May 1942: duodenal ulcer demonstrated one month after birth of first child;
Repeated exacerbations since 1942;
Gregersen Slide Test repeatedly positive;
Gregersen Slide Test +++ for past 4 weeks;
Seriously considering further investigation with a view to partial gastrectomy.

Case No.

9. Mrs M.- S.- : Age - 32:

Indigestion and pain with periodicity of duodenal ulcer;
Gregersen Slide Test +++;
Barium Series June 1946; definite spasm of duodenum;
duodenitis (?); no ulcer seen;
Also marked rheumatoid arthritis

10. Mrs A.- R.- : Age - 26:

Year's history suggestive of duodenal ulcer;
(NHI) Gregersen Slide Test +++;
Barium Series Jan. 1947; ulcer crater present.

11. Mrs M.- P.- : Age - 33: Occupation - Cleaner.

Recurrent duodenal symptoms over several months;
(NHI) Gregersen Slide Test ++;
Barium Series Aug. 1940: ulcer crater demonstrated

12. Mrs F.- C.- : Age - 40;

Barium Series 1942: "Duodenal ulcer" and cholycystitis
Gregersen Slide Test +++ Jan. 1946?7
Barium Series March 1947: ulcer still active.
Exacerbation coincident with leucotomy on husband.

13. Mr J.- C.- : Age - 38: Occupation - Baker

Gregersen Slide Test ++;
Barium Series + Oct. 1937;
Operated on for queried perforation March 1946;
(NHI) Healing duodenal ulcer found;
Digestive symptoms much improved since gave up responsible duties of foreman.

14. Mr C.- C.- : Age - 30: Occupation - Radio Salesman.

Went into Forces 1935;
(F) Discharged in 1942;
Benzidine Test in army +++;
Barium Series "ulcer crater 1st part duodenum".

Case No.

15. Mr W.- A.- : Age - 45: Occupation - Miner

Typical history of duodenal ulcer since 1938 with an
(NHI) attack of haemetemesis in 1938;
Gregersen Slide Test +++ Jan. 1940;
Duodenal ulcer confirmed X-ray Jan. 1940;
Symptoms still active.

16. Mr R.- B.- : Age - 27: Occupation - Bath Attendant

Gregersen Slide Test +++;
(F) Barium Series "Active crater" 1937;
Went into Forces against advice;
(NHI) Invalided out again with duodenal ulcer 1940.

17. Mr R.- B.- : Age - 26:

Typical duodenal syndrome;
(NHI) Gregersen Slide Test +++;
Barium Series + Nov. 1939

18. Mr W.- B.- : Age - 30: Occupation - Electrical Worker

Two years' history suggestive of duodenal ulcer;
(NHI) Gregersen Slide Test +;
Barium Series + June 1942.

19. Mr J.- B.- : Age - 43: Occupation - Electric Welder

4/5 years' intermittent history suggestive of duodenal ulcer;
(NHI) Gregersen Slide Test ++;
X-ray Feb. 1946; "active crater demonstrated"

20. Mr T.- A.- A.- : Age - 40: Occupation - Chartered Accountant.

Duodenal syndrome 1943;
Gregersen Slide Test -;
Barium Series - ;
Recurrence of symptoms March 1945;
Gregersen Slide Test +;
Active crater demonstrated X-ray.

Case No.

21. Mr J.- D.- : Age - 63

(PA) Duodenal syndrome;
Faeces ++;
Barium Series March 1945; ulcer demonstrated

22. Mr J.- M.- : Age - 33: Occupation - Store Keeper

Long history of symptoms suggestive of duodenal ulcer 1934-1940;
Barium Series 1936; ulcer present
Barium Series 1937; ulcer present
Gregersen Slide Test +++ Feb. 1938;
Pain acute and boring through to the back;
Referred to hospital - query impending perforation;
Symptoms passed off with treatment;
Barium Series May 1938; ulcer crater visible;
Test Meal high acidity; total 85; free 65
Operation June 1939; small scar on anterior wall of
(NHI) duodenum immediately beyond the pylorus and on palpation there appeared to be a very small duodenal ulcer at this point. There was no stenosis;
Ulcer appeared to be so small and superficial and free from inflammatory reaction that it was doubtful whether it was the whole cause of the symptoms;
The appendix was long, tortuous, quite definitely thickened, and it seemed likely that it was giving rise to reflex dyspepsia. It was removed.
Symptoms remained unrelieved, and gastrectomy was performed on 3.9.40.
Convalescence stormy, with basal pneumonia.
Patient has been completely well since then, and has no symptoms, even with an unrestricted diet.

23. Mr R.- M.- : Age - 60: Occupation - Corporation Tram-Man

Duodenal ulcer ~~is~~ syndrome;
Gregersen Slide Test ++;
Barium Series 1935; "crater present";
June 1941 angina of effort;
(NHI) Oct. 1941 attack suggesting coronary thrombosis;
E.C.G. not confirmatory;
7th March, 1944 further attack;
E.C.G. shows no changes, although a B.S.R. of 14 mm. was present suggesting the possibility of a tiny infarct.

Case No.

24. Mr J.- McD.- : Age - 52: Occupation - Printer's Reader

(NHI) Duodenal syndrome;
Long recurrent history since was in Forces 1914-18 War;
Gregersen Slide Test ++;
Barium Series Feb. 1946; "deformity of cap; no crater"

25. Mr J.- E.- : Age - 43

(NHI) Gregersen Slide Test ++;
Barium Series 1943; duodenal ulcer crater found;
Off work many months; recommenced 29th Nov. 1943;
Gregersen Slide Test + 17.1.47;
Gregersen Slide Test ++ 21.2.47;
Gregersen Slide Test - 4.3.47

26. Mr J. - F.- : Age - 37

(NHI) Duodenal syndrome;
Year's history;
Gregersen +++
Barium Series 1940: "Irritable Cap";
Died suddenly, heart failure, 1943

27. Mr J.- F.- : Age - 50: Occupation - Barber

(NHI) Many months' history suggestive of duodenal ulcer;
Gregersen Slide Test ++;
Barium Series Dec. 1941; "Deformity of Cap"

28. Mr T. McK. - : Age - 59: Occupation - Brewery Worker

(NHI) Acute exacerbation Nov. 1943 with very acute persistent pain.
Gregersen Slide Test +++;
Sent into hospital, where was immediately operated on.
At operation found a duodenal ulcer "on the point of perforation, if not already perforated."

Case No.

29. Mr F.- N.- : Age - 31: Occupation - Barman

(NHI) Duodenal syndrome of some months' duration;
Gregersen Slide Test +++;
Barium Series Dec. 1945: large ulcer crater

30. Mr J.- S.- : Age - 30:

Invalided from the Forces for cholycystitis demonstrated radiologically;
Barium Series Jan. 1943; nothing abnormal revealed;
(F) Many acute exacerbations of abdominal pain since his return home;
(NHI) Gregersen Slide Test +++;
Barium Series Feb. 1947; duodenal ulcer demonstrated

31. Mr J.- K.- : Age - 45: Occupation - Plumber with Gas Department.

Soon after operation for left varicocele complained of persistent pain in left lower scapula region.
As he was highly nervous and introspective, this was
(NHI) looked upon for a while as probably neurotic. He continued to complain, and routinely a Gregersen Slide Test was done and found to be +++ on several occasions.
Barium Series June 1941; showed a duodenal crater
Pain in the back disappeared on ulcer regime

32. Mr W.- F.- : Age - 68: Occupation - Civil Servant

Duodenal syndrome
Gregersen Slide Test ++;
Barium Series Nov. 1939: "Constant irregular duodenal cap with ulcer niche on posterior wall".

33. Mr R.- W.- : Age - 39: Occupation - Brewery Worker

Long history of indigestion;
(NHI) No marked periodicity;
Gregersen Slide Test ++;
X-ray several times with negative results
Barium Series Dec. 1942 showed deformity of cap.

Case No.

34. Mr W.- H.- : Age - 34: Occupation - Motor Driver

Was discharged A.1 from Forces but soon began to have typical duodenal symptoms;
Says these had been present nine months before discharge;
(F) Gregersen Slide Test +++;
Barium Series Sept. 1946: "ulcer deformity of cap"
(NHI) Gregersen still periodically positive;
Pension claimed and granted.

35. Mr A.- W.- : Age - 25

Five years' history suggestive of duodenal ulcer;
Gregersen Slide Test ++ 2.7.46;
(NHI) Gregersen Slide Test - 20.7.46;
Barium Series July 1946: pylorospasm and deformity of duodenal cap.

36. Mr A.- R.- : Age - 50: Occupation - Plumber

Barium Series 12.12.36: "gastric ulcer lesser curvature"
Recurrence of symptoms May 1946;
(NHI) Gregersen Slide Test +;
Barium Series 18.5.46: "duodenal ulcer crater demonstrated".

37. Mr A.- H.- : Age - 50: Occupation - Fireman

Persistent duodenal syndrome over 2/3 years;
(NHI) Gregersen Slide Test +++;
Barium Series 1940; showed duodenal ulcer

38. Mrs C.- N.- : Age - 62

Duodenal ulcer with haemetemesis;
Barium Series 1939 showed irritable cap
Acute exacerbation Feb. 1947;
At time of writing (with complete rest in bed) Gregersen Slide Test has been +++ for 4 consecutive weeks. Further investigation will be required if this does not soon become negative.

Case No.

39. Mr J.- McN.- : Age - 42: Occupation - Printing Machine-man's Help

Discharged Forces with duodenal ulcer;
Barium Series in Army 1942;
Duodenal crater;
(F) Haematemesis and gastro interostomy at Edenhall;
Benzidine + in Army;
(NHI) Frequent exacerbation of symptoms;
Gregersen Slide Test +++ 22.2.47;
Gregersen Slide Test +++ 26.2.47;
Gregersen Slide Test ++ 1.3.47;
Referred to Ministry of Pensions for further treatment

40. Mr J,- F. - : Age - 44

Discharged Forces duodenal ulcer;
(F) X-ray positive Oct. 1941;
Benzidine ++ subsequent to discharge
(NHI) Gregersen Slide Test ++ several times; last time 7.3.47

41. Mr R.- O'R. - : Age - 50: Occupation - Shop Porter

Duodenal ulcer confirmed X-ray 1941;
(NHI) Gregersen Slide Test ++ during subsequent attacks
melaena 1942

42. Mr W.- P.- : Age - 32

Discharged from Forces with duodenal ulcer, 1943;
Symptoms acute and persistent after discharge;
(F) Gregersen Slide Test repeatedly positive;
Referred back to Ministry of Pensions;
(NHI) Died a day or two after partial gastrectomy, Oct. 1946

43. Mr A.- F.- : Age - 49: Occupation - Joiner

Barium Series Feb. 1936: shows duodenal ulcer
(NHI) Gregersen Slide Test +++ Feb. 1946;
Still frequent exacerbation of symptoms

Case No.

44. Mr W.- M.- R.- : Age - 24: Occupation - Miner

2 years' history suggesting duodenal ulcer;
(NHI) Barium Series + Feb. 1946;
Gregersen Slide Test + 17.2.47

45. Mr J.- S.- : Age - 40: Occupation - Corporation Labourer

Discharged from Forces because of duodenal ulcer;
No symptoms previous to enlistment;
After symptoms of 12 months' duration Barium Series 1942 showed a duodenal ulcer;
(F) Barium Series 1944 - ulcer still present;
Gregersen Slide Test - 1944 in spite of exacerbation
(NHI) symptoms;
Gregersen Slide Test +++ 27.1.47
Gregersen Slide Test +++ 6.2.47
Gregersen Slide Test - 17.2.47

46. Mr J.- W.- : Age - 40: Occupation - Grocer's Assistant

Discharged from Forces 1942 because of duodenal ulcer
(F) X-rayed 1942: ulcer demonstrated
Subsequent haemetemesis 1946;
(NHI) Haemetemesis 25.1.47;
Gregersen Slide Test continued positive till 12.2.47

47. Mrs M.- McT.- : Age - 48

Duodenal ulcer syndrome and mital stenosis;
Admitted to hospital 1939 with haemetemesis;
X-ray showed active crater;
Test meal showed marked hyperchlorhydria;
Barium Series 1940: active duodenal ulcer;
(NHI) Exacerbation of symptoms 1946;
Gregersen Slide Test +++;
Re-admitted to hospital and died soon after of heart failure.

(ii) Cases with Gregersen Benzidine Slide Test POSITIVE and X-ray NEGATIVE

Case No.

48. Mrs I.- McN.- : Age - 54: Occupation - Cloakroom Attendant

Typical history of angina of effort relieved by trinitrini; 200/100 Feb. 1946;
Began to complain of epigastric pain which she said had been present more markedly for the past three
(NHI) years; it was relieved by food and alkalies.
Appetite good; occasional regurgitation and heartburn;
Gregersen Slide Test +++ thrice;
Barium Series March 1946 entirely negative.

49. Mrs C.- D.- : Age - 53

Duodenal syndrome
Gregersen Slide Test +++
X-ray negative 1940.

50. Miss J.- F.- : Age - 21

Duodenal syndrome
(NHI) Gregersen Slide Test +++ on several occasions;
Barium Series Oct. 1941; "negative"
Clinical symptoms positive

51. Miss A.- B.- : Age - 44: Occupation - Housewife

Recurring symptoms suggesting duodenal ulcer since 1937;
Gregersen Slide Test +++ on repeated occasions;
(NHI) X-ray findings Oct. 1946, negative;
Symptoms controlled by strict ulcer regime, although still recurrence of symptoms accompanied by positive Gregersen Slide Test.

Case No.

52. Mrs P.- : Age - 62: Occupation - Housewife

History of indigestion of over a year;
Pains very acute at night;
Used to get relief from magnesia tablets;
Epigastric tenderness on pressure with some guarding;
Gregersen Slide Test ++ on several occasions;
Barium Series 1944; negative;
Symptoms disappeared with ulcer diet;
Still remains on ordinary ulcer regime.

53. Mrs M.- McC.- : Age - 33: Occupation - Housewife

Haemetemesis with long history of indigestion suggestive of duodenal irritation;
No doctor previously consulted;
Gregersen Slide Test ++ 6 weeks after haemetemesis;
Barium Series Oct. 1946: no abnormality discovered;
Almost certainly an acute peptic ulcer;
Comfortable on ulcer regime.

54. Mrs C.- P.- : Age - 40: Occupation - School Cleaner

Duodenal syndrome;
(NHI) Typical history since 16 years old;
Gregersen Slide Test +++ on several occasions;
Barium Series negative April 1944.

55. Mr G.- B.- : Age - 64: Occupation - Laundry Van Driver

Duodenal syndrome;
(NHI) Gregersen Slide Test +;
Barium Series March 1945; negative;
Died coronary thrombosis Jan. 1947.

56. Mr A.- L.- : Age - 65: Occupation - Hairdresser

Haemetemesis with no previous history except flatulence all his life;
Gregersen Slide Test positive for 14 days;
Barium Series and Test Meal revealed no abnormality;
Probably peptic ulcer.

Case No.

57. Mr J.- B.- : Age - 59

Attack of melaena
Barium Series July 1940; negative
Test meal showed "climbing acid curve"
Barium Enema negative
(NHI) Keeps well on restricted diet;
Sent to R.I.E., March 1944, with an attack suggestive of Coronary Occlusion.
E.C.G. shows evidence of myocardial degenerative changes;
Feb. 1947 - still much flatulence and acidity, with Gregersen Slide Test ++.

58. Mr J.- M.- : Age - 34

Symptoms of dyspepsia for several months;
No definite periodicity;
Gregersen Slide Test ++ twice at weekly intervals;
(NHI) Third weekly specimen negative;
Barium Series 1946; no abnormality discovered;
Kept well on ulcer regime.

59. Mr T.- J.- : Age - 72: Occupation - Baker.

Haemetemesis and melaena; no previous symptoms;
Gregersen Slide Test positive for ten days;
Barium Series negative Feb. 1946;
In view of collapse while attending M.O.P.D., E.C.G. carried out; it showed changes indicating myocardial infarction;
Clinician's Report - probably an acute peptic ulcer;
Now no symptoms as long as maintains ulcer regime.

60. Mr T.- H.- : Age - 58: Occupation - Joiner

Typical duodenal ulcer syndrome with epigastric
(NHI) tenderness and guarding
Gregersen Slide Test +++;
Barium Series negative March 1946
Symptoms still present.

Case No.

61. Mr T.- R.- : Age - 29: Occupation - Plumber

History of 2 years of symptoms strongly suggestive of duodenal ulcer;
Gregersen Slide Test +++;
Barium Series 8.2.46 negative;
Gregersen Slide Test +++ March 1946;
(NHI) Continued to attend Dietetic Dept.
31.5.46 - Referred back to M.O.P.D. because of acute pain
Tinc. belladona prescribed

(ii) (a) Because of repeated POSITIVE Gregersen Benzidine Slide Test results, the following cases - whose X-ray Tests were originally NEGATIVE - were re-examined radiologically, with POSITIVE results.

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62. Mrs D.- F.- : Age - 37

Long history of psychoneurosis; domestic trouble;
Began to develop digestive symptoms suggestive of duodenal ulcer;
Gregersen Slide Test +++;
Barium Series Negative 1943;
(NHI) Appendix removed 1943;
Gregersen Slide Test persistently +++;
Referred back to M.O.P.D. 20.7.46;
Barium Series: "deformed duodenal cap and small ulcer crater";
Gregersen Slide Test still positive from time to time, usually concurrently with meetings with her husband, who is now divorced.

63. Mr R.- C.- : Age - 44: Occupation - Labourer

Long history of indigestion - suggestive of duodenal ulcer;
Gregersen Slide Test ++;
(NHI) Barium Series negative May 1946;
Gregersen Slide Test persistently +++;
Barium Series done again June 1946; "Persistent flake on posterior wall of duodenum".

Case No.

64. Mr J.- McC. -: Age - 53

Indigestion 9/10 years;
Sent for X-ray Aug. 1943 by my Locum;
Barium Series negative;
Persistence of symptoms;
On further examination definite epigastric hernia midway between the xiphisternum and umbilicus;
Gregersen Slide Test ++;
(NHI) Referred to hospital for further investigation, Sept. 1945;
Duodenal ulcer confirmed radiologically Oct. 1945;
Nervous and highly strung;
4.11.46 marked attack of melaena following a stormy quarrel at home with his step-daughter.

(iii) Cases with Gregersen Benzidine Slide Test NEGATIVE and X-ray POSITIVE

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65. Mr A.- B.- :

(F) Duodenal Ulcer;
X-rayed Navy 1939: "ulcer first part of duodenum"
(NHI) Benzidine test in Navy negative

66. Mr J.- C.- : Age - 52

Year's history of indigestion;
Duodenal syndrome;
(NHI) Lost 1 stone in 1 year;
Gregersen Slide Test - 20.1.47
Gregersen Slide Test - 25.1.47
Barium Series 7.2.47: "no ulcer but well marked deformity of duodenal cap and good deal of pylorospasm"

67. Mr J.- McL.- : Age - 60: Occupation - Car Conductor

Duodenal ulcer;
Gregersen Slide Test - April 1941;
Barium Series 1941; commencing pyloric obstruction
(NHI) at deformity of cap.
28.2.47: symptoms still active, mostly nausea and occasional vomiting;
Gregersen Slide Test -

Case No.

68. Mr J.- D.- L.- : Age - 56: Occupation - Painter

Ulcer syndrome;
(NHI) History of 10 years during which there was much domestic worry;
Gregersen Slide Test -;
Barium Series Dec. 1944; ulcer deformity of cap.

69. Mr C.- J.- : Age - 52: Occupation - Motor Driver

Duodenal syndrome with history of few months;
(NHI) Gregersen Slide Test repeatedly - at weekly intervals;
Barium Series Nov. 1945; "deformity of cap; no crater"

70. Mr P.- K.- : Age - 38: Occupation - Store Keeper

Invalided out of Forces;
Barium Series in Army Feb. 1943: "deformity of cap;
(F) small crater in duodenum posteriorly"
Recurrent attacks of acute pain since discharge;
(NHI) Been twice called out in the night to see him, to find him suffering from acute abdominal pain;
Gregersen Slide Test repeatedly -

71. Mr A.- R.- : Age - 34: Occupation - Slater

Discharged from Forces;
(F) X-ray in Algiers 1943; findings positive;
X-ray in England 1946; findings negative;
(NHI) Symptoms active Feb. 1947;
Gregersen Slide Test -.

72. Mr J.- G.- : Age - 39: Occupation - Checker in Brewery

X-ray in Army 1943; duodenal ulcer found;
(F) Benzidine negative;
Gastric acidity high before going into Army;
(NHI) Symptoms still active;
Gregersen Slide Test - 23.1.47.

Case No.

73. Mrs M.- F.- : Age - 67

A nervous old woman markedly psychoneurotic with a multiplicity of aches and pains, who had nephrites in 1946.
Complained a good deal of pain and discomfort in right infra-hepatic region;
Physical examination always negative;
Was anxious to have an X-ray; to ease her mind, I referred her to hospital, first routinely doing a Gregersen Slide Test which was negative.
Barium Series Feb. 1947; "tenderness over duodenal cap and distal part of cap seemed a little narrow. No crater present".
Query duodenitis.

74. Mrs P.- : Age - 35

Complained of stomach pains and flatulence. Pain not definitely related to food. Some slight tenderness and guarding over upper right epigastrium.
Gregersen Slide Test -;
Barium Series Oct. 1942; irregular duodenal cap due to ulcerous scarring.

75. Mrs M.- J.- : Age - 40: Occupation - Book-binder

Duodenal Ulcer confirmed in X-ray 1932;
(NHI) Operation recommended but not performed;
Symptoms active 1946;
Gregersen Slide Test -.

76. Mrs McL.- : Age - 38: Occupation - Housewife

Symptoms of indigestion for several years;
Gregersen Slide Test -;
Barium Series showed duodenal ulcer; May 1943.

(NOTE. - Father operated upon for duodenal ulcer:) these
Sister has a perforated ulcer:) are
Brother has a duodenal ulcer demonstrated) not
X-ray.) my
(patients

(iv) Case with Gregersen Slide Test POSITIVE and no X-ray

Case No.

77. Mr R.- B.- : Age - 46: Occupation - Carter

Long history of indigestion;
Not very typical of duodenal ulcer;
Gregersen Slide Test +++ several times;
Referred to R.I.E., 1942;
Not X-rayed;
Symptoms recur when departs from ulcer diet;
Also mitral stenosis.

(v) Cases with X-ray or Clinical Symptoms POSITIVE and no Gregersen Slide Test

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78. Mr E.- C.- : Age - 34: Occupation - Dining Car Attendant.

Discharged Forces duodenal ulcer;
(F) X-rayed 1941;
Says found both gastric and duodenal ulcer;
(NHI) Also history of melaena.

79. Mr G.- R.- : Age - 56: Occupation - Printer's Machineman

Large haemetemesis with melaena;
Collapsed into fire; sent in to hospital with shock
(NHI) and severe burn of ear;
Barium Series negative Jan. 1945;
Probably acute peptic ulcer;
Epigastric discomfort if departs from ulcer regime.

80. Mr J.- W.- : Age - 63: Occupation - Mason

Haemetemesis and Melaena;
Haemoglobin 35% June 1938;
(NHI) Refused investigation in hospital;
Symptoms suggestive of duodenal ulcer.

Case No.

81. Mr W.- H.- T.- : Age - 40: Occupation - Brewery Worker

Discharged from Forces with duodenal ulcer 1944;
(F) No previous symptoms;
X-ray demonstrated tender cap filling poorly,
(NHI) possibly with small ulcer.

82. Mr H.- W.- :

(F) Discharged from Forces with duodenal ulcer 1943;
Operation - query nature.

83. Mr J.- T.- : Age - 33: Occupation - Painter

(F) Invalided from Forces;
Duodenal ulcer demonstrated X-ray Aug. 1941;
(NHI) No symptoms prior to enlistment.

84. Mr H.- S.- : Age - 35: Occupation - Labourer

Discharged from Forces;
(F) No symptoms when went into Forces in 1939;
Symptoms began in 1941;
(NHI) Operated on for perforated duodenal but none found;
there was seen a duodenal ulcer but no perforation.
Melaena 1945.

85. Mr W.- S.- :

(F) Discharged from Forces with duodenal ulcer 1943.

(NHI)

86. Mr G.- S.- : Age - 22

(F) Invalided from Forces;
Duodenal ulcer demonstrated radiologically Jan. 1946

(NHI)

Case No.

87. Mr H.- S.- D.- :

(F) Discharged Forces 1944;
Intermittent dyspepsia 10 years;
(NHI) Duodenal ulcer demonstrated X-ray.

88. Mr M.- H.- : Age - 34: Occupation - Builders' Labourer

(PA) Barium Series demonstrated crater;
History of 7 years.

89. Mr J.- F.- : Age - 36: Occupation - Shop Salesman

Haemetemesis;
X-ray 1931: duodenal ulcer;
(NHI) Gastro-enterostomy;
Symptoms not improved till a subsequent appendicectomy;
Symptoms had commenced at the age of 12 years

90. Mr R.- E.- : Age - 40: Occupation - Brewery Lorry Driver

Duodenal ulcer;
Several operations

91. Mr J.- E.- : Age - 32

(F) Discharged from Forces 1944 with duodenal ulcer.

(NHI)

92. Mr A.- McG.- : Age - 41

(F) Discharged from Forces with duodenal ulcer;
Barium Series positive June 1945.
(NHI)

Case No.

93. Mr H.- K.- : Age - 40: Occupation - Painter

(F) Discharged from Forces with duodenal ulcer;
Barium Series in Forces Dec. 1944; "ulcer crater
(NHI) present";
No symptoms previous to enlistment.

94. Mr A.- D.- : Age - 38: Occupation - Milk Roundsman

Discharged from Forces;
(F) Digestive symptoms 1941;
Become worse 1942;
(NHI) Saw specialist in 1946;
Barium Series; "duodenal ulcer demonstrated"

95. Mr G.- T.- : Age - 44: Occupation - Engineer.

Duodenal Ulcer;
(NHI) Barium Series August 1941; Crater demonstrated

96. Mr T.- McM.- : Age - 65: Occupation - Light Labourer

Sent into hospital by my Locum, with a history of
(PA) vomiting and epigastric pain after food;
Barium Series Sept. 1945; "active duodenal ulcer"
X-rays negative after a month on ulcer diet.

97. Mr S.- B.- : Age - 44: Occupation - General Labourer

(PA) Operated upon for peptic ulcer 1941;
Previous history 10 years.

98. Mrs H.- F.- : Age - 44

Haemetemesis;
(NHI) Barium Series 1945 shows deformity of cap

Case No.

99. Mrs E.- M.- : Age - 44: Occupation - Cleaner

Long history of indigestion;
(NHI) Barium Series positive;
Symptoms still active.

100. Miss M.- W.- : Age - 41

Haemetemesis and melaena 1938;
Barium Series 1938 shows slight deformity towards
(NHI) the apex of the caput.

101. Miss M.- McT.- : Age - 19: Occupation - Shorthand-Typist

Haemetemesis followed by melaena preceded by sickness
(NHI) but no pain for three weeks previously;
X-rayed four weeks subsequently, 1940, but no abnormality was discovered;
Probably acute peptic ulcer.

(vi) Case of Recurrence of Active Symptoms with Gregersen Benzidine Slide Test and X-ray both NEGATIVE

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102. Mr J.- McL.- : Age - 43: Occupation - Moulder

Duodenal ulcer 1930;
X-ray positive;
(NHI) Recurrence of active symptoms Sept. 1946;
Gregersen Slide Test -;
Barium Series - .

DUODENAL ULCERS - PERFORATED

(i) Cases with Gregersen Benzidine Slide Test and X-ray both POSITIVE

Case No.

103. Mr A.- W.- : Age - 52

History of abdominal pain going back 30 years;
Symptoms became more persistent about 1937;
Perforated duodenal Nov. 1940, 10 p.m.;
Readmitted to hospital May, 1941;
Stool benzidine positive during the first part of his stay in hospital;
Acute abdominal pains by day and night persisted unrelieved by any treatment till he died in May, 1944;
During the last three years of his life he was prevented from carrying on his work as a commercial traveller because of his abdominal pains;
It was extremely difficult, because of repeatedly negative X-ray findings, to discover to what extent his symptoms were physical or psychological. He was seen by a psychiatrist in Feb. 1944 who, in view of his previous history, could not exclude the possibility of an organic basis, but who found sufficient evidence in his psychological make-up to be unable to exclude a psychological origin;
He corroborated our knowledge that there was frequently an association between emotional events and the occurrence of the pain;
The possibility of partial gastrectomy was now considered, but on going into hospital the physical findings were so indeterminate that for the time being the idea was abandoned;
From time to time during the last three years of his life the Gregersen Slide Test was weakly positive, but more often negative;
Because of the acuteness of his pain, he was taken into hospital in March 1944. His pain did not react to treatment and on April 7th he had a haemetemesis, followed on May 4th by a much more severe one, of which he died, in spite of many transfusions, and before surgical aid could be given.
Post Mortem findings:- A large duodenal ulcer was found and two large vessels eroded at its base.

Case No.

104. Mr T.- W.- : Age - 46: Occupation - Brewery Worker

Perforated duodenal ulcer while at work Aug. 1943, 10.30 p.m.;
Slight indigestion for 3/4 months previously;
Recurrence of symptoms June 1946;
Gregersen Slide Test +++;
Mild haemetemesis Jan. 1947;
Gregersen Slide Test persisted positive for 3 weeks.

105. Mr A.- R.- : Age - 43: Occupation - Motor Driver

Perforated duodenal ulcer July 1937, 9 a.m., at
(NHI) the age of 33;
Also history of haemetemesis;
Gregersen Slide Test ++ Nov. 1946.

106. Mr T.- McK.- : Age - 58: Occupation - Lamplighter

Duodenal ulcer perforated April 1941, 3.30 p.m., at work - age 52;
Haemetemesis and frequent recurrence of symptoms;
Gregersen Slide Test frequently ++;
Partial gastrectomy 1946.

107. Mr T.- K.- : Age - 45: Occupation - Brewery Worker

Perforated duodenal ulcer May 1940, 4 a.m. - aged 39
Gregersen Slide Test ++;
(NHI) Barium Series Aug. 1943; crater present;
Gregersen Slide Test ++ on frequent occasions since;
Symptoms still active;
Has marked symptoms of angina of effort relieved by Trinitrini.

Case No.

108. Mr G.- C.- : Age - 37: Occupation - Builder

Perforated duodenal ulcer Dec. 1925, 11 a.m., aged 16 years;
(NHI) Acute exacerbation March 1942;
Gregersen Slide Test +++;
Gastric Ulcer shown X-ray 1942

109. Mr A.- B.- : Age - 48: Occupation - Brewery Worker

Duodenal ulcer;
Many attacks of haemetemesis;
Had suffered for a long time from tetany and renal glycosuria. Serum calcium has been as low as 7 mm. per cent.;
Admitted to hospital with perforation Jan. 1941;
Re-admitted June, 1941 with haemetemesis;
Stools remained benzidine positive for two weeks;
Continued to suffer from acute abdominal pains;
Barium Series negative, except for deformity of cap due to perforation;
Barium Enema also negative;
Abdominal pain continued and his Benzidine Test was 5 times negative in hospital, July 1942;
He continued to complain bitterly, and it seemed highly possible that his pains were functional;
Re-admitted Nov. 1942 with a haemetemesis and perforated while in the ward;
Gastrectomy performed with excellent results;
No digestive symptoms now present, but still has attacks of tetany controlled by calcium lactate and plentiful draughts of salt water.

110. Mr J.- T.- : Age - 47: Occupation - Labourer

Perforated duodenal ulcer June 1928, 10.30 p.m., aged 29 years; same day as wife had a child;
Gregersen Slide Test +++ 29.8.46;
Much worry at home; wife psychotic

Case No.

111. Mr A.- S.- : Age - 36: Occupation - Engineer

Duodenal syndrome;
Barium Series Aug. 1940; ulcer crater demonstrated;
Subsequent attack of haemetemesis;
Sent in to hospital Dec. 1946 because of symptoms suggestive of leaking ulcer (acute abdominal pain
(NHI) with boarding - and pain reflected to right shoulder);
Treated conservatively and did well;
Gregersen Slide Test ++ Jan. 1947, although symptoms absent.

112. Mr S.- H.- : Age - 34

Gregersen Slide Test ++;
(NHI) X-ray 1943 pre ulcer stage;
Perforated July 1945 in the evening.

113. Mr A.- M.- : Age - 32

Perforated August 1931 - aged 16;
Recurrence of symptoms 1941;
(NHI) Gregersen Slide Test ++;
Barium Series ++ showed a healing duodenal ulcer;
Test Meal showed surprisingly low total and free HCl.

(ii) Case with Gregersen Benzidine Slide Test NEGATIVE and X-ray POSITIVE

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114. Mr L.- A.- : Age - 52: Occupation - Wholesale Confectioner

Perforated duodenal ulcer July 1915, 6 p.m. ;
Occasional attacks sub-acute obstruction;
Gregersen Slide Test - January, 1947.

(iii) Case with X-ray or Clinical Symptoms POSITIVE and no Gregersen Slide Test

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Case No.

115. Mr J.- S.- : Age - 65

Perforated duodenal ulcer in Canada;
(NHI) Symptoms still active;
Died of cerebral haemorrhage 1945.

116. Mr C.- T.- : Age - 44

Perforated June 1937, 5 p.m.
(F) Barium Series April 1944 showed deformity of the duodenal cap;
(NHI) Symptoms still active.

117. Mr R.- T.- : Age - 60

Perforated duodenal 4th July, 1943, at work at 10 a.m. - aged 56;
Symptoms still active;
Also has angina of effort.

118. Mr P.- B.- : Age - 38: Occupation - Waiter

Perforated Duodenal ulcer April 1939;
(NHI) Symptoms 8/9 years.

119. Mr C.- McC.- : Age - 44: Occupation - Maintenance Labourer

Came out of the Navy quite fit, although
(F) symptoms while in the Service, and X-rayed several times;
(NHI) Perforated at cinema 1946, 9.30 p.m.

Case No.

120. Mr W.- N.- McC.- : Age - 75

Duodenal ulcer; operation in America;
Perforated November, 1932, 10 a.m. - aged 60.
Symptoms still active.

121. Mr R.- McK.- : Age - 23

(F) Discharged from Forces with a perforated duodenal ulcer, 1943.

122. Mr J.- D.- : Age - 51: Occupation - Brewery Labourer

(NHI) Duodenal ulcer perforated 12th March, 1925, noon - age 29;
Still on ulcer regime.

123. Mr P.- D.- : Age - 50: Occupation - Rubber Worker

(NHI) Perforated duodenal ulcer;
No symptoms till a few days previous to perforation;
Perforated Sept. 1934 at midnight, after drinking a lot of beer;
Symptoms still active.

124. Mr W.- F.- : Age - 58: Occupation - Warehouseman

(NHI) Perforated duodenal ulcer Sept. 1938, 9 p.m. - aged 42.

125. Mr T.- H.- : Age - 58: Occupation - Letter Press Printer

(NHI) Duodenal ulcer perforated Sept. 1943, noon, before lunch at work;
History of constant discomfort since 1912.

Case No.

126. Mr T.- H.- : Age - 48: Occupation - Labourer in Gas Department

Perforated duodenal Feb. 1944, 1 p.m., after lunch;
(NHI) Previous indigestion without treatment 4/5 years.

127. Mr J.- H.- : Age - 40: Occupation - Street Orderly, Cleansing Dept.

Discharged from Forces;
(F) Duodenal ulcer perforated Sept. 1945, 5 p.m., while attending R.I.E. with wife, whom he was accompanying to have a minor operation;
(NHI) No symptoms till 14 days previous to perforation.

128. Mr A.- P.- : Age - 57

Perforated duodenal ulcer Jan. 1937, 5.30 p.m.
(NHI) Mother died day previously.

129. Mr J.- W.- : Age - 49: Occupation - Postal Worker

Perforated duodenal ulcer April 1936, 4.30 p.m. - aged 39;
(NHI) Previous history of long standing.

130. Mr R.- R.- : Age - 47: Occupation - Messenger

Perforated duodenal ulcer Sept. 1940, 8 a.m., before breakfast - age 41;
(NHI) No previous history of indigestion.

Case No.

131. Mrs G.- : Age - 47

Two operations 1920;
Perforated duodenal ulcer Sept. 1926, 7 a.m. - aged 26.
Haemetemesis 1936;
Now symptom free.

132. Miss J.- McL.- : Age - 43: Occupation - Wool Worker

Perforated duodenal ulcer Jan. 1942, 4 p.m., at
(NHI) work and just before a meal - aged 38;
Some retrosternal pain after meals for a year before, but never sought medical advice.

GASTRIC ULCERS

(i) Cases with Gregersen Benzidine Slide Test and X-ray both POSITIVE

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133. Mrs M.- F.- : Age - 38

Ulcer syndrome; query duodenal ulcer;
(NHI) Gregersen Slide Test +++;
Barium Series, 4.1.45: "gastric ulcer".

134. Mrs M.- C.- : Age - 57

Gregersen Slide Test +++;
Barium Series 1944; "large Gastric Ulcer; also
(NHI) plaque of calcification on anterior part of aortic arch."

Case No.

135. Miss J.- C.- : Age - 48

(PA)
Gastric Ulcer;
Barium Series March 1943: "crater high on lesser curvature";
Benzidine negative in hospital;
Readmitted Dec. 1943;
Barium Series: "slight spasm of duodenal cap. Stomach negative";
Benzidine test weak positive.

136. Mrs I.- N.- : Age - 65: Occupation - Cleaner

(NHI)
X-ray Nov. 1935; Gastric ulcer demonstrated;
Angina of effort commenced in 1943;
Recrudescence of symptoms with pain shooting through to back, Feb. 1947;
Gregersen Slide Test ++ two successive weeks;
Still positive at time of writing.

137. Mrs G.- P.- : Age - 41: Occupation - Domestic Service

(NHI)
Duodenal syndrome for several months;
Gregersen Slide Test ++;
Gastric Ulcer demonstrated 1939.

138. Mr T.- R.- : Age - 56: Occupation - Commercial Traveller.

Symptoms suggestive of gall bladder disease;
Recurrent attacks of pyrexia with jaundice;
Long history of C_2H_5OH
Gregersen Slide Test +++;
Barium investigation 1940: Gastric ulcer, lesser curvature and cholelithiasis;
Died after operation in London at which there was revealed advanced cirrhosis of the liver.

Case No.

139. Mr S.- : Age - 63

Loss of appetite; pain aggravated by food;
Symptoms of long standing;
Gregersen Slide Test +++;
Barium Series May 1943: "rather shallow peptic ulcer at about mid point of the lesser curvature".

140. Mr R.- S.- : Age - 38: Occupation - Biscuit Factory Worker.

Invalided from Forces;
(F) Benzidine test while in the Forces + 11.5.42;
X-ray 26.5.42: showed ulcer on lesser curvature
(NHI) of stomach;
Given pension for duodenitis;
No symptoms till went into Forces.

141. Mr J.- K.- : Age - 27

3 months' history of indigestion;
periodicity not marked;
(NHI) Gregersen Slide Test +;
Barium Series March 1947: Gastric ulcer demonstrated;
History of tetany of long duration.

142. Mr J.- A.- : Age - 55: Occupation - Brewery Worker

Discharged from Forces with duodenal and gastric
(F) ulcers;
X-rayed Feb. 1944;
(NHI) Recurrence of symptoms 1945;
Gregersen Slide Test ++;
Referred to Ministry of Pensions hospital; detained 6 weeks.

Case No.

143. Mr J.- M.- : Age - 23

Discharged from Forces with prepyloric ulcer;
(F) X-rayed 1944;
Recurrence of symptoms 1945;
(NHI) Gregersen Slide Test +

(ii) Case with Gregerson Benzidine Slide Test POSITIVE and X-ray NEGATIVE

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144. Mr N.- F.- : Age - 28: Occupation - Panel Beater

Duodenal syndrome;
Gregersen Slide Test ++ several times;
(NHI) Barium Series negative 22.5.42;
Barium Series negative 4.6.42;
Test Meal: "High acid curve; blood present in test meal", suggesting gastric ulcer.

(iii) Cases with Gregersen Benzidine Slide Test NEGATIVE and X-ray POSITIVE

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145. Mr H.- S.- : Age - 45: Occupation - Conductor

"Indigestion" since 1924;
Exacerbation of symptoms Jan. 1941;
(NHI) Gregersen Slide Test -;
Barium Series: "ulcer high on lesser curvature".

146. Mr G.- A.- : Age - 44: Occupation - Baker

Gastric ulcer 11 years' history;
(NHI) X-rayed several times in 1936;
Gregersen Slide Test - 1946;
Symptoms still active from time to time.

(iv) Cases with X-ray or Clinical Symptoms POSITIVE and no Gregersen Slide Test

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Case No.

147. Mr J.- G.- : Age - 31: Occupation - Commercial Traveller

(F) Barium Series Army June, 1941: Prepyloric ulcer.
Symptoms return if departs from ulcer diet;
(NHI) No symptoms prior to joining Army.

148. Mr W.- P.- : Age - 36: Occupation - Vagrant Labourer

X-rayed Nov. 1933: Gastric ulcer;
Total acidity moderately high;
(PA) Symptoms again active;
X-rayed again Oct. 1941; large deformed duodenal cap, but no active ulceration.

149. Mr M.- : Age - 62

Haemetemesis 1942;
Previous X-ray examination in London had shown:
"shortening of the oesophagus with a thoracic stomach and some obstruction of the cardia";
Case of congenitally short oesophagus or partial thoracic stomach.

150. Mr W.- M.- :

(F) DISCHARGED from Forces with Gastric Ulcer; 1943.

(NHI)

Case No.

151. Mr A.- L.- :

Discharged from Forces with gastric ulcer;
(F) Barium Series 1942: gastric ulcer present;
Barium Series 1945: gastric ulcer still present;
(NHI) No history prior to enlisting.

152. Mrs M.- C.- : Age - 58

Operated on for Gastric Ulcer 16 years ago;
Gastric Ulcer;
Barium Series 1943; active ulcer.

153. Mr J.- C.- T.- : Age - 57: Occupation - With Corporation Parks Dept.

(NHI) Sent in to hospital with haemetemesis;
Barium Series negative 1944.

154. Mrs M.- G.- : Age - 40

Vomited 1½ pints blood Sept. 1942;
(PA) Barium Series negative;
No symptoms now;
Probably acute peptic ulcer.

GASTRIC ULCERS - PERFORATED

Cases with X-ray or Clinical Symptoms POSITIVE and no Gregersen Slide Test

Case No.

155. Miss M.- W.- : Age - 26: Occupation - Catering Manageress

(NHI) Perforated gastric ulcer 1941;
Many years' symptoms previous to perforation;
Now symptom free.

156. Miss C.- B.- : Age - 31

Gastric Ulcer;
(F) Perforated January, 1942, 10 a.m., after only 10 days in the Service (A.T.S.);
(NHI) No previous history.

NEOPLASMS

(i) Cases with Gregersen Benzidine Slide Test and X-ray both POSITIVE

157. Mr J.- A.- : Age - 61: Occupation - Railway Surfaceman

Came to see me because of difficulty in swallowing, of some 3 months' duration;
Gregersen Slide Test +;
X-ray; neoplasm of lower end of oesophagus;
Gastrostomy, Jan. 1943;
Died June 1943.

Case No.

158. Mr W.- H.- : Age - 79

Vague abdominal discomfort for some time previous to consulting me;
Occasional colicky pains down the right side;
Abdominal examination negative;
Examination p.r. negative;
Gregersen Slide Test persistently positive;
(NHI) Barium Enema: "carcinoma of the ascending colon";
Operation: tumour was found involving the hepatic flexure; it was movable, and no secondary glands were palpable; no nodules in liver.
Ileotransversotomy performed Jan. 1944;
Tumour not removed in view of age;
Died Jan. 1944 with symptoms of intestinal obstruction.

159. Mr C.- F.- : Age - 61: Occupation - Printer's Operator

3 months' history of indigestion - no periodicity;
He thought it might be relieved by alkalies;
(NHI) Epigastric tenderness and palpable mass;
Gregersen Slide Test persistently +++;
X-ray - Gastric neoplasm 1.4.46;
Inoperable.

160. Mr J.- F.- : Age - 64

Consulted me March 1943 because of abdominal pains of 4/6 weeks' duration. These now forced him to leave work. Suspicion of palpable mass in
(NHI) descending colon, which persisted in spite of passage of faeces;
Gregersen Slide Test +++ on 3 consecutive occasions;
Barium Enema showed neoplasm of descending colon;
Successfully removed May, 1943;
Still at work.

Case No.

161. Mr A.- McM.- : Age - 72

A case of myocardial degeneration;
Developed symptoms of sub acute abdominal obstruction;
Visible abdominal peristalsis;
Gregersen Slide Test persistently +++;
Probably neoplasm of lower bowel;
Died with symptoms of intestinal obstruction.

162. Mr J.- McC.- : Age - 64

Symptoms suggestive of gastric neoplasm;
Palpable mass present when he first reported;
(NHI) Gregersen Slide Test repeatedly +++;
X-ray 1939: carcinoma of stomach;
Inoperable.

163. Mrs T.- R.- : Age - 52

Began to have some persistent discomfort in swallowing.
Gregersen Slide Test +++;
Barium Swallow, 1942: neoplasm of lower end of oesophagus;
Possibility of transplural resection considered;
In order to try and discover the nature of the growth, oesophogoscopy performed; specimen removed for biopsy;
Pathologist's report - findings suggestive of spread from cardiac end of stomach, not primarily oesophogeal;
Laperotomy: inoperable; carcinoma of cardiac end of stomach;
Gastrostomy performed.

Case No.

164. Mrs M.- T.- : Age - 33

A few months' history of intermittent diorrhea and lower abdominal pain.
She said she had lost weight;
A mass was palpable per rectum, apparently in the pouch of Douglas;
Vaginal examination showed it to be independent of the uterus;
Gregersen Slide Test +++ on several occasions;
Referred to surgical waiting ward, 16.5.44;
Because of negative findings with Barium Enema was referred to the gynaecological ward;
Investigated 29.6.44 in gynaecological ward and referred back to surgical ward for further surgical investigation.
Patient was brought to operation on Oct. 19th, and found to have a carcinoma of the colon;
A palliative colostomy was performed as there were many secondaries in the pelvis.

165. Mrs M.- McA.- : Age - 82

Gastric discomfort and disinclination for food;
Small palpable mass in the epigastrium;
Gregersen Slide Test repeatedly positive;
Barium Series; "small filling defect adjacent to pylorus corresponding to palpable mass. No pyloric stenosis";
Patient not in a condition to stand operation.

166. Mrs I.- : Age - 65

Abdominal pain and discomfort for several months;
No medical advice previously sought;
Abdominal examination negative except for some fullness of the abdomen;
Gregersen Slide Test +++;
Barium Enema: neoplasm of pelvic rectal junction;
At operation condition was found to be inoperable;
Patient died soon after.

(ii) Cases with X-ray POSITIVE and no Gregersen Benzidine Slide Test.

Case No.

167. Mr J.- L.- :

Seen January, 1940 at Salvation Army Home;
Had vomited a considerable amount of blood;
Another haemetemesis occurred a few days after admission to hospital;
No free HCl in fasting juice;
(PA) Passage of Ryles Tube impossible;
Oesophagoscopy showed carcinoma of oesophagus or oesophagitis;
Barium Swallow confirmed carcinoma of oesophagus;
Gastrostomy performed March 1941;
Died May, 1941;
In this case, first symptom was haemetemesis.

168. Mrs M.- A.- S.- : Age - 69

Admitted to hospital 1943 with history of loss of weight and general cachexia;
X-rayed August 1943;
Barium Meal Enema revealed no abnormality;
(PA) Test Meal findings within normal limit;
Sternal puncture showed an aplastic marrow;
Left hospital on her own;
Readmitted 1944;
X-ray now shows gastric neoplasm at cardiac end of stomach and oesophagus;
Treatment was refused and patient died 1945.

(iii) Case with Gregersen Benzidine Slide Test POSITIVE, X-ray NEGATIVE, and subsequently POSITIVE.

Case No.

169. Mrs G.- : Age - 52: Occupation - Housewife

Since 1943 complained of intermittent dyspepsia;
Sometimes associated with upper abdominal pain; sometimes only flatulence;
Attacks never lasted long;
1943 Radiological examination of the alimentary tract and gall bladder was carried out with negative results;
In March 1946 symptoms were severe, particularly upper abdominal pain, and there was persistent vomiting and anorexia; acetone in urine;
Two samples of faeces at this time were Gregersen Slide Test ++;
A further Barium Series, March 1946, showed no evidence of ulcer or carcinoma;
There was a considerable nervous element since two relatives had recently died of carcinoma;
In consultation, in view of the negative radiological findings it was decided to treat the condition as functional, and with a view to persuading the patient to this point of view, categorical assurance was given and no further stool specimens were taken; the bleeding was put down to mucous membrane ozzing or superficial ulceration;
Although there were occasional mild symptoms, patient put on $1\frac{1}{2}$ lbs. during the summer, but in late Dec. 1946, there was an acute recurrence of the vomiting and Gregersen Slide Test was +++;
Barium Series Jan. 1947; "the appearance suggested a large crater in pyloric antrum towards the lesser curvature side. The duodenal cap showed appearances consistent with old ulceration".
There was doubt from the radiological point of view as to whether the ulcer was benign or malignant;
Partial gastrectomy was successfully performed;
Except for a few local enlarged glands no obvious secondaries in the abdomen could be seen on inspection;
Although the naked eye appearance of the ulcer was of the benign type, the pathological report showed it to be malignant and of the Linitis Plastica type.

DIVERTICULA

(i) Cases with Gregersen Benzidine Slide Test and X-ray both POSITIVE

Case No.

170. Mr P.- D.- : Age - 69: Occupation - Riveter

First seen March 1944 because of irregular abdominal pain not related to food;
Fullness in region of the right hepatic flexure;
Gave history of attacks suggesting melaena;
(NHI) Gregersen Slide Test in absence of any obvious melaena, several times +++;
Referred to R.I.E.
Barium Enema March 1944: reveals presence of diverticula in the descending and sigmoid colon;
Keeps fairly comfortable on ulcer regime.

171. Mr J.- A.- : Age - 67

Bowels regular once daily till about 3 weeks previous to consulting me, when he began to have 3 or 4 loose motions daily, and also an occasional motion at night;
Rectal examination negative;
(NHI) Gregersen Slide Test +++ for 3 successive weeks;
Gregersen Slide Test - 4th week;
Referred for X-ray investigation Feb. 1947;
Findings: "marked irregularity of the sigmoid colon with the formation of diverticula, the appearances being those of diverticulitis".

172. Mrs M.- T.- : Age - 62

Complained of constant feeling of discomfort on swallowing;
Gregersen Slide Test ++;
Barium Swallow Aug. 1943 showed a diverticulum arising from the middle third of the oesophagus.

Case No.

173. Mrs M.- H.- : Age - 76: Occupation - Housewife

History several years of indigestion with periodicity suggesting duodenal ulcer;
Gregersen Slide Test +;
Barium Series March 1946; negative to ulcer but diverticulum at junction of 1st and 2nd part of duodenum.

(ii) Case with Gregersen Benzidine Slide Test POSITIVE and X-ray NEGATIVE (and subsequently POSITIVE)

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174. Mrs A.- H.- : Age - 61

Dyspepsia 10 years and also angina of effort;
Gregersen Slide Test +++ 18.2.46;
Referred for E.C.G. and Barium Series;
E.C.G. showed evidence of coronary vessel disease;
Barium Series negative Feb. 1946;
Gregersen Slide Test +++ 4.4.46;
Gregersen Slide Test +++ 8.5.46;
Gregersen Slide Test +++ 15.6.46;
Referred back for further Barium investigation 28.6.46, which showed: "inflammatory changes" in sygmoid colon associated with diverticulitis.

(iii) Cases with X-ray POSITIVE and no Gregersen Benzidine Slide Test

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175. Mrs M.- McD.- : Age - 62: Occupation - Domestic Service.

Large haemetemesis with no previous symptoms;
Gregersen Slide Test negative after 7 days;
(NHI) Barium Series Dec. 1946: two diverticula, one in the middle third of the oesophagus; other in second part of the duodenum.

Case No.

176. Mr H.- McL.- : Age - 18

Reported on his own to R.I.E. because of occasional pain in his chest;
He is a long standing case of mitral stenosis;
(NHI) Barium Series 1943: "two diverticula on second part of inner side of loop";
Is on unrestricted diet, and has no symptoms; is doing light work.

177. Mrs M.- H.- : Age - 48

Melaena Jan. 1940;
Barium Series: "diverticulum in 2nd part of duodenum";
Melaena recurred 1946;
Still suffers from hunger pain.

MISCELLANEOUS

(i) Cases with Gregersen Benzidine Slide Test and X-ray both POSITIVE

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178. Mrs C.- K.- :

History of passing blood and mucous in the bowel for 6 months;
Occasional attacks of vomiting;
Gregersen Slide Test +++ repeatedly;
Barium Enema in hospital gave a picture of severe ulcerative colitis extending back to hepatic flexure;
In spite of transfusion patient died;
P.M. refused.

Case No.

179. Mrs W.- : Age - 42

Reddish-brown purulent-looking discharge from rectum for over 5 months when she came to consult me;
The discharge independent of bowel movement;
When I first saw her she had been constipated for 14 days;
No external piles and rectal examination negative, but bloody discharge on examining finger;
Proctoscopic examination shows no internal piles;
Vaginal examination negative;
Referred to hospital; query carcinoma of colon;
Sigmoidoscopy: "rectal mucosa somewhat granular and largely inflamed; no ulcer or tumour found as far as could be examined".
X-ray investigation indicates malrotation of caecum;
Ilium and appendix appearing on the lateral side;
Query bleeding due to torsion in this region.

180. Mrs E.- M.- : Age - 32

During her first pregnancy in 1937 had constant pain in the right iliac region which persisted with constant vomiting all through pregnancy;
Pain and vomiting persisted after pregnancy, which I conducted at her home;
Referred to gynaecological dept., when inflammatory mass not apparently associated with the uterine adnexae discovered;
Appendix removed;
Symptoms continued;
Gregersen Slide Test +++;
Referred to surgical side, where after repeated X-ray examinations, regional iliitis was diagnosed;
Resection of ilium was performed;
Pain and vomiting have now disappeared, and patient feels well, but has many stools daily.

181. Mrs M.- D.- : Age - 42

20 years' history; intermittent diorrhoea lasting 2/3 days;
Gregersen Slide Test + several times;
Barium Enema 1942: "spastic colon".

(ii) Case with Gregersen Benzidine Slide Test POSITIVE and X-ray NEGATIVE

Case No.

182. Mrs J.- McE.- : Age - 78

Been receiving liver injections for pernicious anaemia for several years;
Developed symptoms of indigestion; not typical in incidence;
(PA) Gregersen Slide Test +++ on several occasions;
Query carcinoma of stomach;
Barium Series Aug. 1946 negative.

(iii) Case with Gregersen Benzidine Slide Test POSITIVE and no X-ray

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183. Mr W.- S.- : Age - 64: Occupation - Brewery Worker

Attacks of haemetemesis since 1941;
(NHI) Probably cirrhosis of liver;
Gregersen Slide Test ++ from time to time.

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The cases described in the preceding Section enable one to form an estimate of the reliability of the Gregersen Slide Test in the diagnosis of gastric and duodenal ulcers, and of certain other pathological conditions in the digestive tract. For this purpose, the results of the Gregersen Slide Test have been compared with two other diagnostic criteria, namely:-

(i) The results of X-ray examination

(ii) Clinical findings, including in some cases confirmation after surgical intervention.

The cases can be divided into three main groups, namely:-

(1) Those in which the Gregersen Slide Test and X-ray examination both gave a positive result, confirmed by symptoms and subsequent history;

(2) Those in which the Gregersen Slide Test was positive and the X-ray negative, but clinical observations and subsequent history confirmed the positive diagnosis;

(3) Those in which the Gregersen Slide Test was negative and the X-ray positive, but the diagnosis was confirmed clinically.

Cases in group (2) are those which might have been missed if the Gregersen Slide Test had not been used, while cases in group (3) represent the failures of the Gregersen Slide Test. In the detailed statistical analysis of the results, group (1) was further subdivided into:-

(a) Cases in which the Gregersen Slide Test was performed first, and its findings were confirmed subsequently by X-ray;

(b) Cases in which the X-ray was taken first, and confirmed by the Gregersen Slide Test.

Group (2) was also subdivided as follows:-

(a) Cases with positive Gregersen Slide Test and negative X-ray;

(b) Cases with persistent positive Gregersen Slide Test, in which the X-ray in the first instance gave a negative diagnosis, and which on being X-rayed again because of persistently positive Gregersen findings, subsequently gave a positive result.

There are 183 cases in the present series, distributed as follows:-

GASTRIC ULCERS:-		
Non-Perforating	22	
Perforating	2	
Total		24
DUODENAL ULCERS:-		
Non-Perforating	102	
Perforating	30	
Total		132
NEOPLASMS		13
DIVERTICULA		8
MISCELLANEOUS		6

In 124 of these cases, both a Gregersen Slide Test and an X-ray result are available. In one instance, a recurrence of symptoms after many years in a case of duodenal ulcer, both tests were negative. The remaining 123 cases are analysed in Table 2.

It will be seen that in more than two-thirds of the cases, both the Gregersen Slide Test and the X-ray were positive. All but 22 of the 87 cases in this category were first diagnosed by means of the Gregersen Slide Test. Where the two tests gave differing results, the Gregersen Slide Test was right rather more often than the X-ray - 22 cases and 14 cases respectively. It is of interest to note that in the 19 ulcer cases with initial negative X-ray, three cases (Nos. 62, 63 and 64) on re-testing after an interval gave a positive. Also, the single cases of neoplasm (No. 169) and diverticulitis (No. 174) with initial negative X-rays subsequently had positive

TABLE 2

	G.S.T. + X-Ray +	G.S.T. + X-Ray -	G.S.T. - X-Ray +	TOTAL
GASTRIC ULCERS:				
Non-Perforating	11	1	2	14
DUODENAL ULCERS:				
Non-Perforating	47	18	11	76
Perforating	11	0	1	12
TOTAL ULCERS	69	19	14	102
NEOPLASMS	10	1	0	11
DIVERTICULA	4	1	0	5
MISCELLANEOUS	4	1	0	5
TOTAL CASES	87	22	14	123
PERCENTAGES:				
ALL ULCERS	67·7	18·6	13·7	100·0
OTHER CASES	85·7	14·3	0	100·0
TOTAL CASES	70·7	17·9	11·4	100·0

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(NOTE. - G.S.T. = Gregersen Slide Test)

X-ray findings. Considering ulcers only, the Table shows that both tests agreed in two-thirds of the cases, that the X-ray failed in slightly under 1 case out of 5, and the Gregersen Slide Test in slightly less than 1 out of every 7 cases. For the other diagnoses, the numbers are too small to give more than a strong indication. So far as they go, they show no failures for the Gregersen Slide Test in 22 cases. Out of the 3 in which the X-ray was initially negative, 2 subsequently registered a positive result.

However useful the Gregersen Slide Test may be, if the general practitioner is to derive the fullest benefit from its use, he will have to keep in mind not only its advantages, but also its limitations. For reasons already stated - the most important being that the quantity of blood giving a positive reaction in the stools is not constant, but varies from individual to individual because of factors not yet fully understood - a negative reaction cannot by itself be considered conclusive.

A positive result is always significant, and provided precautions have been taken to exclude extraneous sources of blood, an endeavour must always be made to trace the source of the bleeding. As in the case of every other aid to diagnosis, it is only in conjunction with a carefully taken history, careful physical examination, and sound clinical judgment that the real purpose of the Gregersen Benzidine Slide Test can be truly fulfilled.

STATISTICAL ANALYSIS of ULCER CASES

Although the number of cases of gastric and duodenal ulcer is too small to form a basis for firm statistical conclusions about incidence of the condition in the population, it is of interest to compare the statistics of the present series with figures already published.

Table 3 gives the yearly incidence of gastric and duodenal ulcers in my practice, together with the number perforating. It will be seen that there is an apparent increase in incidence from 1940 or 1941. This may, however, be illusory, as with the institution of extra rations for ulcer cases there would be every incentive for patients with symptoms to disclose them. But the table also shows that perforations increased about the same time. Although the numbers are small, the rise from 1940 to 1943 can hardly be a chance fluctuation, especially as they bear out the findings of Illingworth and his team in their remarkably thorough investigation published in the British Medical Journal in 1944. As far as they go, my cases support the belief that there was an increase in the incidence of new cases and of perforations during the war years.

In the whole series of cases there are 132 duodenal ulcers and 24 gastric ulcers. The ratio is 5·5 to 1, which agrees fairly closely with the generally accepted figures.

It is not possible to use the whole series to calculate incidence rates in the population, as the number of private

TABLE 3

YEARLY INCIDENCE of ULCERS

	MALE		FEMALE		BOTH SEXES	
	TOTAL	PERF.	TOTAL	PERF.	TOTAL	PERF.
Before 1937	11	7	3	1	14	8
1937	5	3	2	-	7	3
1938	3	1	2	-	5	1
1939	4	1	3	-	7	1
1940	7	4	3	-	10	4
1941	15	3	4	1	19	4
1942	10	1	6	2	16	3
1943	21	5	4	-	25	5
1944	10	1	3	-	13	1
1945	12	1	5	-	17	1
1946	14	1	4	-	18	1
1947	3	-	2	-	5	-
TOTAL	115	28	41	4	156	32

TABLE 4.

ULCERS in PANEL PATIENTS

	MALE		FEMALE
Total Cases, 1937 - 1946	85		23
Yearly Average	8·5		2·3
Average Panel	2300		1400
Incidence per 1000	3·7		1·6
Scotland 1938	3·38	Single	0·72
		Married	1·09

patients who, if they had ulcers, would consult me, cannot be accurately assessed. But fairly precise figures of population at risk are available for my National Health Insurance Patients. I have, therefore, abstracted all ulcer cases in panel patients during the ten years 1937 to 1946 inclusive, and calculated incidence rates for each sex. The details are shown in Table 4. I have compared my results with those for the insured population of Scotland in 1938, published by the Department of Health. Considering the very small numbers, the agreement is quite good. The slight excess in my practice may be due to the fact that my cases cover the war period, for which national figures are not available.

It was found by Illingworth (1944) that perforations occurred more frequently in December, less frequently in August, September and October. He noted that they occurred more frequently between 3 p.m. and 6 p.m., and were comparatively uncommon during the night and morning. I have analysed my perforation cases by month of occurrence, and by time of day. The figures by month are as follows:-

January	1	July	4
February	1	August	2
March	1	September	5
April	4	October	0
May	1	November	2
June	2	December	2

When grouped into quarters, the figures are:- 3, 7, 11, 4. They seem to be more common in the summer and autumn, and less common in winter.

The time of day at which perforations occurred was as follows:-

	MALE	FEMALE
Midnight to 6 a.m.	2	-
6 a.m. to noon	10	2
Noon to 6 p.m.	6	1
6 p.m. to Midnight	6	-

In my cases, therefore, perforation was most frequent in the late morning, and least frequent in the early hours.

Not the least useful and instructive of the consequences of the routine use of the Gregersen Slide Test in general practice is the amount and variety of clinical material it brings to one's notice, and the added interest it gives to the diagnoses and treatment of conditions which could otherwise be dull and uninteresting.

FAMILY HISTORY and PEPTIC ULCER

An analysis of my cases shows that there are six families in which peptic ulcer has been diagnosed in more than one member. The most outstanding one is where five children out of seven in one generation all suffer, or have suffered from duodenal ulcer. They are two males (Cases Nos. 116 and 95) and two females (Cases Nos. 131 and 54). One male and one female have

perforated, and one male and one female have had haemetemesis. These four are patients of my own. I have recently interviewed the fifth, and have satisfied myself that he is an established case of duodenal ulcer. The father, also my patient, who recently died of cardio vascular disease, as did the mother, had an operation for peritonitis in 1919, having been taken straight from his work to hospital, where he remained many weeks. It is possible that he too may have had a perforated peptic ulcer.

Cases Nos. 104, 46 and 33 are brothers. No. 104 has perforated and had a haemetemesis; No. 46 has had a haemetemesis, and No. 33 is also a typical case of duodenal ulcer.

Cases Nos. 47, 5 and 101 are mother and two daughters. Nos. 5 and 101 have had a haemetemesis, and both suffer from mitral stenosis.

Cases Nos. 172, 83 and 2 are mother, son and daughter; the mother has a diverticulum of the oesophagus, the son and daughter duodenal ulcers.

Cases Nos. 25 and 91 are father and son.

Case No. 76 tells me that her father was operated upon for duodenal ulcer, her sister had a perforated duodenal ulcer and her brother has duodenal ulcer demonstrated X-ray.

Hurst (1929) was the first to draw attention in this country to the fact that there is frequently a family history of the condition in patients suffering from peptic ulcer, and quotes evidence showing that patients with peptic ulcer give a

family history of indigestion much more frequently than patients suffering from other disorders. Necheles (1937) has shown strong evidence for an inherited constitutional factor in the form of a particular secretory pattern in peptic ulcer cases.

In 82 healthy male and female relatives of ulcer patients not having any gastro-intestinal complaint, he found distinctly low values for free acid after an Ewald Meal, while in the residue of the fasting stomachs in the greater number of instances a higher degree of free acid. In his paper he says nothing of controls. It would have been interesting to compare the result of a similar experiment in a group with no ulcer relatives.

McCarrison (1944) has noted the absence of peptic ulcer among the simple, untroubled dwellers in the Himalaya Mountains, and has shown that it is fifty-eight times as common in the south as it is in the north. By putting rats on diets nutritionally equivalent to that of both communities, he has demonstrated that there is some relationship between nutrition and the incidence of ulcer. Peptic Ulcer is evidently a disease of civilisation, crippling and incapacitating most under the strain of city life. It is perhaps not unreasonable to suggest that just as the environment of civilised life is conducive to the appearance of ulcer in man, so common environmental circumstances in the home may be one of the factors responsible for its more frequent appearance in certain families. Stewart (1929) tells us that 10 per cent. of the general population suffer at some time from chronic peptic ulcer, and it may well be that for more than one member of a family to

suffer from the condition does not lie outside the bounds of normal expectancy. Nevertheless, when it is recalled that women in relation to men suffer from duodenal ulcer in the ratio of about 1 to 4, it is difficult, particularly in Cases Nos. 47, 5 and 101, to exercise strict scientific objectivity and to remain unprejudiced in favour of an hereditary predisposition.

DIVERTICULITIS

Rankin and Brown (1930) quoted by M. J. Stewart (1931) estimate the probable frequency of diverticulosis in the whole population at 1 per cent., and state that of these diverticulitis probably occurred in ·17 per cent. of all cases with diverticulosis. As all my cases except one came to me because of symptoms and had a positive Gregersen, they, therefore, probably had diverticulitis. The one exception was a youth of 17 years who suffered from mitral stenosis. He is an only child, and his parents being rather anxious about a pain in his chest of which he complained, took him directly to the hospital Out-Patient Department, where in routine examination he was given a Barium Series and discovered to have two diverticula in the second part of the duodenum. I saw him recently, and he suffers from no digestive symptoms, "eats everything", and a sample of his faeces was negative to occult blood.

Except for the youth of 17, all cases showed the usual age incidence of about 50 years or over. Whereas diverticula are usually found more frequently in the descending colon, in this small series of cases the larger number was found in the

duodenum.

The case of Mrs M. T. (No. 172) was somewhat unusual, because diverticula of the middle third of the oesophagus seldom give rise to symptoms. Because of her discomfort in swallowing and the presence of occult blood she was ultimately seen by a surgeon. No other treatment than a suitable diet was required. The discomfort soon passed, and she has since had no further symptoms.

Mrs M. H. (No. 117) has the symptoms of a duodenal ulcer, and it is possible that the X-ray picture of a diverticulum at the junction of the first and second part of the duodenum is due to distortion because of a duodenal ulcer.

The most significant series of cases are those numbered 62, 63, 64, 169 and 174, in which, with a positive Gregersen, X-ray finding was at first negative. When the radiological examination was repeated, because of the persistence of occult blood, the X-ray findings were reversed.

In the case of Mrs G. (No. 169) I was in full agreement with the consultant concerning the line of treatment; indeed it was I who stressed the psychological background. Nevertheless, had I continued to carry out examinations for occult blood, the patient might well have been brought to operation sooner.

MYOCARDIAL DISEASE

It has often been observed that there is a frequent association between cardio vascular disease and disease of the gastro-intestinal tract, and in this series there are ten such cases, Nos. 23, 26, 48, 55, 57, 59, 107, 117, 134, 136. It was Virchow who first suggested that the thrombosis of small vessels might be one of the causal factors in peptic ulcer. Morlock (1939) in cases of hypertension was able to show in vessels of the gastro-intestinal tract a thickening of the arterial wall with diminished lumen and subsequent degeneration and fibrosis of the media. The late Professor Wilkie (1911) showed how precarious was the normal vascular supply of the first part of the duodenum, and Beattie and Dickson (1943) suggest that restriction of the blood supply to the first part of the duodenum by arterio sclerosis, thrombosis, pressure or kinking may well be a factor in lowering the vitality of the mucosa, where it is most exposed to the acid juice. The explanation of the frequently observed association between cardio vascular disease and peptic ulcer might possibly be a constitutional diathesis in the form of a generalised vascular disturbance affecting the nutrition of both myocardium and mucosa. On the other hand, cardio vascular disease, like peptic ulcer, occurs also so frequently among the population, that this apparently frequent association may also be within the limits of normal expectancy.

PSYCHOLOGICAL FACTOR

The last word has not yet been said in the etiology of peptic ulcer. For a long time it has been observed that

nervous disturbances play a part in the life history of patients suffering from this condition.

Hurst & Ryle (1932) in this country and Alvarez (1945) and Draper (1932) have drawn attention to this association. The hypothalamic theory, which postulates a nervous origin acting through vascular or muscular spasm and hence by interference with local nutrition in the gastro-intestinal mucosa, was first advanced by Cushing (1932). The psychologist emphasises the great emotional strain of civilised life as the primary factor in the causation of the condition. Whatever the cause may be, it is no longer considered sufficient to demonstrate the lesion by X-ray, prescribe the necessary diet and medicines to combat the acidity so generally associated with the condition. It is now recognised that faithful treatment requires also a full investigation of the personal habits, psychological make-up and environment in which the patient lives and works. Mr McC (Case No. 64) after the exercise of almost unbearable restraint in a quarrel with his step-daughter, the daughter of his wife to whom he is greatly attached, came to see me with an attack of melaena very soon after a long period of careful encouragement and treatment, during which I had managed to get him free of occult blood and back to work. His was a striking example of the frustration and repressed conflict which Wolf and Wolff (1943) in their fascinating monograph on "Tom" show to be the cause of hyperaemia and increased gastric motility.

Another example of the relationship between emotional conflict and gastric activity is Mrs D.- F.- (Case No. 62),

who for a long time attended the psychological clinic. After almost every occasion when she has been followed and accosted by her bullying husband, whom she had already divorced, she presents herself with a wry smile and a sample of her faeces, knowing as I do that we are pretty sure to discover occult blood.

Mrs C. (Case No. 12) after a complete absence of symptoms for five years, shows occult blood and a fresh ulcer crater X-ray soon after her husband had a leucotomy because of a prolonged mental illness.

Three cases of perforated duodenal (Nos. 110, 127 and 128) all in circumstances of acute emotional strain, demonstrate even more dramatically the striking influence of the psychological factor in patients with peptic ulcer.

C O N C L U S I O N

It is possible that in general practice the Gregersen Slide Test or the Benzidine Test in one of its other forms is more widely used than the literature and personal experience might lead one to suppose. The General Practitioner is often so preoccupied and over-tired by his day-to-day work that he has neither the opportunity nor the urge to contribute from his unsurpassed fund of experience the information which if methodically recorded and scientifically analysed would undoubtedly provide a rich fund of material capable of throwing much light on many aspects of medical science.

Figures are not known of the total incidence of peptic ulcer in this country. Five to ten per cent. of all autopsies are said to show evidence of active or healed ulcer, but not all ulcers come to autopsy, and every post mortem does not reveal the condition of the stomach and duodenum. We do know that in 1938, 3·38 in every thousand of the insured population of Scotland were incapacitated for work because of peptic ulcer.

Ours is a shrinking and ageing population, and if there is not an actual increase of peptic ulcer, an increasingly large number of cases is being diagnosed. With our shortage of man-power the problem is very much a national one, and presents a serious challenge to the medical profession and to the community as a whole.

Illingworth (1944) has strikingly reminded us that the mortality rate from perforation increases with age and delay in treatment. This applies equally to haemorrhage, which, according to Bulmer (1932) carries off 10 per cent. of the cases admitted to hospital for this serious complication. Both these conditions can in many cases be anticipated and prevented by the early detection of occult blood in the stools.

Peptic ulcer can lead to so much chronic ill-health that it is obvious that thorough treatment should be undertaken as soon as possible; the earlier the diagnosis, the simpler and more satisfactory is the treatment. There again lies the value of a test for occult blood. In the fight against gastro-intestinal neoplasm it is even more important, because successful intervention depends on its earliest possible detection.

It is to the general practitioner that the patient will first come, and who must be constantly vigilant in the lookout for its earliest manifestations. With the help of this simple test, we can sift and control our dyspepsias, with more assurance and a better knowledge of the underlying conditions, and we can more rapidly decide which require the more expensive and time-consuming forms of investigation. We can by resting them till all occult blood has disappeared, often guard our patients from the risks of perforation and more serious haemorrhage and thus perhaps prevent a fatal issue.

It may not be too much to suggest that the evidence found in this thesis is sufficiently convincing to show that in the investigation of alimentary disfunction, the test for occult blood is as important as the routine examination of the urine for albumin and sugar in suspected cases of nephritis and diabetes.

Often in our Journals the view is expressed that the increasing complexities of modern medicine are leaving to the general practitioner the mechanical role of signpost to the specialist. The opposite may well be the case. The sulpha drugs and penicillin have in the realm of therapeutics brought to the general practitioner the power to treat successfully many cases before which previously he stood helpless.

In his Thesis of 1907, discussing the then recently discovered tests for occult blood, Leech wrote: "Certain points are essential if this method of diagnosis is to come into general use, in other words if the tests are to be available

to practitioners. The apparatus must be inexpensive and simple. The testube is preferable to the microscope and spectroscope. The test must be rapid: it must not be too fine nor too crude". The Gregersen Slide Test fulfils perfectly each of these requirements, and by its faithful application in practice we can best pay tribute to the work of Boas, Adler, Leech, Clark, Gregersen and the many others who first conceived the idea of occult blood, and later achieved method and perspective in detecting its presence in the faeces.

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SUMMARY

1. For many years I have been using the Gregersen Benzidine Slide Test as an aid to the diagnosis of alimentary neoplasm and peptic ulcer.

2. An account of its history and evolution is given, with a review of the published opinion of the various well-known workers concerning its reliability as an aid in the diagnosis and treatment of gastro-intestinal disease.

3. The technique of the test and the preparation of the patient are described.

4. It has generally been found that the great majority of cases of active peptic ulcer and alimentary neoplasm excrete occult blood in the faeces and give a positive result with the Gregersen Slide Test.

5. 183 case histories are given. These include 132 cases of duodenal ulcer; 24 cases of gastric ulcer; 13 cases of neoplasm; 8 cases of diverticulitis and 8 other miscellaneous conditions.

6. In the majority of these cases the Gregersen Slide Test or an X-ray investigation, or both, were performed.

7. A statistical analysis has been made of these cases, with special reference to the reliability of the Gregersen Slide Test in comparison with X-ray examination and clinical signs and symptoms.

8. The Gregersen Slide Test is found to be positive in about 88 per cent. of cases, and also gives a positive result in about 18 per cent. of cases clinically confirmed, for which the X-ray findings were negative.

9. The yearly incidence of peptic ulcers, perforated and non-perforated, in the present series of cases is compared with already published findings.

10. These show that there is some indication of a rise in the incidence of perforated and non-perforated ulcers during the war period.

11. The association of certain other pathological conditions is noted, and their significance in the etiology of peptic ulcer is discussed.

12. Emphasis is laid on the ease and rapidity with which the Gregersen Slide Test can be performed in general practice.

13. The opinion is expressed that in cases with symptoms of persisting alimentary dysfunction its application is as important as are the tests for sugar and albumin in suspected cases of diabetes and nephritis.

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