

DIAGNOSIS AND TREATMENT IN HAEMORRHAGE
FROM THE UPPER ALIMENTARY TRACT,
WITH SPECIAL REFERENCE TO THE MASSIVE TYPE.

THOMAS HARRISON, M.B., Ch.B. (Edin.) 1921.
D.M.R.E. (Liv.) 1927.



I N D E X

	<u>Pages.</u>
Introduction.	1-2
Review of literature.	3 - 12
Mortality.	13 - 14
Diagnosis and Prognosis.	15 - 17
Aetiology of this series. (Table I).	18
Relative proportions of acute and chronic ulcer. (Table II).	19
Incidence of acute and chronic ulcer in relation to age and sex. (Table III).	20
Importance of length of history. (Chart 1).	20
Incidence of mild and severe haemorrhage. (Table IV).	22
Factors used in assessment of severity.	23
Pulse. (23) Blood pressure. (24)	
Blood volume. (24-25) Haemoglobin. (26 - 27)	
Recurrence.	27 - 34
Recurrence, classification of.	29
Recurrence, incidence of. (Table V).	27
Blood urea. (31-32) Clinical impression. (32-34)	
Radiology in diagnosis during bleeding.	35 - 52
Localisation in thorax.	35 - 43
Localisation in stomach, duodenum, or jejunum.	43 - 49
Detection of accompanying perforation.	49 - 52
Treatment.	
Liberal feeding - fluid and electrolyte requirements.	53 - 55
Blood transfusion.	55 - 61
Surgical treatment.	61 - 62
General management.	70 - 71
Fatalities.	64 - 49
Mortality in various age groups. (Table VI).	69

Detailed reports on cases of massive haemorrhage.	72 - 100
(Appendix 1).	
Summary and conclusions.	100 - 101
Summary of case reports.	102 - 112
(Appendix 11).	
References.	113 - 117

FROM THE UPPER GASTRO-INTESTINAL TRACT,
WITH SPECIAL REFERENCE TO THE MASSIVE TYPE.

Introduction.

"In spite of the number of papers on gastro-duodenal haemorrhage in recent years there is still room for good clinical studies

The results of emergency surgery and the demonstration of geographical differences are particularly wanted. Other subjects for study are the diagnosis on admission, prognosis, and indications for surgery. The initial diagnostic difficulties tend to get lost in tables of final diagnosis".

62B

(British Med.J. July 1950).

The consideration of haematemesis and melaena, mainly associated with peptic ulcer, must inevitably recur from time to time, since there is still a high mortality in haemorrhage from the upper gastro-intestinal tract and no universal routine in management has been established.

62

As recently as July 1950, Needham and McConachie record a mortality of 13.9% at Aberdeen Royal Infirmary. They venture to state that "this mortality probably represents that obtained in most general hospitals".

Difference of opinion in regard to procedure produced rival schools of thought in the early years of the century; Mayo Robson
20
and Byrom Bramwell were prominent as leaders in this rivalry.

47

73

Hurst and Ryle, and Gordon Gordon-Taylor, representatives of medicine and surgery respectively, again debated this question in 1937 - 1938. No agreement was reached, and during the succeeding few years opposite methods of treatment were advocated by

58

41

Meuelgracht and Finsterer.

In the course of time the problem itself began to change. For example, it has become apparent to every practitioner during the last thirty years that the acute ulcer of young women is disappearing and is being replaced by a much higher incidence of chronic ulcer in men over forty. ⁷⁵ (Tidy). ³¹ (Craig).

The ancillary sciences have progressed and their rapid advance during the last ten years has made diagnosis more exact and treatment more effective.

The confused, controversial and changing appearance of the subject prompted this study in detail, and it was decided to review the literature, collect a series of cases and seek to draw definite conclusions from whatever facts might be encountered.

This enquiry, then, is an attempt to establish criteria, firstly for diagnosis, secondly for prognosis and, in the light of these, to determine subsequent procedure in treatment. The subject matter is based on the findings in 120 cases of bleeding from the upper gastro-intestinal tract which came under the care or personal observation of the writer between 1942 and 1947.

It is concerned mainly with the diagnosis and treatment of haemorrhage, particularly of the massive type, arising from peptic ulcer; ulceration was present in 110 cases of this series. The patients were seen in general practice, in the Preston Royal Infirmary, where the writer had a radiological appointment, and in St. Joseph's Hospital, Preston, where he was visiting physician. Post mortem findings were used as the enquiry progressed in order to assess diagnosis and to correct errors.

Literature.

The literature, which is considerable, is here reviewed. It falls broadly within two groups - historical and scientific. Those writings which have a bearing on present day practice will

be considered side by side with personal findings in the main body of the thesis.

In surveying the papers of recent British and American authors, abstracts of their opinions and findings were collected under numerous headings to form a sectional key. Constant reference has been made to this key and it follows, therefore, that one author may be quoted several times in the different sections of the thesis.

There is, of necessity, some overlapping of these two groups of papers where recent history has merged imperceptibly with the present. The papers of Meulengracht, Finsterer, and Mariott and Kekwick, for example, although of comparatively recent date, have passed into history and their findings are no longer seriously questioned.

Isolated recognition that haemorrhage from the upper gastrointestinal tract was a cause of serious or fatal illness is recorded in early history.

The first known reference is to be found in the Eber's Papyrus. After describing the signs and symptoms of shock and blood loss associated with "his obstacle in his cardia" (literally the mouth of his stomach (cf. the Greek of Alexander Trallianus *Τὸ ὄστρομα τῆς γαστροῦς ἢ καρδία*), it continues - "Thou shalt prepare for him..... there comes in this case from his mouth or from his anus like pig's blood after it is fried. It is a blood nest which has not yet attached itself". (Ebell).

It has become apparent that Greek medicine derived much of its information from Egyptian medicine, for exactly the same statements about disease are found in Hippocrates' work whilst a less marked similarity is seen in the works of Galen. It was probably from Greek medicine that Hippocrates drew his familiarity

with haematemesis and melaena; both were known to him. He says that "those suffering from brain fever have convulsions and vomit brownish red material and some of these die rapidly." In the aphorisms we find "The vomiting of blood of any kind is bad".

In writing of diseases of women he says "If a woman vomit blood, this ceases with the onset of menstruation". Nothing could be found to suggest that he was aware of the ulcer syndrome.

25

Celsus recognised it, however, for he wrote in "de Medicina" in A.D. 30, "At si exulceratio stomachum ifestat; eadem fere faciendae sunt: quae in faucibus exulceratis praecepta sunt." A century later Galen does not specifically mention ulceration of the stomach.

Dissection of the human body fell into disfavour at this period and throughout mediaeval times; and it was not until the end of the fifteenth century that frequent post mortems were revived in Italy. (Guthrie).⁴² The mistaken meaning given to a Bull of Pope Boniface VIIIth, issued in 1300, forbidding the boiling and dismemberment of dead crusaders was probably a late factor in maintaining public disfavour.

Arabian Medicine records a curious case of haematemesis in "Relief after Distress" by a contemporary of the famous 10th century physician Rhazes. A man of Baghded came to consult Rhazes complaining of haematemesis. Rhazes elicited that he had drunk of the water of stagnant ponds and returned next day with two vessels filled with waterweed. He forcibly fed the patient with this weed until violet vomiting occurred. The vomited material contained a leech which had been the source of the trouble. (Browne).²²

The earliest post mortem report of gastric ulceration was made by Marcellus Donatus of Mantua in 1586.³⁷ He described a fatal case of pyloric stenosis in which he found an ulcer at the pylorus or lower orifice "Quod mali causam fuisse non dubitavimus".

More than a century elapsed after Donatus' description before Bonetus¹⁸ of Geneva described the post mortem findings in a case of

perforation and haemorrhage. The patient was the eighteen years old wife of a doctor. She had never menstruated and had suffered for many months from a quartan fever, as a consequence of which her spleen was enlarged. She was seized with great pain on the 25th of October and died on the 29th between two and three o'clock in the morning "semper mente constans".

Bonetus found a perforation in the stomach and attributed the condition to a spread of inflammation from the spleen "qui circa ventriculi tunicas impactus inflammationem perperistet".

The blood, he thought, had come from some open vessel when the tumour had ruptured into the stomach.

Twenty seven years later, a case of pyloric stenosis, terminating fatally, after four years, in gastric perforation, was described by Christopher Rawlinson.⁶⁶

John Hunter, and other writers of his period, make no reference to gastric ulceration as a case of haemorrhage. Hunter attributed the finding of gastric contents in the peritoneum to the absence of "vital principle" which prevented gastric self digestion.

Autolytic changes confused the picture, for post mortem examination was often long delayed.

⁴⁰
John Hunter's first contribution alone to the Philosophical Transactions was an essay (June 18:1773) on post mortem digestion written at the request of Sir John Pringle.

¹⁰
John Hunter's nephew Matthew Baillie described gastric and duodenal ulceration in his "Morbid Anatomy" published towards the close of the eighteenth century. He says that ulcers of the stomach frequently "have a peculiar appearance. Many of them are hardly surrounded by any inflammation, have not irregular eroded edges as ulcers have generally and are not attended by any particular diseased alteration in the neighbourhood. They

appear very much, as if, some little time before, a part had been cut out of the stomach with a knife and the edges had healed, so as to present an uniform smooth boundary round the excavation which had been made". He describes, therefore, their round punched out appearance with little surrounding inflammation and differentiates them from sigmoiditis of which he gives an account later. He refers to John Hunter's work on autolytic digestion which was mentioned above.

76

In the next century Benjamin Travers, writing in association with John Crampton, described three cases of gastric perforation and also mentioned a patient who had died from haematemesis. Post mortem examination had shown a posterior ulcer, adherent to the pancreas and opening into the splenic artery.

Eleven years later an excellent account of the pathology of gastric and duodenal ulcer was given by John Abercrombie of Edinburgh. He says that these cases may be

- (1) fatal by gradual exhaustion,
- (2) fatal by perforation,
- (3) fatal by haemorrhage.

He gives no details of the treatment he employed.

This preceded the classical work of Cruveilhier who gave his name to gastric ulcer.

36

Cruveilhier did not describe duodenal ulceration but distinguished between simple and malignant ulceration, which Abercrombie had failed to do.

Cruveilhier said that simple chronic ulcer of the stomach was confused in practice with chronic gastritis and more frequently with cancer, and that the pathological anatomy made it clear how haematemesis or black vomiting occurred. He further states that if the surface of an ulcer be examined there will be seen "une foule d'orifices vasculaires", and if a large vessel be

encountered, large and frequent haemorrhages had occurred.

He continued that it was not uncommon to see an ulcer perfectly cicatrized except just at the mouth of the vessel, and since the vessel cannot solidly heal except by obliteration, if this does not happen the haemorrhage may recur. (He might have said that he found an open mouthed vessel involved in a mass of fibrous tissue).

Cruveilhier did not recommend a starvation diet for more than twenty-four hours and on the second day he gave a milk diet four hourly. Following this, the patient was given a full diet of veal, chicken, jelly, rice, barley or boiled potatoes.

If the patient could not take this diet, a food should be found which the stomach could take and here "l'instinct des malades nous dirige souvent beaucoup mieux que tous les préceptes".

It is important, he states, in chronic maladies to relax from a severe regimen and not to prolong "la diète adoucissante"; here we see Meulengrecht foreshadowed, although if the haemorrhage recurred during the first twenty-four hours, the patient was still starved and deprived of fluid.

In 1847 William Brinton reviewed a series of patients on whom he had conducted post mortem examinations. He regarded the acute perforating ulcer associated with anaemia in women as in a class apart. He says that the source of haemorrhage may be mucous, sub-mucous, arterial and visceral (substance of liver or spleen penetrated by ulcer) and that the fourth is by far the most important as at least two-thirds of the fatal cases are due to this. He continues "as a rule the erosion of either of these vessels only takes place after the adhesion and fixation of the ulcerous portion of the stomach. Hence, as a corollary to this fact, the haemorrhage generally occurs in a comparatively old or chronic lesion." He treated haemorrhage with opium and Gallic

Acid. Iron citrate was given later. (Ashwell 1836)

Brinton thought Ashwell's conception of vicarious menstruation was mythical and wrote of the pathogenesis of ulcer. "There can be no doubt that as to the physiological circumstances which predispose to this disease.....

Old age, privation, fatigue, mental anxiety and intemperance are such frequent coincidents of its occurrence that we are fully entitled to regard them as its more or less immediate cause."

This is the first occasion on which anxiety is considered as a factor and this provides a link with the observations of Beaumont, 82
83 86
Wilson, and Wolf and Woolf. Wilson pointed out that haematemesis had often^{been} preceded by a period of mental stress.

35
In 1842, Curling, who gave his name to this syndrome, described a series of cases of duodenal ulceration following surface burns; he included two cases of Cooper who had described the condition earlier.

45
Hodgkin, in his lectures, stated that he had repeatedly, although not very frequently (sic) met duodenal ulceration and that vomiting may lead to perforation and that ulceration may occur in conjunction with softening of tuberculous deposit beneath the lining membrane. He continues that "the most remarkable instances of ulceration of the duodenum, but which have not been strictly confined to the pyloro-valvular space, have been occasioned by the softening of malignant tubercle, situated either in the liver or about the absorbent gland near the porta or pancreas".

23
Duodenal ulcer was first diagnosed during life by Bucquoy in 1887. He noted the remedial benefit of bicarbonate of soda and stressed the importance of distinguishing duodenal ulcer, primarily a disease affecting males, from gastric ulcer. He described five cases which bled and maintained their appetites,

particularly after the haemorrhage.

63

In his first text book Osler commented on the frequent association of chlorosis and gastric ulcer in young women, but up to this date he had seen only nine cases of duodenal ulcer, seven of them in males.

It would appear that duodenal ulcer did not become common until the present century.

Surgical treatment of haematemesis, or, indeed, of any abdominal condition, was not practised before Lister's day and no attempt to deal surgically with haematemesis was recorded until the last year of the nineteenth century. Scrutiny of earlier standard works shows that abdominal surgery was not even attempted.

69

Samuel Sharpe's textbook has no section on the abdomen and Sir Astley Cooper's Surgical Lectures, nearly half a century later, contains only the instructions for carrying out paracentesis for general or ovarian dropsy.

30

The advent of antiseptic surgery established laparotomy, and attempts were now made to treat haemorrhage from peptic ulcer by operation.

Describing haemorrhage from the stomach in chlorotic women between twenty and forty years of age, Hale White states that there is a liability to haemorrhage quite apart from actual ulceration. He writes that in these cases operation, or post mortem, usually reveals no bleeding points, although these may be seen by transmitted light; in this condition operative handling may lead to actual ulceration: if left alone and treated with iron and good food they recover; if operated on they die.

81

This type of haemorrhage was named gastrostaxis.

In the previous years, Mayo Robson⁶⁷ had said that sometimes on laparotomy "no ulcer could be found anywhere and bleeding

points prove to be capillary or from small undiscoverable ulcers".

20

A year later, Byrom Bramwell stated of the same class of case "I am disposed to think that gastric ulcer in the young chlorotic female is a less serious disease than in man".

This early picture of gastrostaxis, and Hale White's conclusions as to the bad results of surgery, acted as a deterrent for many years to surgical interference, and was quoted to me as recently as 1944 by an elderly surgeon, who gave it as his reason for refusing to open an abdomen. Nevertheless, Mayo Robson, in an address to the Edinburgh Medico-Chirurgical Society in February 1901, stressed that after a second hæmorrhage, or even during the course of that hæmorrhage, as soon as the patient's condition will permit, the operation should be done.

Byrom Bramwell expressed strong disapproval and the Lancets of 1901 contain examples of how literary war was waged in those days. Surgical interference was however, gaining in popularity largely as a result of Moynihan's work between 1901 and 1905. Moynihan operated on twenty seven cases for recurrent hæmorrhage and there were four deaths. He performed posterior gastro-enterostomies but the severity and frequency of the hæmorrhage are vaguely stated and most were operated on during the intervals between hæmorrhages from chronic ulcer.

This century, then, saw the beginning of the long controversy as to the best method of dealing with gastric hæmorrhage of the massive type.

The decay of stalwart individualism and the rise of the conception of team work have no doubt contributed to the modern opinion, that claims are not rival but complementary. All must play their part.

This opinion has evolved slowly and, until 1935, when Meulengracht introduced liberal feeding, no real progress was made.

As Dunlop said, "We all remember the ill and dehydrated patient whose need for fluid was apparent only to Nature and who was driven in desperation to filch water from the flower vase on his bedside table".

Almost simultaneously with Meulengracht's advance, Mariot
55
and Kekwick introduced continuous drip blood transfusion, a life saving procedure of the greatest importance.

47
Despite the opinion of Hurst and Ryle that neither blood transfusion nor liberal feeding were of benefit and might even be harmful, many physicians noted the reduction in mortality rate
84
effected by the new regime and in 1939 Witts became a keen exponent of drip transfusion and also of the early liberal feeding of Meulengracht, which he modified slightly.

After the outbreak of the second World War in 1939 blood banks were universally established in this country and blood became easily procurable. Blood transfusions were more widely employed and the dangers of incompatibility and pyrogen reactions were countered.

This was a further inducement to the physician to persist with conservative therapy and it was not obvious at first that these advances served the surgeon as well; he need not now meet the moribund, starving and exsanguinated patient of former years. Adequate feeding with fluid, and blood transfusion reduced the operative risk considerably.

Clinical experience, therefore, in cases of alimentary
17
bleeding had confirmed Blacklock's findings.

In his experiments on bleeding dogs he had found that secondary shock was prolonged by starving and dehydration and could be relieved by transfusion and feeding.

These improvements decreased the mortality of cases treated

conservatively and some physicians still believe today that surgery has no part to play in treatment.

Mortality.

The relative merits of both forms of treatment are difficult to assess from a survey of the literature because there is a lack of uniformity among the various collections of cases from which statistics have been compiled and there is, therefore a wide discrepancy in mortality figures for groups of cases which have had identical treatment.

52

As Avery Jones pointed out there are many variable factors responsible for this, e.g. age, sex and social status of the group.

It is recognized that the mortality rises after the age of forty-five with all forms of therapy and that a higher nutritional standard goes hand in hand with a higher social (or economic) status.

It is therefore to be expected that mortality will be higher in a series collected in a Municipal Hospital, which in many localities still retains the stigma of the Workhouse, and where patients are poorer and belong to the older age groups, than in a nursing home or Voluntary Hospital in a wealthy centre.

The severity of the cases also varies.

Some workers include all cases; others severe ones only.

Again, the criteria for assessment of severity vary. Many authors fail to state the relative proportions of acute and chronic ulcer - the former being associated with a lower mortality rate - others exclude those who have been transferred from medical to surgical ward or those who have died from complications.

An all important factor in evaluating operative mortality is the skill and experience of the surgeon.

Some of the mortality figures were compiled in the pre-transfusion era and before anaesthetics and surgical procedure had reached modern high standards.

41

Finsterer, for example, operated on all cases of bleeding from chronic ulcer while others interfered only in massive

The highest mortality is to be expected in those cases transferred to a surgeon after much delay and as a last resort.

The following table gives some published mortality figures and illustrates their wide variability which depends on some of the unseen factors mentioned above.

	34	
Cullinan and Price		18% (60% in cases having more than one recurrence).
	27	
Chiesman		25% (74% in cases having more than one recurrence).
	24	
Bulmer		10%
	3	
Aitken		11%
	29	
Conybeare		5% - 10%
	58	
Meulengracht		1%
	47	
Hurst and Ryle		4%
Surgical Treatment		
	41	
Finsterer		5% (when operation performed in first forty-eight hours).

He operated on all cases of haemorrhage from chronic and doubtful ulcers.

	73	
Gordon-Taylor		10%
	19	
Bohrer		18% (Some operations delayed for more than forty-eight hours).

In my own series the mortality was 9% and 50% in those cases who had more than one recurrence.

Diagnosis.

Here, as elsewhere, it is axiomatic to establish a definite diagnosis.

On admission to hospital the condition of the individual varies, depending upon the severity of the accident, the length of time which has elapsed since haemorrhage, and the degree of recovery.

In general practice, the summons is usually urgent because of haematemesis and/or melaena accompanied by the alarming onset of dizziness, weakness and sweating, pallor and collapse of varying severity.

This may have been preceded by a history of active ulcer symptoms with the sudden cessation of pain after the onset of bleeding, or there may have been an established diagnosis of ulceration before, or a history of chronic periodic ulcer distress. In such a case the tentative diagnosis of peptic ulcer is justified.

Previous x-ray examination may have established both the diagnosis and the ulcer site; this is invaluable if there has been no free period since the examination and later emergency demands surgical interference. Surgeons have been found more willing to operate if they know the lesion is localised and that no condition other than ulcer is responsible for the haemorrhage. It must be stressed, however, that a previous and accurate diagnosis must not be relied upon unless it be recent and there has been no considerable symptom-free interval preceding the haemorrhage.

On the other hand, no previous history can be obtained in some cases and it is not unusual to examine such by gastroscopy and x-rays two or three weeks after admission and discover no lesion. The source of haemorrhage in these cases is believed

to have been an acute ulcer.

At the beginning of the investigation, x-rays and gastroscopy were not employed for three weeks after the onset of haemorrhage; later on radiology was undertaken on the day after admission if the haemorrhage had been massive or immediately after recurrence, if an interval of improvement occurred, however brief.

The method employed is discussed in the section on Radiology.

As soon as possible after admission, a detailed history and examination were recorded and a tentative diagnosis was made.

Only light palpation was employed for abdominal examination on account of the danger of dislodging clot, but particular attention was paid to the presence or absence of abdominal rigidity as perforation sometimes accompanies the haemorrhage and may be overlooked in the old and feeble patient, especially if the outside practitioner has given morphia. (Case 26).

Enlargement of liver and spleen, spider angiomata and ascites or an abnormal abdominal mass were looked for in every patient.

The progress of the patient was constantly observed, hourly pulse rates taken and the state of shock and response to treatment repeatedly assessed.

The diagnosis was finally established radiologically and if a negative result was obtained this was further checked by gastroscopy soon afterwards.

At the beginning of the enquiry this was not done until twenty one days had passed since the haemorrhage and a negative radiological and gastroscopic finding was assumed to exclude chronic ulcer and gastritis and to indicate that the haemorrhage had been due to an acute ulcer which had healed in the meantime.

These assumptions are not beyond criticism and it would have been better, from the purely diagnostic point of view, to perform gastroscopy on admission, but this procedure, even in the hands

of an expert gastroscopist, is a definite trial and is not justified when no such expert is available.

52

F.A. Jones, who employed gastroscopy from the 3rd to the 10th day after admission, stated that no serious complications occurred but advised extreme care and discretion.

Gastroscopy was recommended more frequently in cases of haematemesis by Tanner who stated that x-rays often failed to reveal a crater because:-

67

(1) The crater was filled with blood clot;
 (11) An acute ulcer was too shallow to hold barium;
 (111) Lesions in the region of the incisura often escape notice.
 Conditions other than gastric or duodenal peptic ulcer are responsible for haemorrhage from the upper gastro-intestinal tract, i.e.:-

Multiple hereditary telangiectases;

Chronic nephritis with hypertension or ureemia;

Cesophageal or gastric varices in portal cirrhosis;

Carcinoma of the stomach;

Carcinoma of the pancreas;

Blood dyscrasias;

Congenitally short oesophagus with ulceration;

Duodenal diverticula;

Jejunal diverticula;

Diaphragmatic hernia with ulceration in the thoracic cloaculus.

These are all a possible source of haemorrhage and must be considered in the differential diagnosis.

The incidence of the various diseases responsible for the haemorrhage in this particular series is shown in the following table.

<u>Aetiology of haemorrhage.</u>	Total cases 120.	
	Males 90.	Females 30.
Peptic ulcer	87 (97%)	23 (77%)
Banti's disease	1	3
(including hepatic cirrhosis)		
Carcinoma	0	0
Chronic nephritis	0	1
Diaphragmatic hernia	0	2
Congenitally short	1	0
oesophagus with peptic ulcer.		
Duodenal diverticulum	1	0
Blood dyscrasias	0	0
Pyorrhoea alveolaris	0	1

Further procedure in differentiating type of ulcer at the bedside.

Having decided that the bleeding was due to a peptic ulcer it was then necessary to determine whether the lesion was acute or chronic since the treatment and prognosis are quite different in the two types.

It is now an accepted fact that healing occurs in almost all cases of acute ulcer treated medically whereas surgery may be necessary in the chronic type.

The relative proportions of acute and chronic ulcers which were encountered is shown in the table below; the percentages are very similar to those recorded by F. Avery Jones, whose table is shown along-side.

T A B L E 11.Relative proportions of acute and chronic ulcer.

	<u>A. Jones, 1947.</u>			<u>Present series.</u>		
	No:	Deaths.	Mortality.	No:	Deaths.	Mortality.
Chronic gastric ulcer	122 (21%)	24	20%	21 (17.5%)	5	25%
Chronic duodenal ulcer	241 (42%)	20	8.3%	42 (35%)	4	9.5%
Acute ulcer	217 (37%)	4	1.9%	35 (29%)	0	0
Stomal ulcer				12 (10%)	1	8.5%
	580	48	8%	110	10	9%

Before discussing the criteria used in the diagnosis of acute ulcer it is necessary to define what is meant by acute ulcer.

Hurst and Stewart defined an acute ulcer as one which had lasted only two to three weeks, was found to have penetrated no deeper than the submucosa and was therefore unlikely to involve a vessel of anatomical calibre.

The natural tendency of this type of ulcer is to heal rapidly, leaving no trace; if not too large, it was found by experiment that it epithelialized within three weeks of its onset.
48
(Ivy).

Age and sex as factors in differential diagnosis of acute and chronic ulcers.

Acute and chronic ulcers occurred in both males and females, and in most age groups. Apart, therefore, from the relative preponderance of acute ulcer in the female, which finding is in agreement with that of other workers, it appeared that age and

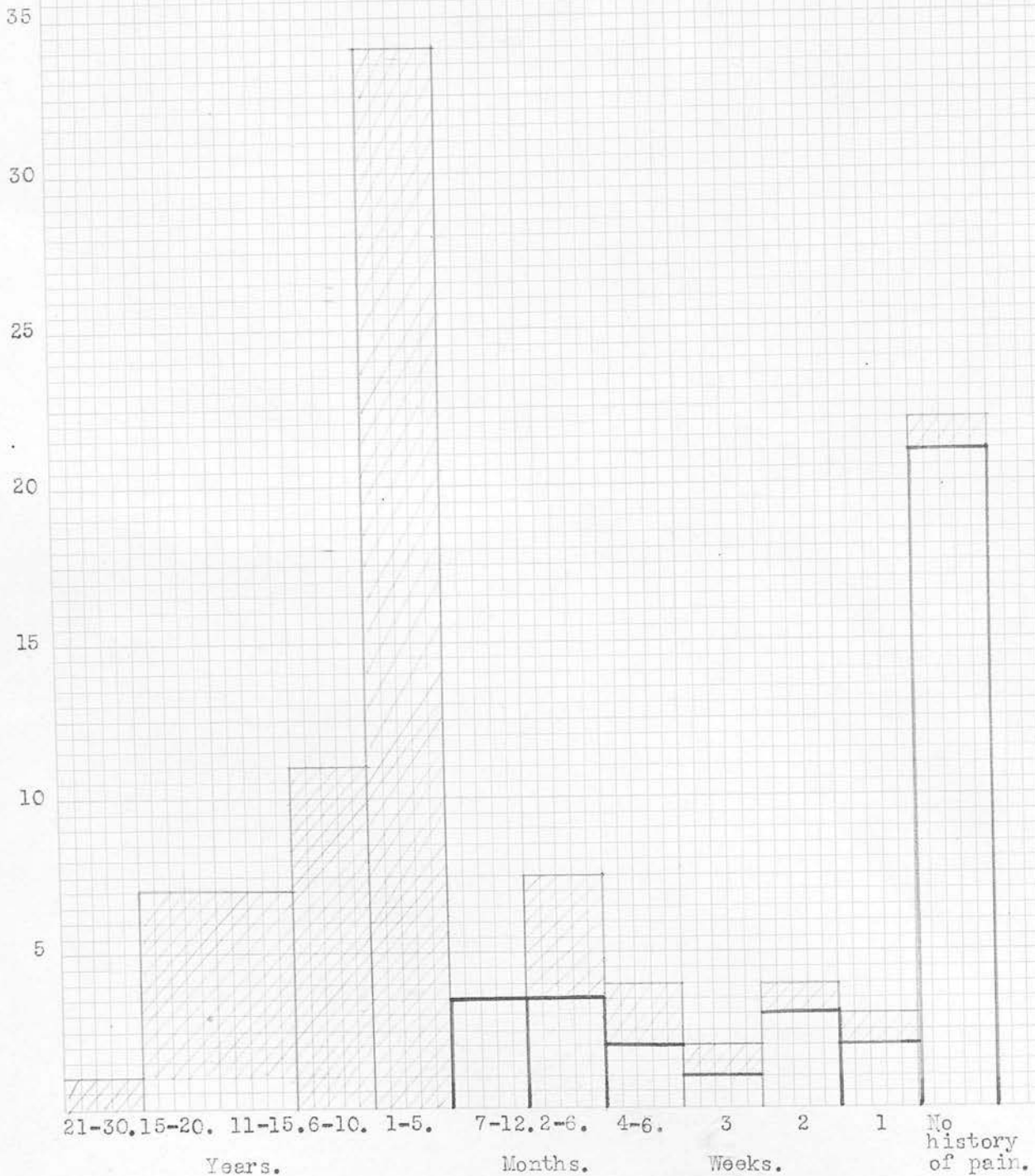
CHART 1.

Correlating the number of chronic and acute ulcers
with duration of history of pain.

Chronic ulcers. (70).

Acute ulcers. (35).

5 unclassified because of
doubt of history.



sex were of little practical value in the differential diagnosis.

T A B L E 111.

Incidence of acute and chronic ulcers in relation to age and sex.

Age	Acute.	<u>Males.</u>		Stomal	Total
		Chronic Duodenal ulcer	Chronic Gastric ulcer.		
20-29	1	2	0	1	4
30-39	3	8	6	2	19
40-49	8	11	6	3	28
50-59	10	9	3	3	25
60-69	2	4	1	1	8
70-79	1	2	0	0	3
	25	36	16	10	87
		<u>Females.</u>			
20-29	4	3	0	0	7
30-39	1	1	2	0	4
40-49	2	2	0	0	4
50-59	3	0	0	0	3
60-69	0	0	3	1	4
70-79	1	0	0	0	1
	11	6	5	1	23

At this stage it was thought probable that two other criteria might be of use in distinguishing the two types clinically at the bedside, viz;

- (1) Duration of symptoms of pain and dyspepsia;
- (11) Severity of haemorrhage.
- (1) Duration of symptoms.

As the investigation progressed it was found that the length of history of pain related to food and relieved by alkalies and/or food was a most reliable guide.

The accompanying chart (Chart 1) shows that most chronic

ulcers were associated with a history of pain for one or more years before haemorrhage, but the acute type generally caused no pain or dyspepsia. Where these symptoms had existed, however, the duration was usually less than eight weeks. In making a provisional diagnosis complete reliance was placed on the length of history of pain and/or dyspepsia, in distinguishing between acute and chronic ulcers in this series.

A short history may be obtained in chronic ulcer when the patient suffers from complicating disease, and symptoms must become obvious before they are observed against the background of chronic invalidism, to which they may be attributed. (Case 6.)

There still remains, however, a small number of cases in which an ulcer may have become "chronic" very rapidly and with no duration of symptoms; the work of A.E. Barclay throws new light on this type of case.

13

Barclay showed that in shock the mucosa becomes anaemic due to a shunt between Zones 2 & 3, in a similar manner to the renal cortex, and Alvarez pointed out that this mechanism may explain the sudden formation of penetrating peptic ulcers.

All authorities do not concur with the value of length of history.

84

Witts (1937), for example, affirmed that acute and chronic ulcers could not be distinguished by the clinical history alone, and, when haemorrhage occurs, gastroscopy is the only method of differentiation. He quotes Schindler as saying that gastritis may simulate ulcer in its signs, symptoms and history.

In some of the latter's cases of gastritis who had died from haemorrhage, the average duration of the previous history had been five years.

52

In the opinion of Avery Jones (1947), who examined over 100 patients between the third and tenth day after admission, gastritis

is a rare cause of haemorrhage and in the absence of signs and symptoms of chronic ulcer or of other sources it may be inferred that the bleeding has been due to acute ulcer.

The findings in this series support this view.

(2) Severity of haemorrhage.

84

In 1937 Witts also states that severity of haemorrhage was no help in differentiating acute from chronic ulcer and ten years later Avery Jones drew the same conclusion, as he found that bleeding might be equally severe in both.

52

The findings in the males in this enquiry were similar to those of Witts and Jones but as there were only two cases of severe bleeding in the female no comparison could be made.

T A B L E I V.

Incidence of mild and severe haemorrhage in (a) chronic and

(b) acute ulcer.

<u>Males.</u>				<u>Females.</u>			
<u>Chronic.</u>		<u>Acute.</u>		<u>Chronic.</u>		<u>Acute.</u>	
Mild.	Severe.	Mild	Severe.	Mild.	Severe.	Mild.	Severe.
41	21	18	7	11	2	10	0
(33%)		(28%)		(17%)			

It is necessary here to define a severe haemorrhage and how it can be graded as severe.

64

Papworth and Lontitt regarded as severe cases:

(1) Those with recurrent haemorrhage;

(2) All cases with secondary shock particularly if this had been allowed to persist.

This is true, but it is sometimes difficult to assess clinically or by laboratory methods when a case is suffering from secondary shock because this may be obscured by compensatory mechanisms.

The various factors which have been used in assessing the severity of haemorrhage are:

- (1) Pulse;
- (2) Blood pressure;
- (3) Blood volume;
- (4) Haemoglobin percentage;
- (5) Recurrence;
- (6) Blood urea,

and to these has been added in this enquiry,

- (7) Clinical impression.

These factors will now be considered in turn.

(1) Pulse. It is agreed by most writers that this is unreliable in assessing severity.

Witts and subsequent authors concluded that a pulse rate over 140 was an index of a severe haemorrhage and an indication for transfusion. This suggested that, other things being equal, a slow pulse was a favourable sign.

This present investigation shows clearly that this is not so and supports the findings of Wallace and Sharpey Schafer⁸⁰ when bleeding human volunteers, that many react with a vaso-vagal mechanism with slowing of the pulse.

A slow pulse in my series was unreliable; it might be a favourable sign in a small haemorrhage or unfavourable in a large haemorrhage which is associated with this vaso-vagal mechanism.

On the other hand, it was found that a rapid, particularly thready, pulse (over 120 per min.), especially if persisting or progressively increasing after half to one hour from the onset of the haemorrhage, when recovery from initial shock should have begun, proved to be a sign of a severe haemorrhage. The figure of 140 per minute chosen by Witts⁸⁴ would appear to be rather high; in this series it was found only in dying patients.

(2) Blood pressure. Blood pressure readings were found to be a more reliable index of severity than the pulse rate.

There are, however, certain pitfalls.

A compensatory vaso-constriction which may maintain the blood pressure temporarily has been described by Witts (1937) and Avery Jones (1939); this may be followed by a sudden fall - a trap for the unwary.

Circulatory collapse and pallor may appear in an otherwise uneventful case and disappear in an hour or so without transfusion. This was observed in a few of my cases and the probable explanation is that a vaso-vagal mechanism came into play after a small haemorrhage; the shock was psychological and not oligaemic. (Cases 4 - 46).

Perhaps this is the type of case which appeared to benefit by the small transfusion (10 oz) recommended by Hurst. The unknown hypertensive may be another source of error (Case 1). A systolic pressure of 160 mms.Hg. may be a low reading for a man whose "normal" reading is 240 mms.Hg., but in this case the diastolic pressure was also much more reduced - down to 50 mms.Hg. - and the pulse pressure was obviously quite abnormal.

This fact, then, accompanied by atheroma in the observable peripheral vessels (including retinal) and increase in the size of the left heart is an important factor in deciding whether the blood pressure is normal for any particular individual.

Excluding the above exceptions, it is reiterated that blood pressure readings were reliable in judging severity and if the systolic reading were 90 mms.Hg. or below, especially if the stage of primary shock ought to have passed, the haemorrhage was assessed as severe.

(3) Blood volume.

The relationship between a fall in blood pressure and

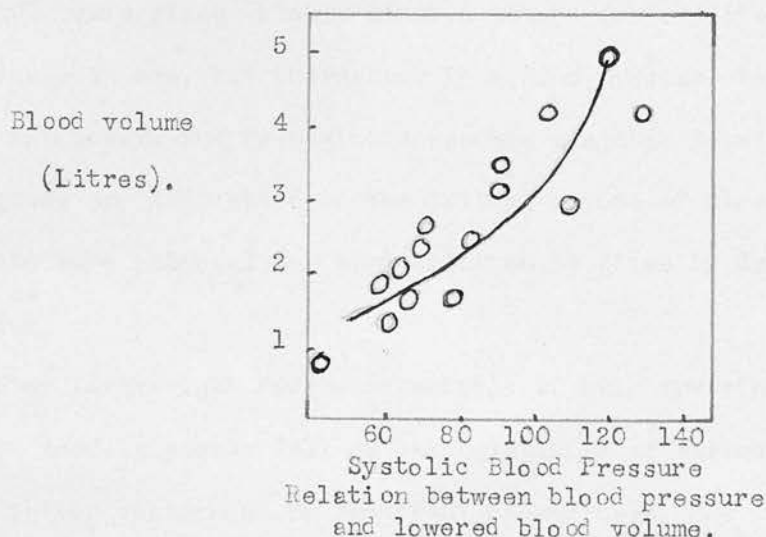
decrease in blood volume of which it is a measure, has been
 57
 investigated by McMichael. He pointed out that the blood volume
 is reduced more than was formerly supposed.

Keith's estimations of blood volume, he says, were made
 with vital red, the size of whose molecular aggregates was
 considered large enough to retain them in the circulation; the
 undue permeability of capillaries in shock however rendered his
 measurements fallacious.

Using a new spectro-photometric carbon monoxide method
 devised by D.K. Hill, it was found that the reduction in effective
 circulating blood volume may be extreme - down to 30%-40% in
 patients with a blood pressure of 60 mms.Hg. and with a pressure
 down to 90 mms.Hg. the blood volume has been reduced to 3 litres
 from the normal value of 5 litres.

The work of McMichael and D.K. Hill correlates the two
 factors of blood pressure - an easily obtained observation -
 and the vital one of blood volume.

The accompanying graph is reproduced from McMichael's paper.



Blood volume estimation is a reliable guide as it indicates
 directly the degree of oligæmia and this estimation was strongly
 advocated by Bennett, Wright & Dow, but, in practice, skilled

staff, not available in a small hospital, is required and reliable methods are not sufficiently rapid, with the possible exception of that of Crooke and Morris.

33

(4) Haemoglobin percentage. It is commonly held throughout the literature that a haemoglobin reading below 40% is an indication of a severe haemorrhage.

The initial reading, immediately after a haemorrhage, is of little value because a low reading may be due to a chronic leak, or a very large haemorrhage may be associated early with a high haemoglobin reading because haemo-dilution has not yet occurred.

Some cases in this collection (Cases 10,12,42,47,57,72) had a low initial haemoglobin reading but it was obvious from the clinical impression that the recent haemorrhage had not been severe. This was explained by a series of haemorrhages in the preceding week, none of which had been severe enough to cause alarm.

Haemodilution is not complete for 24-48 hours (Bennett et alii); according to McMichael blood dilution to the extent of 15% - 20% takes place quickly after a severe gastro-intestinal haemorrhage in man, but thereafter is a slow process, taking a day or two before the haemoglobin reaches a static level (e.g. 40%) which gives any indication of the initial volume of blood loss.

This same interval for haemodilution is given by Cullinan and Price.

34

After forty-eight hours, especially if drip transfusion has been used, a steady fall of haemoglobin is of serious import and indicates continuous or recurrent haemorrhage.

The loss in blood volume is greater than the haemoglobin reading suggests, even after haemodilution is complete, for as Bennett, Wright and Dow point out, it is the plasma volume alone which is made good by tissue fluids; the corpuscular volume is

not quickly replaced. If the total volume lost had been restored by fluid replacement, the haematocrit reading would be a true index of the blood loss, but as the new volume is reduced by the corpuscular deficiency, the haematocrit gives too high a figure and minimises the loss.

(5) Recurrence.

Throughout the literature there is unanimous agreement that recurrence is the most serious prognostic sign.

According to Cullinan and Price death from solitary haemorrhage was rare but rose from 4% to 40% with one recurrence and to 60% with more than one recurrence.

Other authors report even higher figures.

27

In Chiesman's series of 48 males who had recurrences, 38 died; 8 in a similar series of 14 females died - a total mortality of 74%.

At post mortem examination the cause of the repeated haemorrhages was found to be a partially eroded vessel of considerable calibre in the floor of the ulcer.

In a series of cases with recurrent haemorrhage investigated by Avery Jones in 1939, 13 had repeated attacks, and of these only 4 suffered from chronic ulceration.

Cullinan and Price found that slightly more than 50% of their cases had recurrences and that this proportion was unaffected by dietary regime.

The findings in this enquiry are shown below.

T A B L E V.

Incidences of recurrences.

Total cases 110.

Chronic 74. Acute 36.

Deaths.

Deaths.

No recurrence

50

0

32

0

One recurrence	9	1	1	0
More than one recurrence	15	9	3	0

It may be noted that -

(1) No case of solitary haemorrhage died.

(2) One case with one recurrence died.

(3) Of the 28 cases with recurrences, 24 were chronic and 4 acute, i.e. 33.0% of chronic ulcers and 11% of acute ulcers had recurrences.

(4) In those who had more than one recurrence 50% died.

(5) All cases with acute ulceration survived.

The problem of recurrence is not as straight-forward as it appears on paper for much depends on the frequency and severity of the blood loss. It was found that small haemorrhages occurring at infrequent intervals were of minor clinical significance and these were ignored for statistical purposes, and similarly, cases which had a history of several haemorrhages on the days immediately before admission were often mild since a severe bleeding would have allowed no delay in despatch to hospital.

On the contrary, a recurrence of haemorrhage accompanied by signs of oligæmic shock, particularly in a patient who has not recovered from a recent severe haemorrhage, is a very serious incident.

50

A recurrence was defined by Jones as a case which exhibits signs and symptoms of acute haemorrhage not less than twenty-four hours after a previous haemorrhage. He clearly insists that the blood loss must be severe but excludes those cases in which the haemorrhage was repeated within a few hours of the preceding one. Those, judging by this study, are without doubt the most

serious of any type of recurrence and especially so when they are coupled with the clinical diagnosis of chronic ulcer.

My cases of acute ulcer did not die despite the severity of the recurrent haemorrhages; this may be fortuitous since there were only seven cases of acute ulcer with severe bleeding. The writer's impression is that liberal blood transfusion supplemented by early feeding was responsible for their recovery.

The relatively small calibre of the vessel involved - an intra-mural one - and the absence of fibrosis tend to limit the blood loss and allow natural arrest of haemorrhage in acute ulceration.

Nevertheless some cases of the acute ulcer group (e.g. Cases 21 and 54) were so ill that failure to recover without transfusion seemed probable and made the higher percentage of deaths from acute ulcer haemorrhage in the past quite understandable. Three deaths in this type of case were recorded by Bohrer¹⁹ but he does not state whether blood was given: Avery Jones who records four similar deaths also fails to provide details.

Classification of severe recurrent haemorrhage.

Study of the case histories soon made it apparent that severe recurrent haemorrhage associated with a chronic ulcer was a very serious problem.

It would appear that in published papers sufficient weight has not been placed on the severity, frequency, and most important of all, the time between the recurrences of bleeding.

"Recurrence" needs further definition, for the purpose of clinical assessment, of record and of publication.

The above mentioned factors vary in each case and an arbitrary bedside classification was sought by typing severe and recurrent haemorrhage as follows.

Type (1). Patient who have two or more moderate haemorrhages

at intervals of twenty four hours or longer, each attack producing definite signs and symptoms of shock but a fairly rapid recovery follows transfusion.

The blood pressure and blood volume have been restored to reasonable levels before the next haemorrhage occurs and the patient is able to withstand it.

Type 11). Patients having less copious haemorrhages, but at more frequent intervals, e.g. 9 - 12 hours.

The degree of shock does not give rise to immediate anxiety for the patient's life and the haemoglobin is maintained at a safe level by transfusion.

Type 111). Patients who have frequent attacks e.g. 2 - 4 hours, of moderate bleeding or less frequent attacks of severe or catastrophic haemorrhage with marked signs of shock and collapse. In spite of liberal transfusion the patient does not rally completely and there is a progressive deterioration in condition.

It is obvious that this classification is somewhat artificial and that there must be some degree of overlapping of one type on another, nevertheless it has proved to be a simple means of depicting the intimate details of the clinical picture and progress of a case and a guide to both prognosis and treatment. Any case in Type 111 was recommended for surgical intervention.

An attempt should be made to place the case in its correct group, if possible within the first forty eight hours but some of Type 1 or Type 11 may pass into Type 111 and have then to be treated accordingly. Unfortunately this transition may be ushered in by a catastrophic haemorrhage, fatal even before a transfusion can be prepared. This sudden fatal bleeding rarely causes unexpected loss of life - only one case in this

series - and it is not a reason for recommending universal surgery in severe haemorrhage. The outcome in the majority of cases which require operation and which die if surgery is unsuccessful, is predictable.

(6) Blood urea.

Various observers have noted that the blood urea is raised in cases of haematemesis and melaena and that the rise is proportional to the severity of the haemorrhage.

Different causes for the azotaemia have been suggested, among them being the presence of blood in the bowel as a source of nitrogen, the break down of tissue proteins as a result of dehydration, functional renal impairment due to low blood pressure, salt and water deficiency, etc.

Further light has been thrown on this problem more recently by the work of Trueta, Barclay et alii ⁷⁷ showing the anoxia resulting from the cortical shunt mechanism which operates in shock. In a most comprehensive paper Black states that in patients with severe gastro-intestinal bleeding, the blood urea is constantly raised. As a rule the increase is moderate in degree - to about seventy milligrammes per cent - but much higher readings have been recorded.

The rise may begin within two hours of the bleeding and is highest on the next day.

He suggests that from the available evidence the usual moderate rise in blood urea is due to absorption of nitrogen in large quantities from the bowel at a time when kidney function is decreased by diminished blood flow (he had not the shunt mechanism in mind as a cause for this).

Break down of tissue becomes important as a source of nitrogen if the haemorrhage be severe; the blood urea is then raised still further.

He finds that the height to which the blood urea is raised is of definite value in assessing severity of haemorrhage and is a good guide to progress.

The height of the blood urea was also considered by ⁸⁴Witts to be a useful gauge, 100 mgms per cent was regarded by him as an indication of a severe haemorrhage.

In this investigation blood urea estimations were discontinued early; it was not found reliable, as some of the early fatal cases had a blood urea reading very little above normal until a day or two before death. This was probably due to the fact that these cases were treated with liberal blood transfusion and adequate salt and water intake; a high blood urea was therefore terminal.

16

Black, as a result of his investigations recommended liberal transfusions with adequate salt and water intake; he did not however specify the amounts of blood given to his subjects. It is therefore impossible to judge whether these were adequate or not.

The decision as to whether or not a haemorrhage is severe must be made immediately and, as there is a time lag of twenty four hours before an abnormal figure may be obtained, it was concluded that although blood urea figures indicate in retrospect the severity of a haemorrhage, they were of more academic than practical value.

The prognostic significance of blood urea estimates did not favourably impress Crohn and Lerner.

32

(7) Clinical impression.

Some of the accepted criteria of severity may be within normal limits, and yet when these are all considered as the clinical impression, which includes also the history, the

general appearance of the patient - pallor; dry, furred tongue; thin, rapid thready pulse; subnormal temperature; restlessness and prostration - a severe haemorrhage is rarely unrecognized.

The reasons for the unreliability of a hard and fast interpretation of criteria, without a leavening of common sense are not difficult to find.

The readings obtained represent the result of the direct and reflex effects of haemorrhage; they are not the immediate result of diminution of blood volume.

The reflex effects of haemorrhage are believed to be compensatory - an effort to ensure an adequate blood supply to the vital nervous centres - and the operation of more than one reflex mechanism may further complicate the picture.

After a large haemorrhage the blood pressure usually falls and the pulse rate rises (Marey's reflex) but in some cases a vaso-vagal response is produced and the pulse is slowed (Wallace and Sharpey-Schafer).
80

It is true however that the findings of Wallace and Schafer in experimental bleeding were the result of rapid exsanguination and it is possible that there is room for further work with slower rates of bleeding.

Again, although the direct effect of a severe haemorrhage is to cause a fall in blood pressure, the vaso-motor centre may become more active resulting in a peripheral vaso-constriction which may be excessive (Avery Jones 1947) and produce an increase in pallor.

Clinical impression represents the integration by balanced judgement of all factors.

Study of the case history especially with regard to duration of pain and the differentiation of acute from chronic ulcer, detection of complicating disease, collection and assessment of

the various criteria of severity, presence or absence of recurrence and general impression of the patient's appearance, progress and condition at any given time all contribute to the composite picture.

All the foregoing considerations help to establish a probable or tentative diagnosis of the cryptic lesion but further attempt should be made to place the matter beyond dispute.

In haematemesis Radiological examination may still further clarify the picture in the following manner.

(1) Localization of the site of haemorrhage in viscera normally or abnormally situated within the thorax.

(2) Exact localization of site of haemorrhage in stomach or duodenum.

(3) Detection of accompanying perforation.

(1) Localization of the haemorrhage in viscera normally or abnormally situated within the thorax.

The source of bleeding may not be in stomach or duodenum but in thoracic vessels which have ruptured into the lumen of the oesophagus.

Two such spectacular haemorrhages have occurred in my presence.

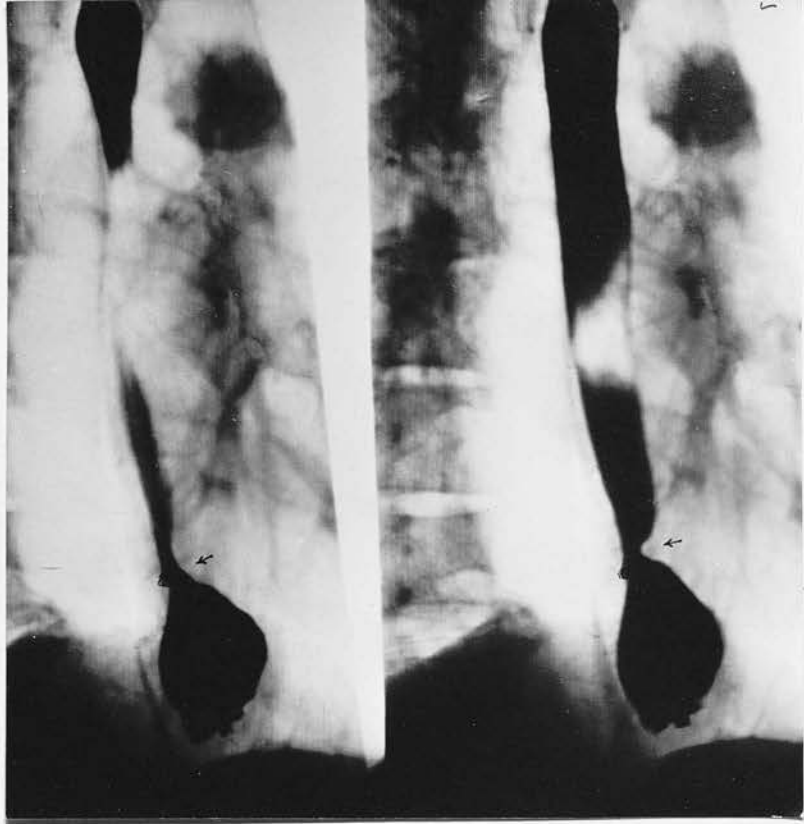
The first was due to haemorrhage from lower oesophageal varices and terminated a case of portal cirrhosis in a dipsomaniac lawyer; the second was due to the rupture into the oesophagus of an aortic aneurysm in a syphilitic charwoman, whose wig fell off at the time of death revealing a total alopecia. This catastrophic type of haemorrhage is of theoretical interest only, as death is inevitable.

In this collection haemorrhage of thoracic origin occurred in:

- (a) Ulceration associated with "congenitally" short oesophagus.
- (b) Thoracic pouch of a paraoesophageal hernia.
- (c) Varices associated with portal cirrhosis.

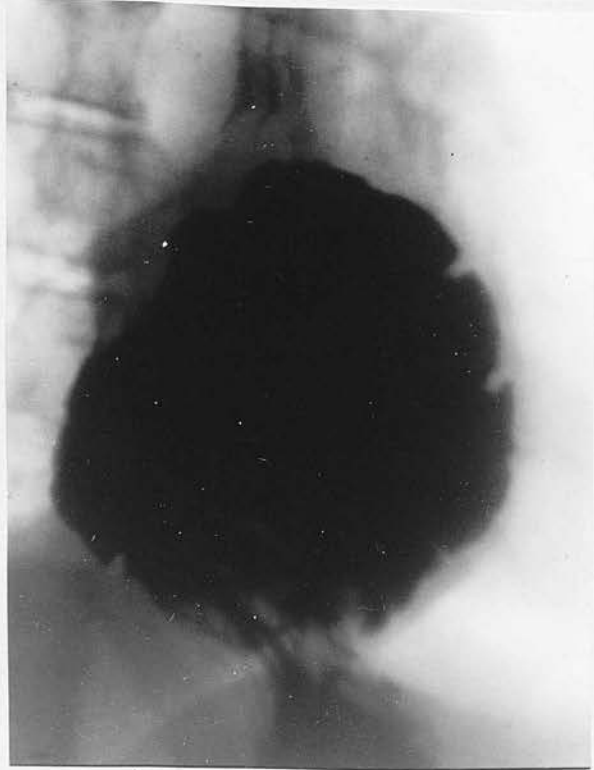
(a) (Case 25) Ulceration associated with a "congenitally

Radiograph 1.



"Congenitally" short oesophagus
with peptic ulcer.

Radiograph 2.



Paroesophageal hernia after
Haematemesis.

short oesophagus. Radiography revealed two narrowings of the gullet, the upper one showing the crater of a small ulcer, the lower one due to stomach herniation. The cardiac end of the stomach showed excess of mucous covering and the duodenal cap was deformed from an old healed ulcer, of which there was a history fifteen years previously. Barium enema revealed disordered segmentation of the colon.

Successive radiographs revealed progressive healing of the ulcer and, ten weeks after haematemesis, examination with the oesophagoscope was considered safe.

The narrowing was seen, not tight; no growth, extrinsic or intrinsic. A white patch of fibrous tissue near the posterolateral surface of the narrowing was believed to be the site of a healed ulcer and corresponded with the crater seen earlier by x-ray.

The upper oesophagus was very capacious, flabby and mobile; the cardiac orifice was more open than usual and the stomach was drawn up into the thorax.

The second case (Case outside this series) where haemorrhage had been due to ulceration associated with short oesophagus presented a picture almost identical with the above, but her bleeding had been both severe and recurrent.

She was not under the personal care of the writer but had been admitted to the surgical ward where she was under the clinical observation of the surgeon.

He was satisfied that the case was one of chronic peptic ulcer and decided to perform laparotomy as her condition was deteriorating despite repeated transfusion.

No source of bleeding was found in the stomach or duodenum and, since she presented no signs of portal cirrhosis, he closed

He then sent her down for x-ray examination and an oesophageal ulcer was found as in Case 25.

I visited her in the surgical ward and found her lying flat in her bed; she was immediately sat up and no further recurrence of her haemorrhage occurred.

It is suggested that routine examination by x-ray would prevent such unnecessary laparotomy and recurrence of haemorrhage.

(b) Bleeding from the thoracic pouch of a paraoesophageal hernia, associated with a mobile lower end of oesophagus of normal length.

(Case 119) Screen examination of the first case revealed an immobile left diaphragm. In the first oblique position the posterior mediastinum was seen to light up more brilliantly than usual at its lower end; this abnormal translucency was due to a thoracic magenblase.

A swallowed bolus, followed down the oesophagus was seen to enter the fundus below the left diaphragm but this could only be appreciated during the brief interval in which the paste coursed down the lower oesophagus which deviated medially round the herniated fundus.

Immediately after passage the oesophageal walls were approximated and the lumen obliterated by the pressure of the adjacent stomach.

The size of the gastric hernia was increased by the supine position and decreased by tilting erect but it was not completely reducible.

Two cases showing this same appearance were investigated after haemetemesis; they were both sthenic women in the seventh

Radiograph 3.



Paraesophageal hernia showing gas in thoracic
loculus in posterior mediastinum.

decade. (Cases 110: 119).

The first complained of breathlessness and symptoms resembling angina before the haematemesis (case 119). There was tightness across the chest and breathlessness on exertion but tightness accompanied by paraesthesia and formication down both arms had also appeared at rest. There was a moderate hypertension of $\frac{170}{100}$ mms.Hg: the left ventricle was somewhat enlarged and the aorta showed some atheromatous unfolding with calcification in the arch.

The electro-cardiograph showed left sided preponderance and ventricular extra-systoles only.

I have examined the gastric loculus in this case repeatedly when there has been exacerbation of symptoms; the size of the pouch varies considerably from time to time, depending on posture and gaseous distension of the stomach and colon. When symptoms are marked and the loculus large there is always temporary immobility of the left diaphragm.

The second case (Case 110) had no "anginal" pain but complained of high epigastric pain related to food and was sufficiently orthodox for the surgeon to make a provisional diagnosis of gastric ulcer.

On the two radiological examinations there was no palsy of the left diaphragm in this case.

Oesophageal hernia may be associated with gastritis, multiple erosions or actual ulceration in the thoracic loculus; the associated lesions are the source of bleeding. (Miller & Doub)⁵⁹

Oesophageal herniae are discussed by Åkerlund who divides them into three types:

1. A true partial thoracic stomach due to oesophageal growth not keeping pace with septum transversum;

2. Paraoesophageal type. The oesophagus is of normal length and there is an irreducible hernia of the stomach fundus.

3. Reversible type of hernia with the cardia and pouch of the stomach slipping freely through the hiatus, the cardiac canal being within the sac.

He quotes Schatzski as saying that the third type is common and fully developed in old age; it occurs, he states, in 50% of people between 65 and 83 and nearly always in the sthenic type. The incidence is increased by colonic inflation to 73%.

Hyperacidity with regurgitation through the lax cardia may produce gastritis or multiple erosions of the stricture neck as in the two cases in my series, or it may produce sizeable ulcer on the lesser curve at the passage through the hiatus. (Miller and Doub.)

49

Johnstone believes that it is the subsequent cicatrization which produces the "congenital" shortening and that this may drag up the mucosa and produce heterotopic islets at the lower end of the oesophagus. (The pull may not be uniform all round the lumen, some areas of mucosa may be dragged up more than others).

The "congenitally" short oesophagus in the elderly, he thinks, is acquired in this way. He associated himself with Allison and Royce in saying that they have never found a simple ulcer in an oesophagus of normal length and that it is always associated with a partial thoracic stomach.

He adds a foot note however, to say that the publication of his paper was delayed, and, since writing it, one such verified case occurred in a youth of twenty three in whom no proved hiatus hernia was found.

Spasm, pressure from the hernial sac and "actual

are the reasons given by Porter Vinson for the narrowing of the oesophagus at its lower end.

Samuel quotes Hodges, Snead & Berger as saying that the circular protuberance or filling defect at the lower oesophageal orifice may be mistaken for a pathological condition and that the mucosal pattern is stellate which is still further suggestive of ulcer.

It is true that this appearance has caused doubt in the mind of radiologists but if, on rotation, no bud can be demonstrated and there is no lasting hold up of an opaque spot of Barium in a crater, the site of its occurrence at the lower oesophageal orifice decides its normality.

Atrophy of the diaphragmatico-oesophageal membrane as age advances and the loose attachment of the peritoneum at the cardia are the causes of hiatus hernia according to Harrington.

He believes that congenitally short oesophagus is a very rare condition and is distinct from hernia since the protruded portion of the stomach is not surrounded by a hernial sac.

Templeton also believes it to be rare and has never met a case.

(c) Varices from portal cirrhosis.

The demonstration of varices in the lower oesophagus and/or varicosity of the short gastric veins supports the diagnosis of haemorrhage from portal venous obstruction and may aid in determining the cause of an enlarged spleen.

An otherwise normal barium meal helps to exclude any other accompanying cause.

The typical large "soap bubble" appearance was described by Samuel in a recent paper.

The first demonstration of oesophageal varices was made

by Wolf in 1928. He employed a thin barium paste and showed rounded, circumscribed areas on the posterior oesophageal wall produced by varices.

The author employs a meal of a thin creamy consistence according to the formulæ recommended by Hampton (p. 44); this meal made up to six ounces appears to be about the right consistence and was used for all cases of recent hæmatemesis.

Various techniques to demonstrate the dilated veins are recommended by different authors; some use a thick, others a thin paste. A thick paste may obscure the varices since it is difficult to control the size of a semi-solid bolus swallowed and the mechanical pressure may empty distended veins. The patient is screened in the first oblique position, both erect and semi-prone and should be rotated to secure the best view.

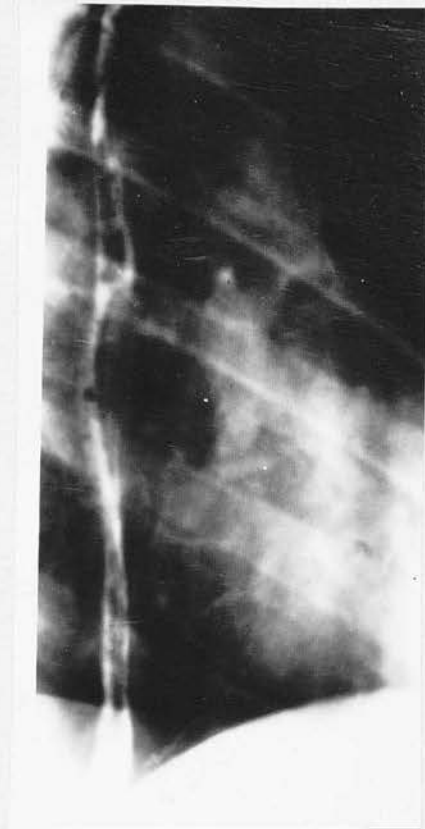
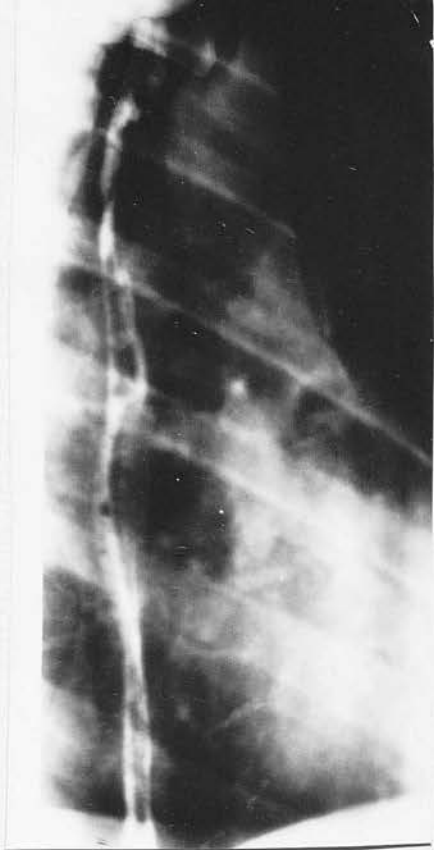
Contraction of the oesophagus empties the veins and diastole should be awaited to secure films. The writer observes the oesophagus through the aperture of a serial compressor with a film in position; the most opportune moment is seized and a radiograph taken after the patient has been instructed to take a deep breath in and hold it, since the inspiration normally produces the maximum distension of the thoracic veins.

There may be delay in thoracic emptying with varices as they may partly occlude the lumen.

If the lower end of the oesophagus is not seen filled, an attempt may be made to produce regurgitation from the cardiac end of the stomach by Valsalva's manœuvre. The patient is instructed to take a deep breath in and hold it and then bear down as if trying to move the bowel; this is, in fact, attempting a forced expiration with the glottis closed and a retrograde flow of Barium may then outline the varices.

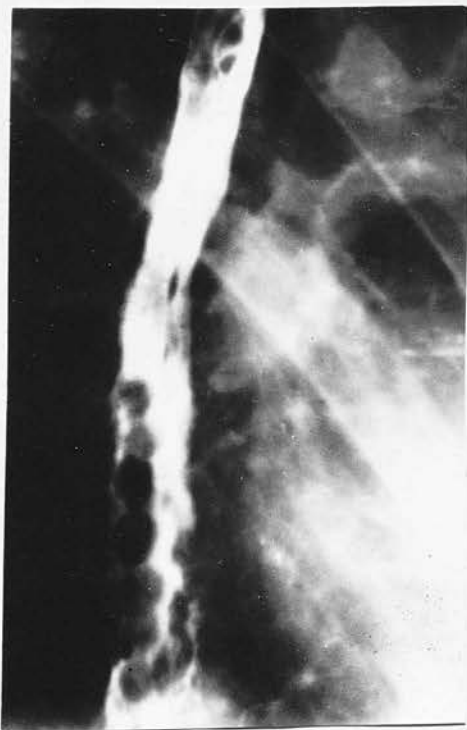
In varices the indentations are seen when the oesophagus is

Radiograph 4.



? Varices in portal cirrhosis 5 days after haemorrhage.
Oesophagus kept under observation but never went into
diastole.
Doubtful circular defects which might be due to air.
Varices have not been shown in this series during stage
of emergency.
Diagnosis by radiography has not been positive except in
intervals between bleeding. See Radiograph 5.

Radiograph 5.



Taken 23 days after bleeding.
Well marked filling defects.
Oesophagus in diastole.

relaxed whereas the "cork screw" type are seen only on contraction; conversely, polypoid nodular infiltration does not disappear completely on contraction. Varices in the fundus are occasionally seen as small rounded masses up to 2 cms. in diameter, around the periphery of which the Barium collects; this may be associated with a splenic enlargement of sufficient size to indent the lateral wall of the stomach. They are said to vary in size with respiration and oesophageal varices and splenic enlargement which accompany them differentiate them from gastric neoplasm. (Templeton).⁷⁴ Although sought in cases which have recent haematemesis, varices in the lower oesophagus have rarely been observed by the writer. In cases known to have oesophageal varices they have not been demonstrated. The oesophagus never relaxed nor could it be filled by Valsalva's manoeuvre in the writer's experience. It may be that recent haemorrhage relieves the congestion temporarily, and that this collapse of the veins is further aided by a fluid diet, rest in bed and oesophageal spasm. The accompanying radiograph shows the appearance of the lower end of the oesophagus, five days after a haematemesis due to portal obstruction; although the oesophagus was observed at intervals for an hour, no diastole occurred; this has been the usual appearance. In late cases where degeneration of the muscularis propria has occurred, spasm is no longer possible.

The radiographic demonstration of oesophageal varices appears to be comparatively rarely possible soon after haematemesis. Radiologists in Manchester, Liverpool, Ulverston and many other North West Hospitals were approached for an example, required for teaching purposes; none was able to produce one.

Radiology, nevertheless, is of use in excluding any other source of bleeding and persistent oesophageal systole after

haematemesis is very suggestive of varices.

In late cases where the diagnosis is more obvious on clinical examination, varices may be seen after the muscular degeneration, mentioned above, has taken place,

71

Tanner (1950) quotes Rowntree et al. (1947) in stating that the introduction of a special Miller Abbott tube with the bag removed from the end, and reinserted 8 - 10 in. higher up will impede the connection between the hypertensive portal and the systemic azygos vein and check bleeding. He shows a radiograph after haematemesis showing oesophageal varices. He does not say whether any muscle relaxant such as amyl nitrite was given or not; to do so, soon after haematemesis, and then distend the varices would appear to invite a recurrence of the bleeding. It is possible that a longer period after bleeding had elapsed before radiography was attempted than in my cases, as Tanner was considering radical cure. Alternatively, the cases may have been more established so that muscular degeneration had occurred.

(2) Localization of the exact site of haemorrhage in stomach or duodenum.

It has been customary to delay the administration of a Barium meal for three weeks after haemorrhage according to the dictum of H.H. Berg who stated "I have never dared to examine a patient before three weeks after a dangerous haemorrhage". His remarks applied to normal routine examination using a heavy meal and a Berg's compressor.

A perforation, personally observed at Liverpool Royal Infirmary in 1927, during the course of screening a stomach filled with the old type of heavy meal and with the patient erect, supported this opinion.

The introduction of liberal feeding by Meulengracht gradually allayed my fears and it seemed unlikely that the addition of a

small amount of Barium to the fluid given would make any difference, particularly if the patient were screened lying down.

Consequently it was decided in 1945 to undertake Barium meals as soon as the patient had recovered from the initial shock and to make use of intervals between bleeding however brief, if the patient were not shocked or collapsed, as it was considered that surgery might be necessary when the case had been diagnosed from the length of history as a chronic ulcer.

43

The meal advocated by Hampton in America was used. The impossibility of persuading local surgeons to operate, without a categorical assurance that they would find a lesion which could be dealt with surgically, made this necessary; point blank refusal had been the usual answer.

Hampton's formula is:

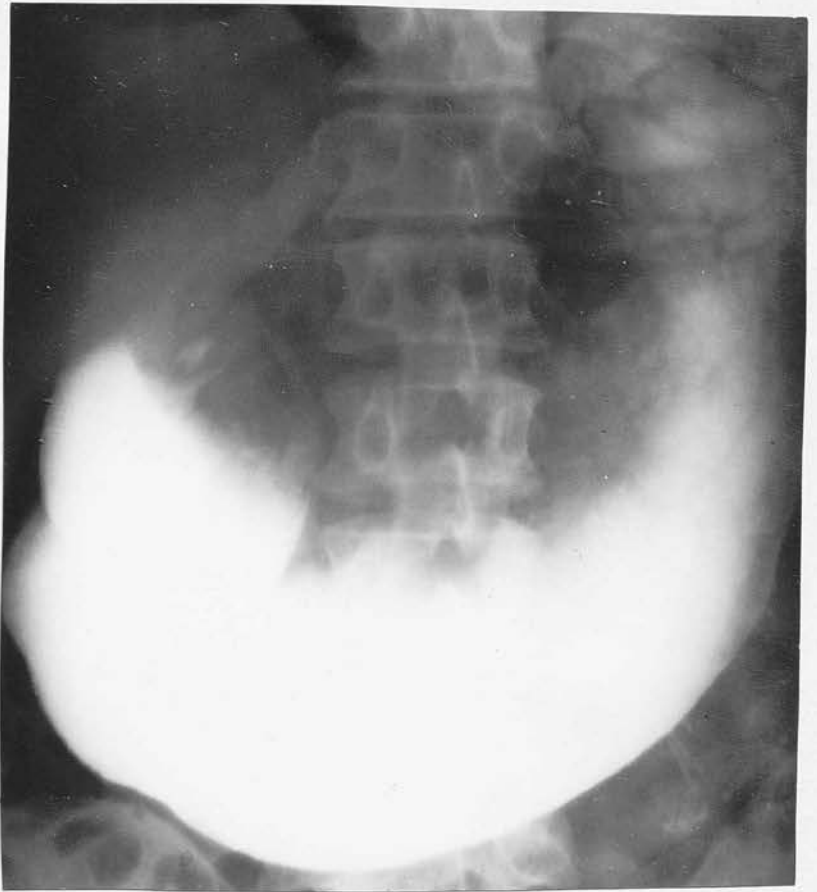
Plain Barium sulphate	4 oz. (volume);
Water	3 oz. (volume);
Plain petrolagar	1 teaspoonful;
Total mixture	4 oz. (volume).

The addition of the petrolagar to the mixture produces a thixotropic like change and Hampton had found that the mixture would adhere to the mucosa even when blood was present. The volume was later increased to 6 oz.

The examination is made with the patient horizontal and without compression or palpation.

The patient is laid first upon his right side and he is left in that position until the duodenum has filled and emptied two or three times and he is then rotated back towards the left side until the pyloric valve and first part of the duodenum are seen in profile. This rotation to the left carries the air from the fundus into the distal antrum and duodenum, and, when the duodenum has emptied, a mucosal relief pattern of the wall may

Radiograph 8.



Pyloric stenosis. Duodenal cap never seen filled.
No crater in stomach. There was a small twenty four
hour residue.
Partial gastrectomy performed four days after moderate
haemorrhage because of 1) Stenosis; 2) Recurrences are
badly borne by cases with accompanying stenosis. (Case 90).

Radiograph 6.



Ulcer crater on lesser curve during stage of bleeding.
Dealt with by partial gastrectomy. (Case 120).

Radiograph 7.



Ulcer crater on lesser curve during
stage of bleeding.
Dealt with by partial gastrectomy.
(Case 88).

be seen in much the same way as colonic relief with double contrast enema. This is observed through the aperture of a serial compressor without bringing the compressor down on to the abdominal wall and serial radiographs are taken at the opportune moment.

Hampton says that it is important to examine the distal part of the duodenum with particular care, for the point of bifurcation of the gastro-duodenal artery is opposite the postero-superior wall of the first part of the duodenum.

The dangerously bleeding ulcer is situated in close proximity to this large vessel and this type of ulcer is more distant than usual from the pylorus. (Allen. Balfour).
5 11

No bleeding ulcer in this series which required surgical interference and which showed a crater in the distal part of the duodenum was met, after this method of examining the duodenum, during the acute stage, was begun.

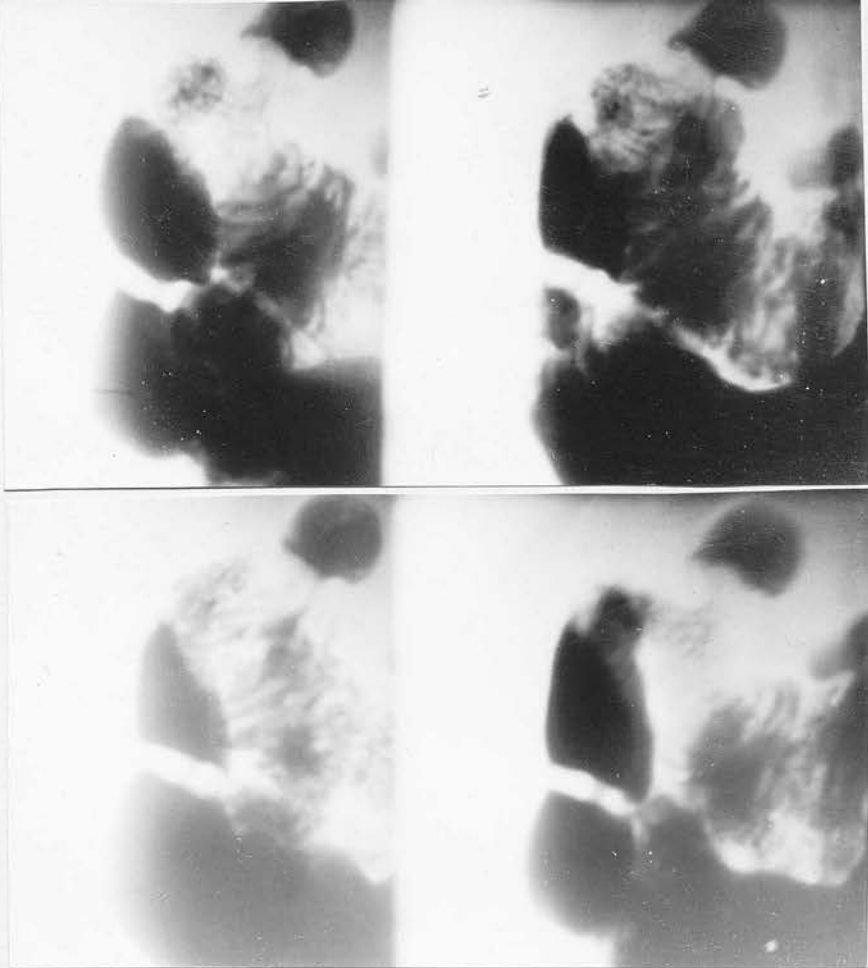
The only radiographed duodenal ulcer which required surgical treatment (case 90) showed a pyloric stenosis with much residual fluid in the stomach and a twenty four hours residue; the duodenal cap was never seen.

During the search for such an ulcer however, cases of severe recurrent haemorrhage were met, all of which showed easily demonstrable large craters on the posterior surface of the stomach or on the lesser curvature. (Cases 120,88,86,87).

These type 111 recurrences were transfused and successfully dealt with by partial gastrectomy (except 87). Since no palpation is attempted, fixation is determined by deep respiration, and this sign may indicate the depth of penetration and the amount of dissection likely to be required.

Diverticulum of the duodenum may produce haemorrhage and a fatal case of such haemorrhage is recorded by Witts.

Radiograph 9.



Radiograph 10.



Series of diverticulum of second part of duodenum. This produced haematemesis. Note abnormal course of duodenum.

One case due to diverticulum of the second part of the duodenum occurred in this series. The duodenum was anatomically abnormal in position. (Case 66).

Diverticula lower down the alimentary tract were also present in this case, as they almost always are, and these may be the source of continuing melaena.

Jejunal diverticulitis may also be the cause of haematemesis and/or melaena and it is now my practice to include the upper coils of jejunum in any radiological investigation for the source of bleeding. If the stomach empty at a normal rate this usually requires only re-radiography at half an hour after the Barium meal.

In 1948 the writer became a whole-time radiologist in the National Health service and the former mixed type of practice ceased.

In the last two years (1949-50) nine cases of jejunal diverticula have been seen in the x-ray departments of the Preston and district group. Two of the cases had haematemesis and melaena (only nine such cases have been found in world literature), and in both these the radiological findings were confirmed at operation.

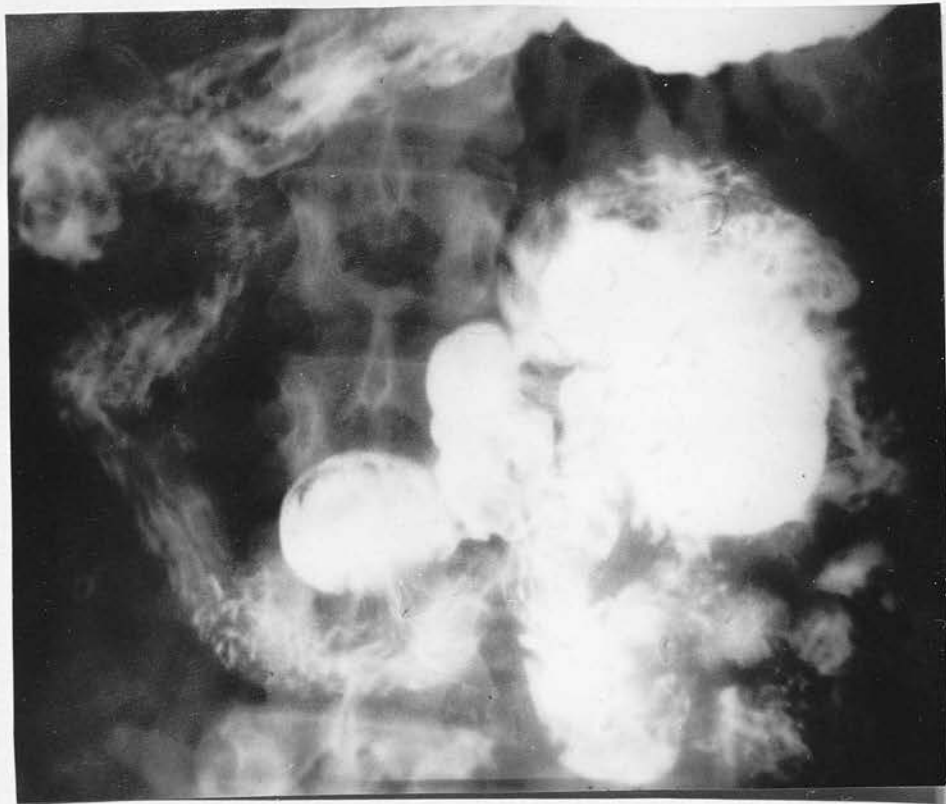
This series, reported by Orr and Russell, is in hand for publication and will shortly appear in the British Journal of Surgery. (September 1951).

The accompanying radiograph shows jejunal diverticula in one of the cases which had haematemesis and melaena.

Absence of an ulcer crater in stomach or duodenum, but evidence of abnormality due to gastritis, makes it unlikely that surgery will be useful.

In the stomach proper, the opinion of radiologists is, or has been until recently, that the diagnosis is best left to the

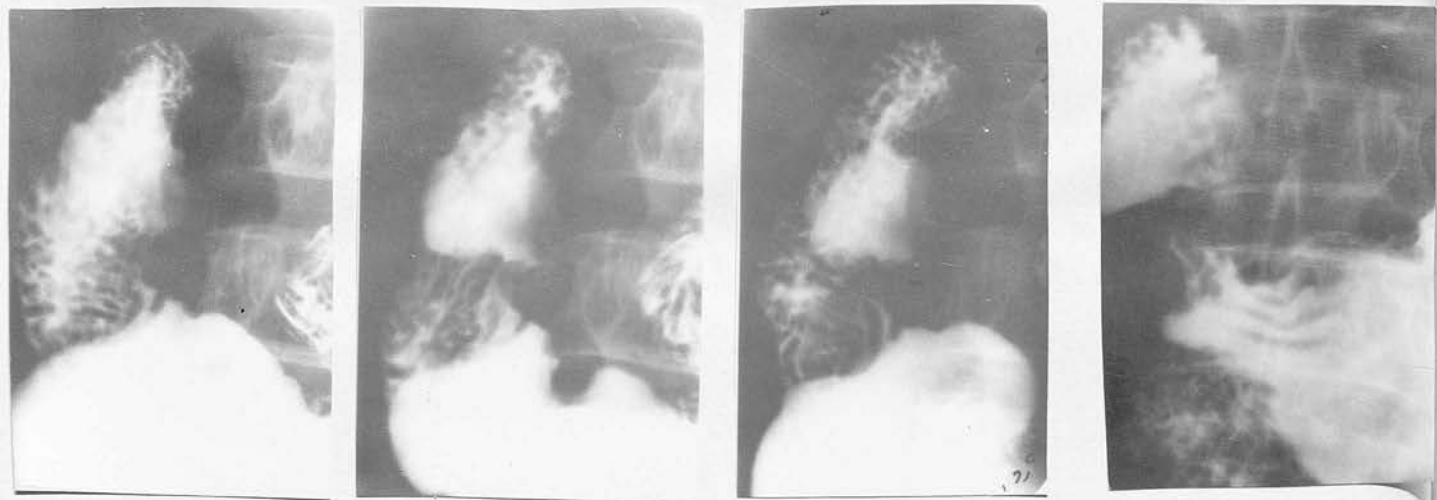
Radiograph 11.



Diverticula of third part of duodenum and of upper jejunum which produced hæmatemesis.

(Confirmed at operation).

Radiograph 12.

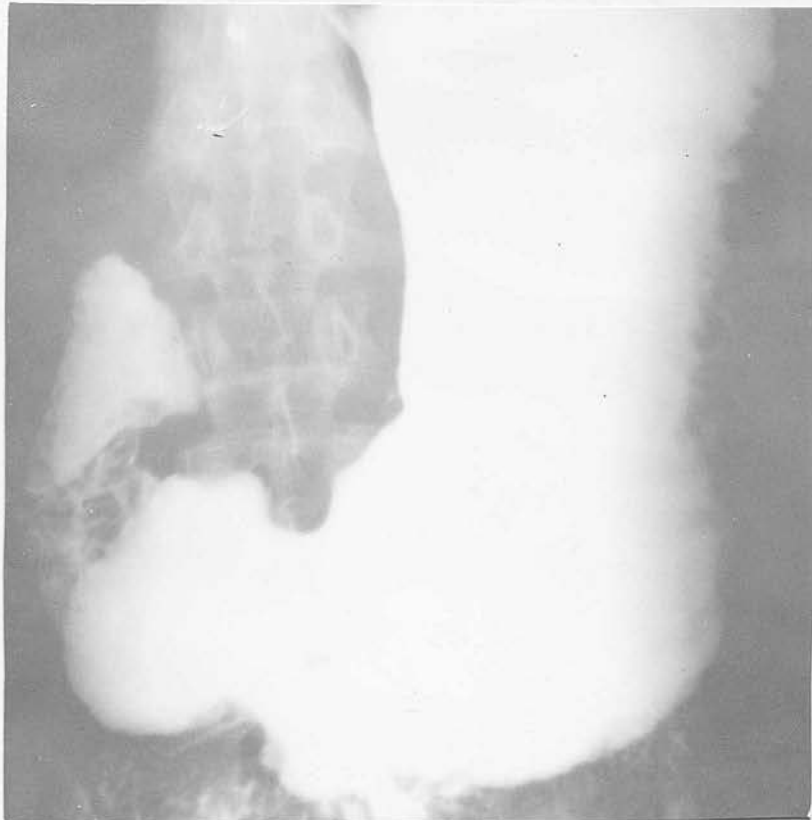


Serials of case of hypertrophied stenosis of pylorus in an adult. This was associated with antral gastritis and a small (undetected) ulcer on the lesser curve. Note inordinate length of pyloric canal.

Serial 4 with compression shows blurring of rugal pattern of antrum.

Twining states that the mucosal folds over the hypertrophied muscle are almost absent and that the hypertrophied mucosa is bound down to the hypertrophied muscle by an unusually thin submucosa.

Radiograph 13.



Same as above.

gastroscope - a principle which applies to all cavities open to direct inspection. The pyloric antrum is not always accessible however, and local antritis produces observable radiological changes.

68

Samuel quotes the following opinions;

Vaughan (1945) states that in antral gastritis there is

- (1) Temporary or persistent spasm of pyloric antrum associated with impaired, irregular and ineffective waves.
- (2) The mucosal pattern is often replaced by coarse folds often running transversely.

Mather Cordiner adds to this the presence of hypersecretion which blurs the outline of the mucosal pattern since the barium fails to adhere to the stomach wall.

Prepyloric narrowing as a sign is stressed by Golden; the spasm may prevent the barium from entering the pylorus, the smooth mucosa in the antrum being strongly suggestive of scirrhus carcinoma.

The spasm disappears after atropinisation.

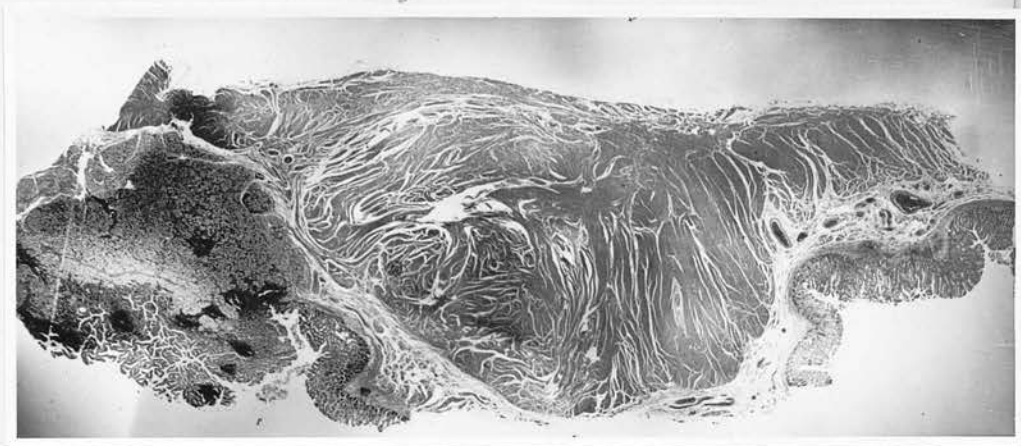
Multiple spiking or crenation of the stomach wall may be present if there are associated superficial erosions.

Two cases were observed by the writer which showed signs of gastritis in the pyloric antrum, one of which was admitted for melaena.

Case 1 had a markedly elongated pyloric canal which was clearly due to hypertrophy of the pyloric muscle and, associated with this, there was non-filling of the distal portion of the antrum and a smoothness due to disappearance of the rugae; on applying pressure these could only be seen in the distal portion.

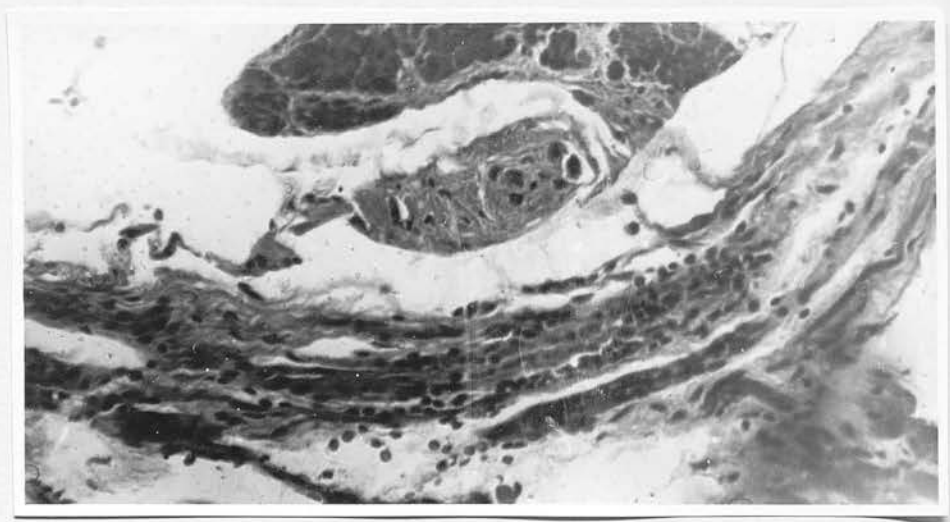
Although this was reported as non-malignant and due to hypertrophy of the pyloric muscle associated with gastritis, the surgeon was not satisfied and performed a partial gastrectomy.

Section 1.



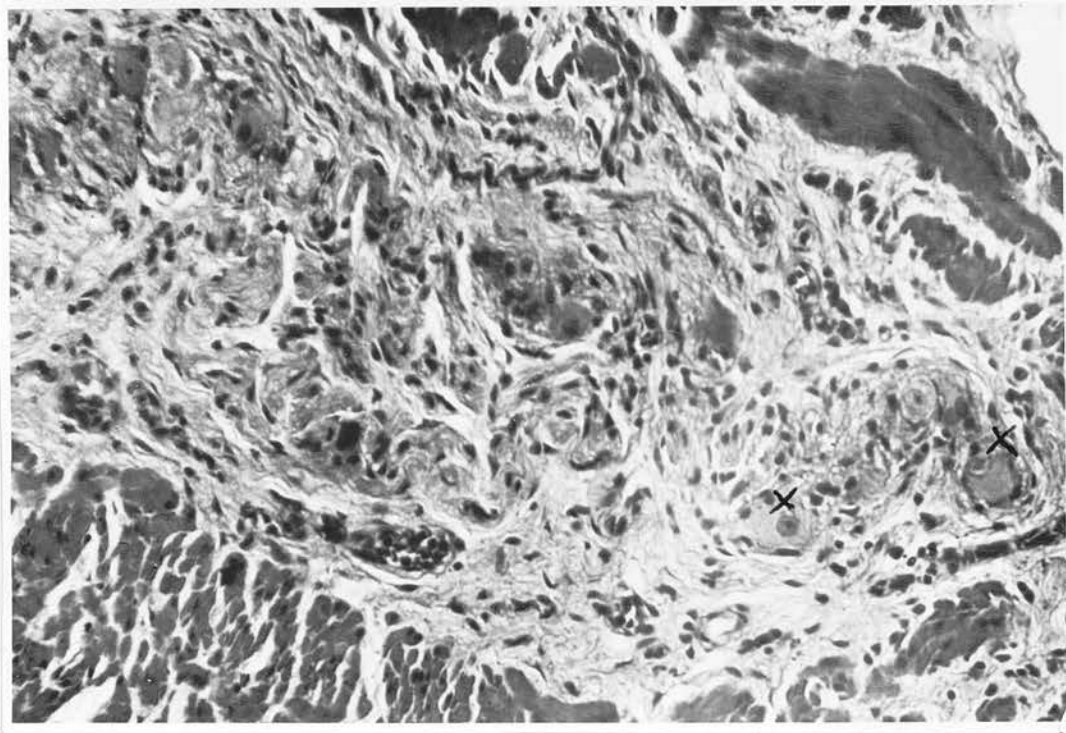
Section of hypertrophic pyloric stenosis. (Low power).
Cleft, described by Twining, dividing pyloric muscle
from prepyloric muscle and containing submucosa,
peritoneum and connective tissue only, is not present.

Section 2.



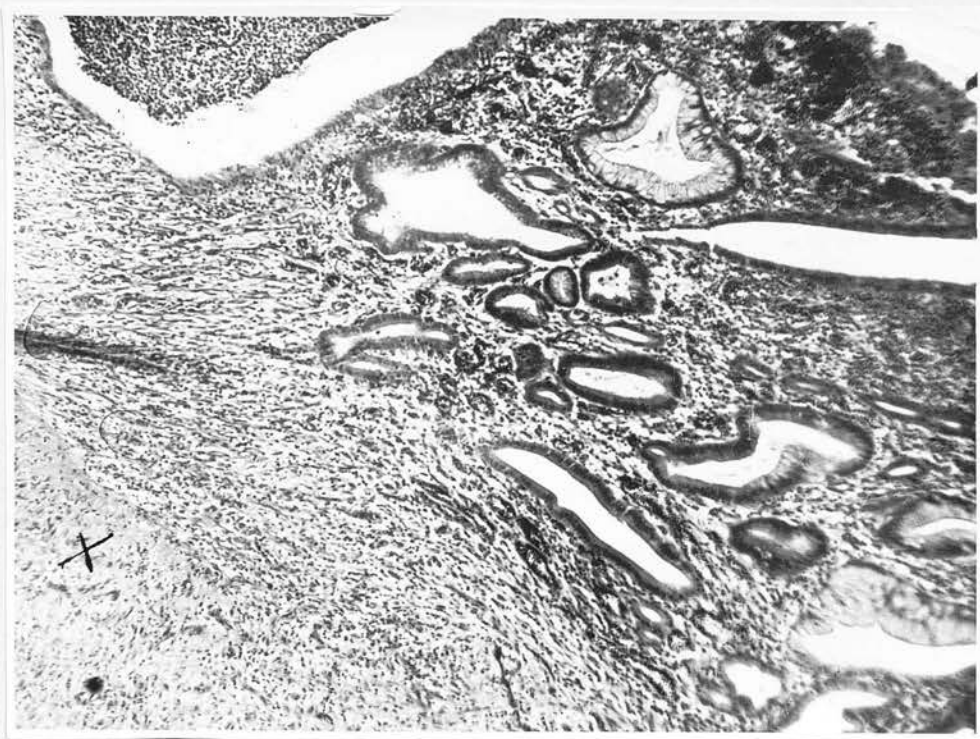
Section showing lymphocytes and plasma cells
in hypertrophied muscle.

Section 3.



Section showing round cell infiltration of muscle and many ganglion cells lying in muscle, e.g. at X and X.

Section 4.



Section through floor of small ulcer on lesser curve. Muscle bundles at X have been penetrated by inflammatory reaction and are shown just at point of discontinuity.

Examination of the resected specimen showed a marked hypertrophy of the muscle of the pyloric canal and of the distal portion of the antrum associated with a profuse round celled infiltration lying between the hypertrophied muscle fibres. A small chronic ulcer was also present on the lesser curve.

60

Morley mentions this condition as a cause of error in the diagnosis of carcinoma. He has records of 9 cases of hypertrophic pyloric stenosis in adults of cancer age. He does not state whether they had an associated gastritis though he says that the condition may be associated with a peptic ulcer in the stomach or duodenum or unassociated with any ulceration.

78

Twining believed that the circular muscle of the pyloric canal only was hypertrophied. Morley states that his own sections do not show this clear demarcation between prepyloric and pyloric musculature; the sections in my cases do not show this demarcation either, and the muscle of the pyloric antrum is also much hypertrophied and there is no clear cut notch between, such as Twining describes.

The third case of Twining's series showed "chronic inflammatory cellular infiltration in the mucosa and submucosa, extending down to the muscle and between its bundles".

Eight hour retention was present.

Neither of my cases showed any retention, the stomach was empty in three hours in both.

Case 2, showed a markedly smooth pyloric antrum - no rugae could be seen at all. A laparotomy was performed, nothing abnormal beyond thickening of the stomach wall was seen, a specimen portion of this was removed for microscopy and the abdomen closed.

Section showed hypertrophy of the antral muscle accompanied by a marked eosinophil infiltration - eosinophil granuloma, and

It would appear ~~that~~ this muscular hypertrophy may be
60
reversible for Morley reports a case of his, which was x-rayed
by E.D. Gray in 1934, and presented the typical x-ray appearance
of hypertrophic pyloric stenosis; operation nine years later
revealed a chronic ulcer of the lesser curvature, for which he
did a Schoemaker gastrectomy. There was then no hypertrophic
pyloric stenosis.

Both the above mentioned cases were radiographed after
this series of 120 cases had concluded.

Twining was an accurate observer and it would appear that
two different conditions are present here.

He regarded his cases as congenital hypertrophic stenosis
which had persisted into adult life.

My case of hypertrophy of the pylorus showed well marked
ganglion cells in the section and is, therefore, not an achalasia.

All these local changes have been seen during the period
under review in this paper, though these cases of gastritis were
not necessarily associated with frank haemorrhage; many who had
alteration in the rugae and irregular antral contraction were
completely relieved of their pain by atropinisation.

86

This is not unexpected for Wolf and Wolff state

"The contractions themselves caused pain, when they were
of sufficient magnitude, but not when the mucosa was pale and
non-oedematous."

(3) Detection of accompanying perforation.

Sub-phrenic gas is a well known sign of visceral perforation;
the occurrence of gas in other sites is not so well known. The
likely whereabouts of such gas should be appreciated since
perforation may be a complication of haematemesis.

Gas may be seen in the abdomen -

1. As a post operative pneumoperitoneum - gas from this source alone may take up to 16 days to absorb.
2. After tubal insufflation.
3. In acute perforations of the stomach, duodenum or bowel.
4. In subdiaphragmatic abscess and other infections (by gas producing organisms).

The first two items of (3) are the only ones which directly concern us here since they may accompany massive haemorrhage.

The appearances of free gas in the peritoneum are the same in perforation of any part of the alimentary tract provided that the perforation is into the greater or lesser sac and not into the posterior retro-peritoneal space.

No localisation of the perforation can be made from a radiographic demonstration of gas; this localisation depends on the history of the case or on some previous demonstration of a lesion in a part of the alimentary tract.

12

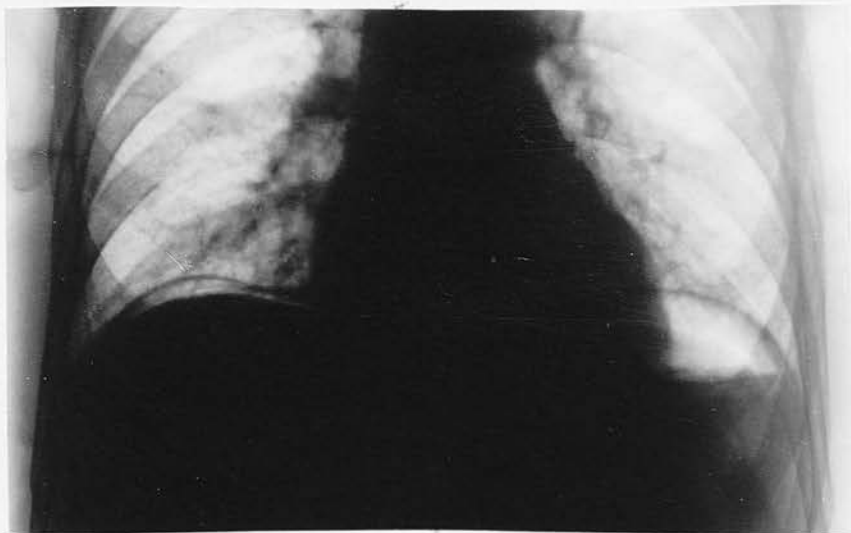
Bannen points out that if free gas is found in some other region than the subdiaphragmatic, in the upright position, a posterior retro-peritoneal perforation should be suspected. This gas does not alter its position on tilting the patient since it is really an emphysema of the loose retro-peritoneal areolar tissue.

Retroperitoneal perforations of the duodenum occur as a result of peptic ulcer but they are more commonly the result of trauma - a kick or blow.

Extravasated gas may be found at

- (1) The root of the transverse mesocolon.
- (2) The root of the mesentery of the small bowel.
- (3) Around the right kidney.
- (4) Into the flanks as a subcutaneous emphysema.

Radiograph 14.



Subphrenic gas after perforation of duodenal ulcer.

Radiograph 15.



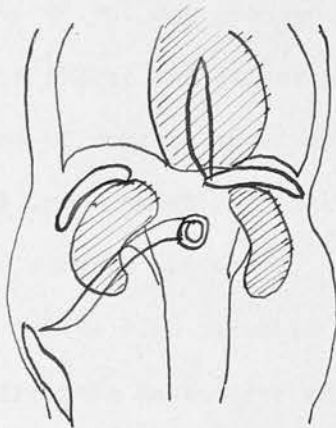
Gas in lesser sac after perforation of gastric ulcer.

(5) Under the leaf of the left diaphragm and

following the great vessels into the chest as a paravertebral gaseous shadow.

Because of the possibility of retroperitoneal duodenal perforation the above facts should be kept in mind. The writer has never seen this type of gas shadow.

The lesser sac may also contain gas. In the upright position gas in the lesser sac becomes loculated at the upper and right border of the sac and shows as a gaseous translucency above the normal gastro-intestinal tract. (Bannen).



Distribution of retro-peritoneal gas
in posterior perforation.

(After Bannen).

The right border forms a well defined curvilinear shadow in the right paravertebral region, representing the right lateral limit of the sac.

When perforation into the greater sac is suspected a careful and critical search of the subdiaphragmatic region should be made as the amount of gas may be minimal - a thin clear area separating the diaphragm from the upper border of the liver.

The tube should be centred a little above the xiphisternum and the exposure made in full expiration with the patient upright; in the lateral decubitus the gas shifts into the flanks.



These examinations should be made immediately, as small amounts of gas may absorb in twenty four hours. Subdiaphragmatic gas shows a predilection for the right leaf though it may also be found on the left side (the splenic shadow may show up clearly) or beneath both diaphragms.

As general peritonitis develops, general gaseous distension from paralytic ileus and the homogeneous shadow of ascites may be added.

In Bannen's opinion, perforation of the duodenum often occurs unsuspected and may recover spontaneously.

The writer shares the latter part of this opinion, as a number of cases of minimal sub-diaphragmatic gas, which have shown clinical improvement during the first twenty four hours, have recovered without operation. Larger amounts of gas which remain always in the same position on movement of the patient, or which acquire some particular and constant irregular shape, are probably loculated by adhesions and the case may be left alone if there is clinical improvement. In perforated peptic ulcer of the stomach or duodenum the symptoms are not always so startling as might be expected and may be masked by administration of morphia, by old age, or an accompanying haemorrhage (case 26); a scout radiograph early in this case might have saved the patient's life.

The shock was out of ~~prop~~portion to the amount of blood lost and should have aroused suspicion of accompanying perforation.

Treatment.

The two outstanding advances in the treatment of massive haemorrhage from the gastro-intestinal tract are undoubtedly continuous drip transfusion and liberal feeding.

These are the two essentially life saving procedures of modern therapy.

(a) Liberal feeding.

58

Meulengracht was not the first to suggest an early and liberal diet, Cruveilhier had recommended it in 1835 and Spriggs had used it in 1909 and had treated his subsequent cases at Banff and Ruthin in this way.

It would appear that when Meulengracht suggested it in 1935 the time was ripe for its universal acceptance.

The arguments put forward by the supporters of a liberal diet are:

1. Healing is promoted by a full diet, with adequate vitamins.
2. Acid is neutralized by food and clot digestion prevented.
3. Dehydration and starvation are combated.

The most important factor supporting Meulengracht's arguments was his low mortality rate (1%); this was a spectacular improvement on previous results.

His arguments received further theoretical support from Blalock's experimental findings that starvation and dehydration aggravate secondary shock.

52

Avery Jones (1947) used Meulengracht's regime and had a mortality of 48 out of 580, i.e. 8%; in this series of cases with the same method it was 10 out of 110, i.e. 9%.

The percentage of positive x-ray examinations in this series was 55%, 60% in Jones' collection and only 30% in Meulengracht's paper. The impression created, by reading Meulengracht now, is that his cases were mainly of a mild type and his low figure for positive findings suggests that his series included many cases of acute ulcer.

In all fatal cases in this study a lateral window was seen in a large artery at post mortem examination, and, as one gazed at

this lesion, it was difficult to believe that anything short of surgical treatment could have prevented the fatal outcome or that, in this particular case, the recurrence of haemorrhage from an eroded artery of this size could have been prevented by diet.

This question of liberal feeding is, however, linked with fluid requirements and the prevention of dehydration.

51

Jones (1939B) proved that many deaths occurring after haematemesis were due to uraemia, the haemorrhage having ceased some days before. Sufficient fluid had not been given to these patients to make good the loss due to normal daily output, vomiting and haemorrhage.

It was therefore probable that uraemia was largely due to dehydration, as Blacklock found that shock, which is an accepted cause of renal damage, was aggravated and prolonged by dehydration - a permanent cortical shunt had been established in these cases. It is also known that the loss of electrolytes, particularly sodium, is capable of affecting renal function adversely.

56

(McCance).

The routine in my cases was to allow $3\frac{1}{2}$ litres of glucose saline per day by mouth; if there had been persistent vomiting or haemorrhage this was given intravenously.

47

Rectal saline was given by Hurst and Ryle (1937) but as there is often diarrhoea with melaena, or constipation with rectal loading (increased by morphia) this method was not used.

At first normal saline was given but it was found that pulmonary oedema was apt to follow, especially with insufficient oversight in a busy ward.

The oedema is the result of an excessive concentration of sodium ions in the tissues, and it was considered safer after administering $3\frac{1}{2}$ litres of normal saline to infuse a 4.1% glucose

in $\frac{1}{5}$ normal saline whenever more fluid was required; the solution is isotonic, while the glucose provided against ketosis.

The low sodium content gives it a wide safety margin; yet the minimum sodium requirement is supplied especially as $3\frac{1}{2}$ litres of normal saline have already been given.

It seems likely that the danger of sodium retention is real in massive haemorrhage from the upper alimentary tract because the kidneys, already affected by shock, may be incapable of preserving the normal electrolyte balance in the plasma.

The above procedure was adopted on the advice of G.M. Arnott⁸ - a colleague on the hospital staff.

Malnutrition as an indirect cause of death was denied by Hurst and Ryle (1937), nor did they believe that Meulengracht's methods were life saving. Their patients were starved for forty eight hours and then allowed only sips of iced water.

If the bleeding recurred this treatment was repeated, so that the patient's condition with recurrences might deteriorate rapidly as a result of the treatment alone.

It was then thought that food might mechanically dislodge the clot and prevent the stomach from "resting" and as late as 14 1942, Bennett, Dow & Wright re-iterated this argument although it was known that the stomach contracted most violently when empty.

(b) Blood transfusion.

The evaluation of the improvement directly due to liberal feeding is confused by the coincidental introduction of continuous drip blood transfusion which began to be universally employed about the same time.

Transfusion rapidly restored the depleted blood volume - the cause of the immediately fatal issue; liberal feeding maintained the general condition during what might be, if there were recurrent bleeding, an illness of considerable duration and

Blood transfusion was regarded as harmful and dangerous prior to 1935 and was little practised in the provinces.

This view received additional support from the papers of Cullinan and Price and from Hurst and Ryle who quoted and supported their arguments.

Current prejudice retarded the universal use of this remedy for some time but it soon became apparent that liberal transfusion, after the institution of blood banks, had reduced the mortality from haemorrhage.

The arguments against transfusion at that date and their refutation are now summarized;

(1) Reactions due to misgrouping and pyrogens.

These technical difficulties are now understood and have been overcome.

(2) Early transfusion increases blood volume with a

consequent rise in pressure; this was thought to dislodge the clot and produce further bleeding.

Evidence of this theoretical objection has never been produced; in most cases in this series, haemorrhage did not recur until twelve hours or more had elapsed after the blood pressure had returned to normal.

Bleeding did recur, however, when the blood pressure was still low from the previous haemorrhage (case 38). Recurrence is probably due to digestion of blood clot, to further erosion of a blood vessel or to final rupture of the previously leaking aneurysm.

(3) The surgical field was obscured by blood after transfusion.

This fallacious argument was refuted by Bennett, Dow and Wright, who pointed out that no surgeon would

operate on a shocked patient without giving a transfusion.

- (4) The risk of infective hepatitis or an infection disease, e.g. syphilis, is negligible in an emergency. Infective hepatitis is rarely due to whole blood although comparatively common after transfusion with plasma.
- (5) The danger of overloading the circulation, particularly in cases
 - (a) suffering from co-existing heart disease,
 - (b) in the hyperkinetic phase of haemorrhage
70
(Sharpey-Schafer)

can be overcome and the advantages of transfusion obtained by:

- (1) Administration of drip slowly - one pint in eight hours.
- (2) Inspection of veins of neck, where an early rise in venous pressure may be observed (Lewis)⁵³ and auscultation of the lung bases for moist sounds. It was realized that the first symptom of overloading of the right heart was dyspnoea which preceded either of the above signs, and particular reliance was placed on this sign.

If there be any doubt of the ability of the heart to deal with increased load, a preliminary essay may be made by transfusing with normal saline. If the venous pressure rises it is then necessary to proceed with the administration of blood with more than usual care.¹ (Aird).

Despite all precautions, danger cannot be eliminated completely; it must be weighed against the risk of prolonged low blood pressure and circulatory stasis especially in patients with arteriosclerosis. In older people this state of shock is more likely to become irreversible and there is danger in

allowing this to persist even for a short time and it is also in these people that complicating coronary and cerebral thrombosis have been frequently reported.

During the course of this enquiry, two cases of severe anaemia both in females between 60 and 65 years were seen; both were in a state of congestive failure associated with primary achlorhydric anaemia of long standing - the haemoglobin being about 20%. In deciding to give transfusions it was realized that these patients were suffering from cardiac anoxia and were in urgent need of blood.

This was given slowly as a concentrated red cell suspension (McQuaid and Mollison)⁵⁴, and a gradual subsidence of the level of venous filling in the neck resulted, followed by a decrease in the size of the liver and heart and disappearance of the oedema. Mention of these cases is relevant here, because the danger of transfusion in the hyperkinetic phase of haemorrhage is associated with the raised venous pressure and circulation rate found in anaemia.

If these hearts, which had been subjected to continued anaemic anoxia could tolerate transfusion it is to be expected that this treatment would be better borne when the anoxia was of short standing.

Older ideas about the harm done by transfusion were perpetuated, as mentioned above, by the paper of Cullinan and Price³⁴ (1932), which was quoted by many authors between its publication and the second World War.

This paper stated that:

- (a) When transfusion was delayed it was not followed by recurrent bleeding.
- (b) The mortality rate was higher when transfusion was given early to control haemorrhage - of 17 cases, 8 died.

It was concluded from the above that transfusion, given early, does not prevent further haemorrhage but actually causes it, and therefore blood should not be given early, but only after haemorrhage had ceased, to expedite the recovery from anaemia. It now appears obvious that these conclusions are untrue; transfusion does not stop haemorrhage in peptic ulcer and is not given for that reason; continuous drip transfusion is used to meet a state of emergency - haemorrhagic shock.

It seems probable that those patients, who recovered without transfusion and received blood late for anaemia, were of a mild type, whilst those who received it early were severe cases.

It is not surprising therefore that the mortality in the latter group was higher, particularly as the amounts of blood given were small (200 - 500 ccs).

These erroneous beliefs were propagated in paper after paper,⁴⁷ and Hurst and Ryle recommended late transfusion for anaemia in amount of 10 ozs.

They stressed that anaemia, rather than oligaemic shock,²⁸ was the cause of death and quoted Christiensen, an alien and independent opinion, in support of this view. He asserted that the mortality figures in Scandinavia had risen after the introduction of transfusion.

In favour of transfusion are the following arguments:

1. With wider experience of the use of continuous drip transfusion in the treatment of haemorrhage from all causes, the life saving value of the method is now established and is common knowledge. The fall in mortality is the best answer to any criticism from the practical point of view, and, from the theoretical point of view, the practice supports Blacklock's findings. He gave transfusions to dogs and found their recovery from secondary shock more rapid; humans are entitled

The transfusion enables the patient to withstand a further depletion of his blood volume and if operation becomes necessary, the surgeon is confronted by a better operative risk.

2. Cases of amaurosis have been seen following gastro-duodenal and other haemorrhages (Post Graduate School, Hammersmith). The cause of this is unknown, but it has been postulated that the optic atrophy, usually found in these cases, is due to vascular spasm associated with diminished blood volume; restoration by transfusion should be a preventive measure.

3. Tissue anoxia has been cited as the cause of the devitalization of the parenchyma of organs.

These viscera, e.g. stomach, may be the site of later surgery. At post mortem examination of one of the fatal cases in this series (No.87), the stomach wall was found to be astonishingly thick and oedematous and cases have been reported where sutures have torn through devitalized tissue.

Hurst attributed surgical failure to technical difficulties rather than to low haemoglobin; he mentioned friability of tissue as one of the difficulties but apparently did not associate this with anoxia - the probable cause.

Indications for transfusion.

84

In 1937 Witts laid down certain indications for transfusion which had not been universally accepted before that date; these were

- (a) Haemoglobin percentage below 40%.
- (b) Pulse rate over 140 per minute.
- (c) Blood pressure below 90 mms.Hg. (systolic).
- (d) Blood urea over 100 milligrammes per 100 cubic centimetres.

These are the indices of a severe haemorrhage and their value has been discussed in the section on Severity.

Other authors give similar criteria but quote variable figures for haemoglobin: e.g. Avery Jones (1947) - 40%, Hurst and Ryle (1937) - 30%. Papworth and Lontitt (1943) - 60%.

In this study Witts figures were found acceptable and of practical value excepting those for blood urea and pulse rate; a pulse rate of 140 per minute was terminal only.

These figures viz:

(a) Haemoglobin below 40% (not very useful when haemodilution had not occurred).

(b) Pulse rate over 120 per. min.

(c) Blood pressure below 90 mms of mercury (systolic)

were used in combination with

(d) The clinical impression

to decide whether continuous drip blood transfusion should be given.

Continuous drip transfusion was the method used in all cases where transfusion was employed; the rate at which the drip was given was the usual of forty drops per minute. In cases of marked shock with severe blood loss the rate of flow was increased and conversely, the drip was given more slowly where there was cardiac insufficiency.

No limit was imposed on total volume; this varied with the needs of the case.

Surgical treatment.

A certain number of cases - those classified as Type III - continued to bleed and their condition to deteriorate in spite of medical treatment.

Surgery is then the only means of controlling the haemorrhage and should be considered.

The difficulties in assessing the relative merits of medical and surgical treatment were discussed earlier (p. 13) and some of the fallacies resulting from misleading and inadequate statistics considered.

Many objections to surgery have been raised and some of these reproduced from papers already surveyed are now detailed and discussed.

1. The diagnosis of chronic ulcer is sometimes difficult; the history may not be suggestive and a radiographic examination may not have been made.

This difficulty was found to be infrequent in practice and the duration of pain related to food was found to be a reliable guide in the diagnosis of chronic ulcer.

Later in the enquiry when the diagnosis was in doubt, a categorical answer could often be given to the surgeon by x-ray examination during the acute stage of bleeding by the method already described. (p.44)

This method of examining all cases of chronic ulcer by x-ray was also extended to include those cases where there was any suggestion that the symptoms were unorthodox, especially if the bleeding were recurrent; useless emergency operations on conditions outside the stomach and duodenum (see Radiology), or on cases of gastritis were thus avoided.

It quickly became evident that surgeons were much more willing to open an abdomen if it could be stated that "This patient is bleeding from a large chronic penetrating ulcer at a particular site".

Late in this series cases of this Type 111 were treated by gastrectomy with complete success after selection by the means suggested above and had this procedure been adopted at the beginning the mortality would have been much reduced.

2. Mortality is higher than with expectant treatment even when the operation (partial gastrectomy) was performed during a period of choice.

This does not apply with modern surgical methods, anaesthesia and post operative care.

In the hands of a good experienced surgeon, the mortality rate is very low.

3. Liberal transfusion and feeding have greatly lowered the mortality and increased the success of conservative treatment.

It seems probable that if all cases of gastro-duodenal bleeding from chronic ulcer were treated by operation during the acute stage of the haemorrhage, the mortality would be higher than with expectant treatment; many would certainly be opened without need.

If the cases are carefully defined and selected and those which continue to deteriorate, in spite of liberal feeding and transfusion are recognized early and treated surgically there must be a further fall in the mortality figures.

Conservative treatment fails to save these severe cases as they are due to erosion in a large artery, involved in fibrous tissue, in the floor of a chronic ulcer (this is all that is meant by defining them as type lll).

Liberal feeding and transfusion serve the surgeon in the severe case just as much as they aid the physician in the milder case which now recovers with medical treatment alone.

4. A patient who has had a severe haemorrhage is gravely ill and surgery carries the risk of further shock, the risk however, of continued haemorrhage under the conditions now known to obtain in cases classified as type lll is not only greater but inevitably fatal.

Prolonged shock following repeated haemorrhage is as

dangerous as that following operation and is just as likely to become irreversible.

A low haemoglobin, for many days or weeks, should never be permitted, and, if after repeated transfusion, the haemoglobin percentage still remains low, surgery is necessary and should be carried out without delay to avoid prolonged anoxia and tissue devitalization.

5. Expert surgeons are not always available.

This is unfortunately true, but by closer co-operation between physician, surgeon and radiologist and the resulting keener interest in this branch of surgery, great improvements in diagnosis and technique are attained.

The best results, in the cases here reviewed, were achieved late, after this position had been reached.

In support of surgical treatment, Finsterer, Gordon-Taylor and Bohrer claim that the risk of protracted haemorrhage is greater than the risk of operation because most of the severe cases are due to an open vessel in the floor of a chronic ulcer.

In forty five cases of severe haemorrhage Chiesman found thirty and Jones thirty two such lesions in forty five post mortems in his series of 580 cases.

The contention of this paper is that Surgery should be selective and the type of case in which it must be used is defined.

It is appropriate here to review the fatal cases; details of these and comments are contained in Appendix 1.

Fataleties.

It is of value to examine fatalties in retrospect particularly if procedure is still controversial; startling defects in management may be revealed which stimulate further attempts to avoid repetition of errors in the light of real personal experience.

The fatal cases may be divided into four groups:

(i) Those on whom the surgeon refused point blank to operate. (Cases 1: 38: 79).

These might have been saved by experienced surgery. Case 38 was only 40 years of age, and although his general condition was deteriorating, he was in sufficiently good condition to stand operation.

(ii) Those whom operation failed to save. (Cases 6: 62: 87). Failure of surgery may be partly attributable to mismanagement, due to delay in sending the patient to hospital, or in delay in consulting the surgeon, or to an error of judgement by the latter.

This is another group in which, with more experience, the number of fatalities would have been reduced; they are never completely eliminated for even in the best gastro-enterological units, deaths still occur.

(iii) Those whose deaths follow immediately after an unexpected catastrophic haemorrhage. (Cases 63: 71). These are the most alarming and disturbing.

One is feeling pleased with the progress with conservative treatment, when there is a single profuse haemorrhage and the patient dies in a matter of minutes.

This is unpredictable and is due to the rupture of an aneurysm which has previously leaked.

It may occur at any time after the first leak.

It can be prevented only by recommending operation for all cases of bleeding ulcer (Finsterer's mortality is 5%) and there would be then many unnecessary operations.

(iv) Those whose death was partly attributable to complicating disease. (Cases 81: 92).

This disease may be a contra-indication to surgery, e.g. Case 92 who weighed 21 stone and suffered from adiposis dolorosa;

Late surgical results at Preston began to be good after a definite diagnostic procedure had been laid down and with the later appointment after the war of a more experienced surgeon, and it is clear that surgery has a definite place in treatment during the emergency within the limits defined.

The important question as to whether all cases of massive haemorrhage must have operative treatment now arises. If it had been so employed in this series certain cases might have escaped death; but the mortality associated with such universal surgery must also be considered.

73

41

Gordon-Taylor's and Finsterer's figures for mortality of 10% and 5% respectively are low, but few provincial surgeons have the technical skill of these two pioneers in this branch. They both state that the mortality from conservative treatment is greater than with surgery but include certain indications for operation when bleeding is not massive.

Gordon-Taylor recommends that all cases, when a recent radiograph has shown a large penetrating ulcer or where there is a long and definite ulcer history, whatever the degree of haemorrhage, should be treated surgically.

Finsterer increases the range of choice; he believes in operating on cases of doubtful chronic ulcer and advocates gastrectomy for the gastrostaxis of multiple erosions.

The above figures were obtained, therefore, by operating, not only on cases of massive haemorrhage but on mild ones also.

1

Aird had 7 fatalities in a series of 15 cases of massive bleeding dealt with surgically.

That the mortality in less able hands must be much greater is evident in my collection, and this is an argument against

surgery in all cases of massive haemorrhage.

The number selected for operation should be that minimum in which it is imperative.

Each case must be judged on its own merits and the condition of the patient repeatedly assessed; sound clinical judgement and constant observation are essential. If transfusion and treatment are compensating for the blood loss, and the blood pressure, pulse and general condition of the patient are satisfactory and provided that, after consideration of the known factors, this state of affairs seems likely to continue, conservative treatment may be continued; if not an operation is necessary.

The resultant mortality figures, by this method of selection, will perforce include deaths due to sudden, unpredictable massive bleeding but will exclude potential casualties from an unnecessary emergency operation.

Judgement of the value of emergency surgery in massive haemorrhage is difficult and discouraging from the earlier cases in this collection.

At first surgeons were disinclined to operate during the acute stage of bleeding. In a provincial town the surgeon is much nearer the populace than in a large teaching centre; if he has poor statistics, his reputation soon suffers; the public know nothing of the difficulties of treatment. Surgeons had little experience of this branch of surgery and feared they would not find a bleeding point with which they could deal.

This difficulty was later overcome during the acute stage by radiography and a definite assurance of the site of bleeding; this move ushered in a more successful period although a surgeon, who did not wish to co-operate, went to the other extreme and performed a laparotomy for oesophageal ulcer.

At the beginning point blank refusal to operate was the rule and it soon became obvious that the practice of admitting most cases of haematemesis and/or melaena to a medical ward and then a day or two later seeking the opinion of a general surgeon, who had in fact no personal experience of the outlook in these cases, except in the post-operative stage, was irrational.

Only by repeated observation of the patient from the time of admission, coupled with previous experience of the case history and the various degrees of bleeding and by radiography, can that case be selected in which bleeding is unlikely to cease without surgical treatment, and which will be benefited by it.

In the early, inexperienced phase, errors of judgement were consequently made, (see detailed report of severe haemorrhages, Appendix 1).

No attempt is made to blame surgeons; rather it is desired to stress the close co-operation necessary between physician, surgeon, radiologist and general practitioner. Friendly co-operation and the gradual accumulation of experience by all members of the team must eventually obtain the best results.

Ideally, this is best achieved in gastro-enterological units in cities similar to those organized for thoracic and neurological cases, but in provincial hospitals this is impracticable and the general surgeon must be quite prepared to deal with the problem.

His success will depend upon the co-operation outlined above and on the previous experience of the team.

Many papers have stressed the high mortality in patients over 50 years of age. This is attributed to arteriosclerosis with failure of the blood vessel to contract and retract.

In this report there were deaths at the ages of 36, 37, 40 and 47. (Table VI).

In three of these cases the vessel was encased in a fibrotic

mass, which would appear to be a more important factor than arterial rigidity in preventing retraction; in the fourth case post mortem was not permitted.

It follows the refore, that it is wiser not to be influenced greatly by the age of the patient, but to attach more importance to the length of history, clinical progress and general condition of the patient in deciding for or against operation.

Ulcers may be long standing and therefore associated with marked fibrosis in older patients, and it is possible that this may be a factor in the higher mortality: there is also decreased resilience in the older animal.

T A B L E VI.

Mortality in various age groups.

Age.	Males.		Females.		Total Deaths.	Mortality.
	No.	Deaths.	No.	Deaths.		
20-29	4	0	7	0	0	-
30-39	19	2	4	0	2	8.7%
40-49	28	3	4	0	3	9.4%
50-59	25	2	3	0	2	7 %
60-69	8	1	4	1	2	16.6%
70-79	3	1	1	0	1	25 %

Before the recent war it was generally held that to operate after forty eight hours bleeding was nearly always disastrous, as the bowel sutures did not heal.

With rapid massive transfusion this is no longer true and results in this country show that "late" surgery is now often successful. (Aird).

This means that with liberal transfusion and feeding there is now a little more latitude in the time, during which it is

necessary to come to a decision, and the patient may be, on occasion, kept in reasonable condition for three or four days.

When operation is regarded as imperative, its nature must obviously vary with the patient and the surgeon; no rule of thumb can be applied. ⁷³ (Gordon-Taylor).

The simplest procedure is to open the stomach or duodenum and insert mattress sutures over the ulcer. This method although it may temporarily stop the bleeding does not always prevent recurrence (Cases 59: 87), and should be reserved for poor risks.

Partial gastrectomy has been successful in this series and it is now commonly held that this is the operation of choice.

The more important controversial aspects of the conduct of a case of massive haemorrhage from the upper alimentary tract have been discussed; there remain some minor points worthy of mention.

General Management. (Sedatives, posture etc.)

Complete mental and physical rest is essential.

A bed, surrounded by screens, in a quiet corner where there is not much traffic and from which no doors lead to lavatories or sluice rooms, is the best position in a general ward.

The propped up position was adopted here as a routine, as it was considered the best for nursing and transfusing anaemic patients. In this position too, the right auricular pressure is lower than in the supine and the danger of overloading the circulation is minimised.

In addition the likelihood of pulmonary complications is diminished and, because "cough is the watch dog of the respiratory tract", morphia was reserved for the unusually apprehensive and restless patient.

Barbiturates were preferred, phenobarbitone sodium was given subcutaneously in 2-3 grain doses as was necessary, later this was taken by the mouth.

As a general rule the patient was kept in bed until overt bleeding had ceased for two weeks. This was quite possible in general practice, but in hospital a rush of war casualties sometimes prevented this.

Aperients and enemata were withheld until bleeding had ceased for four or five days.

Secondary Vitamin C deficiency may occur in patients with chronic ulcer, particularly if complicated by haemorrhage.

65

This deficiency was demonstrated by Portnoy and Wilkinson, (1938).

Accordingly, two hundred milligrammes of Vitamin C were administered daily, parenterally at first and later orally; a liberal and modified Meulengracht diet was given throughout.

I am indebted to my colleagues on the staff. Drs. F.B. Smith and A.A. Miller (Pathologists), Mr. G.M. Arnott (Surgeon) and to a succession of house physicians for their enthusiastic collaboration in this enquiry.

Appendix IDetailed reports and comments on cases with massive haemorrhage.

Case 1. J.T.I. male, age 59.

Had severe melaena after vomiting blood on day prior to admission. Interval history of epigastric pain for 15 years, two hours after food and relieved by alkalies. Heavy beer drinker and pipe smoker.

On admission:

Clinical condition very poor; patient pale, collapsed and mentally confused - he could not give a rational history. Marked arterio-sclerosis of retinal and peripheral vessels.

Pulse 106. B.P. 160/50. Haemoglobin 40%.

Progress and treatment:

Routine; Morphia gr. $\frac{1}{4}$ given immediately. Blood transfusion started at once and continued since clinical condition did not improve and bleeding was more or less continuous. Ten pints given up to time of death - on third day.

Blood urea 2nd day (i.e. 24 hours after haemorrhage) 64 mgms %

3rd day 70 mgms %

Post-mortem findings:

Heart - 420 grammes. Left ventricle hypertrophied. Marked atheroma and sclerosis of aortic arch. Blood clot in stomach. Ulcer crater 1" wide $\frac{1}{2}$ " deep in duodenum near pylorus, ulcerating into convexity of curve of atheromatous artery, 3 mms in diameter.

Liver fatty. Spleen large and soft. Kidney shows atherosclerosis and endarteritis.

Death probably due to desiccation. (F.B.S.)

Diagnosis:

Chronic duodenal ulcer. Haemorrhage, Type III.

Comments.

Normal blood pressure probably very high, as judged clinically by the

arteriosclerosis and a pressure of 160/50 in spite of an obviously massive haemorrhage. It was realized that this patient was a poor operative risk but on the other hand it seemed probable from the degree of arteriosclerosis present that nothing short of surgery would stop the bleeding.

The surgeon was called in but steadfastly refused to operate.

Case 3. J.R. male, age 33.

Copious haematemesis began at 3.30 p.m. the previous day, and again just before admission. Previous attack three years ago. History of pain after food for three years.

On admission:

Clinical condition poor. Moderate pallor. Tongue furred.

Pulse 105 rising to 120. B.P. 85/50. Hb. 70% (4 hours later).

Treatment and Progress:

Routine: Blood transfusion (continuous drip) - 6 pints in first two days.

Blood urea 1st day 45 mgms %

2nd day 65 mgms %

3rd day 48 mgms %

5th day Hb. 65%

9th day Hb. 100%

There was no more bleeding.

Investigations:

Test meal - sustained hyperchlorhydria.

X-ray - chronic ulcer, lesser curve of stomach.

Diagnosis.

- 1. Chronic Gastric ulcer.
- 2. Haemorrhage, Type I.

Case 6. W.C. male, age 36.

Was admitted as a case of haemoptysis. "Brought up bright-red blood this morning". Had sensation of vomiting. (N.B.)

History of having had a thoracoplasty done 12 years ago for tuberculosis of right lung.

Severe epigastric pain, relieved by food, for past month. Three weeks ago vomited one pint of coffee coloured fluid.

On admission:

Clinical condition fairly good. Moderate pallor. Tongue furred.

Pulse 90. B.P. 115/75. Hb. 55%.

Chest x-ray - collapsed right hemithorax. Active subclavicular focus on left side with rest of lung peppered by multiple foci.

Blood urea - 1st day 42 mgms %

2nd day 50 mgms %

3rd day 65 mgms %

Patient had a further haemorrhage shortly after admission, pulse rising to 105; after continuous drip transfusion, this dropped to 80, two hours later. He then had a further haemorrhage, pulse rate rising to 125 and falling two hours later, following transfusion to 80. Three hours after this he had still another haemorrhage, pulse rate rising to 140 per minute and falling to 120 per min., two hours later.

At this point a laparotomy was performed. The stomach was turned up and palpated between finger and thumb. Nothing abnormal was felt. The surgeon, who before the operation, had held that the lungs were the source of haemorrhage, decided now that there was no doubt that this was so, and without opening the stomach, closed the abdomen. The patient continued to have attacks of haematemesis and in spite of massive transfusions died on the fifth day.

Post-mortem findings:

Lean. Exanguinated.

Surgical collapse, right side of chest. Upper median recent laparotomy scar sound.

Deviation of larynx and trachea to left.

Heart deviated to right.

Right lung adherent and had to be cut out, very small. Small abscess outer surface of apex of right lung with thorax as outer wall - ? tubercular. Left upper lobe hypertrophied and left upper chest very roomy and ballooned out in contrast to left lower chest which is flat. Encapsulated, calcifying, caseous node, about 5 mms in diameter at apex, left lung.

Left ventricle of heart relatively hypertrophied, otherwise heart normal.

Abdomen - Ileum and colon full of tarry melaena. Stomach contains $1\frac{1}{2}$ pints of partly clotted blood; ulcer 25-30 mms in diameter in mid-posterior wall, towards lesser curve, with pancreas as whole of base and bleeding point in middle; this is a lateral window in one of the main branches of the splenic artery, around which there is much fibrosis. Duodenum normal. Liver pale, friable and fatty. Gall bladder and spleen normal. Adrenal cortices deeply pigmented yellow.

Kidneys pale, capsules do not strip quite smoothly. (F.B.S.)

Comments.

(1) The tendency to attribute the source of haemorrhage to the lungs because of the bright-red blood which poured out of the patient's mouth without effort. Long history of chest trouble, and relatively short history of gastric trouble, seemed to support this view. The patient however stated that he experienced the sensation of vomiting.

(2) Comparatively short history of pain compatible, in this case, with marked fibrosis.

(3) Fibrosis responsible for failure of arterial wall to retract. This failure has usually been attributed to arteriosclerosis of the affected artery, none was present in this case.

(4) The importance of opening the stomach and carefully examining the interior when no ulcer is palpable and of carefully defining the stomach boundaries.

In this case the adhesion of the posterior wall of the stomach to the pancreas was mistaken for the lesser curve and the stomach was turned up,

hinged on this point.

(5) The importance of a co-operative and experienced surgeon.

The senior surgeon had refused to open the abdomen and the youngest consultant on the staff was the only one who would undertake this.

(6) It is possible that the associated tubercle produced "indigestion" which the patient regarded as normal and it was only when the pain became very acute that he regarded it as a gastric symptom.

Case 16. J.N. male, age 46.

Haematemesis just prior to admission and also on the preceding three days.

Five years history of epigastric pain after food, relieved by alkalies.

On admission:

There were no signs of shock but pallor was marked.

Pulse 124. B.P. 110/70. Hb. 47%.

Treatment:

Routine. Blood transfusion - 2 pints.

Progress:

After admission and for next three days, vomited altered blood and clot; there was also melaena.

Haemoglobin 4th day 38% - 2 pints of blood given.

8th day 53%

Four weeks later 78%

Discharged one month after admission.

Investigations:

Test meal - sustained hyperchlorhydria.

X-ray report - Juxta pyloric ulcer.

Diagnosis:

1. Chronic Gastric ulcer.
2. Haemorrhage, Type I.

Case 21. C.P. male, age 32.

Woke up last night and vomited three pints of blood. No previous history of pain or dyspepsia.

On admission:

Clinical condition good. No shock or pallor.

Pulse 64. B.P. 120/80. Hb. 90%.

Treatment: Routine.

Progress:

On the third day he suddenly became cold, weak and clammy and appeared pale and shocked. A little later he vomited blood.

Pulse 106. B.P. 85/50.

Two pints of blood were transfused. Haemoglobin was then 62%.

Fourth day B.P. 105/60.

During the next four days he had repeated attacks of haematemesis and eight days after admission the haemoglobin was 33% and blood urea 39 mgms per cent. Two days later he again vomited blood and two pints of blood were transfused. Hb. then 45%. As haemoglobin was still low one pint of packed red cells was given.

There was no further nausea or haematemesis and seven days later haemoglobin was 65% and his recovery proceeded smoothly.

Investigations:

Test meal - sustained hyperchlorhydria.

X-ray report - no abnormality seen.

Gastroscopy. "Hypertrophic gastritis" only.

Diagnosis:

1. Recurrent acute ulceration.
2. Haemorrhage, Type I.

Comments.

Although hypertrophic gastritis was reported by the gastroscopist, this seems an unlikely diagnosis, as the patient had no history of pain or dyspepsia.

It seems probable that the changes in the mucosa were the result of anxiety.

Case 23. D.J. male, age 36.

Haematemesis, 2-3 cupfuls, at first bright-red, darker later.

History of epigastric pain, 2 hours after food and relieved by the next meal for the past 11 years. Recently pain has radiated through to back below left scapula.

On admission:

Clinical condition good; slight pallor.

Pulse 96. B.P. 120/70. Hb. 85%.

Treatment: Routine.

Progress:

At 11 p.m. on night of admission patient suddenly collapsed with severe melaena and a few ounces of blood were vomited.

Pulse 60. B.P. 35/15.

Three pints of blood were transfused.

On second day at 11.30 p.m. there was another attack of melaena; he passed about one pint.

Pulse rate 60. B.P. 125/65. Hb. 48%.

The haemorrhage then ceased and he made an uninterrupted recovery, being discharged four weeks after admission with haemoglobin 82%.

Investigations:

X-ray report. Active duodenal ulceration.

Diagnosis:

1. Chronic duodenal ulcer.
2. Haemorrhage, Type I.

Comments.

The vaso-vagal reaction on two occasions is noteworthy.

Case 26. J.C. male, age 72.

Haematemesis this morning. Epigastric pain on and off for 12 years.

Had morphia gr. $\frac{1}{4}$ before admission (not known at time).

On admission:

Patient drowsy, very deaf and incapable of giving a lucid history.

Skin cold and clammy. Slight anaemia.

No abdominal tenderness or rigidity.

Pulse 100. B.P. 80/50. Hb. 80%.

Treatment: Routine. One pint of blood transfused.

Progress:

Pulse (7 hours later) 95

(19 hours later) 125

Died unexpectedly next morning.

Post-mortem findings:

1. General peritonitis.

2. Perforated duodenal ulcer, 1 in. in diameter just beyond the pyloric ring.

Comments.

Death due to general peritonitis following perforation. Slight haematemesis did not contribute to death.

Note (1) Importance of ruling out perforation before giving morphia for gastrointestinal haemorrhage (this case was sent in as a haematemesis). This patient was the uncle of a local practitioner who gave morphia as soon as he was called in. He was not suspicious of perforation.

(2) In spite of signs being obscured by morphia and old age, perforation should have been suspected as the degree of shock was quite out of proportion to the blood loss. Judged by colour of mucous membranes on admission, he did not appear to have lost much blood and his Hb. was 80%.

(3) Perforation should have been in mind; radiography of abdomen for subphrenic gas would have settled the matter.

Case 27. F.G. male, age 35.

Haematemesis yesterday. Epigastric pain at intervals during last eight

years.

On admission:

General condition good; no evidence of anaemia or shock.

Pulse 80. B.P. 100/70. Hb. 90%.

Treatment: Routine.

Progress:

Two hours later B.P. had risen to 125/80.

On third day he became shocked and collapsed and had severe melaena.

Pulse 120. B.P. 90/60. Hb. 53%.

Two pints of blood were transfused and he continued to have attacks of melaena, none of which was alarming. On fourth day since his pallor had increased another pint of blood was given. No more bleeding occurred.

Recovery uneventful. Discharged four weeks later, Hb. 75%.

Investigations:

Test meal - sustained hyperchlorhydria.

X-ray report - chronic duodenal ulcer.

Diagnosis:

1. Chronic duodenal ulcer.
2. Haemorrhage, Type I.

Case 28. P.M. male, age 53.

Haematemesis three hours before admission. Attack of melaena two days ago. History of epigastric pain two and a half hours after food, relieved by food and alkalies, for past four years.

On admission:

Patient pale and collapsed. Tongue dry.

Pulse 108. B.P. 85/40. Hb. 75%.

Treatment: Routine. Two pints of blood transfused.

Progress: Three hours after admission further haematemesis - about half a pint.

Pulse 100. B.P. 95/50. Hb. 53%.

On sixth day haemoglobin was 62%.

There were no more recurrences and before further investigations could be carried out he was transferred to E.M.S. Hospital to make room for casualties.

Diagnosis:

1. Chronic duodenal ulcer (probable)
2. Haemorrhage, Type I.

Case 29. C.P. male, age 33.

Haematemesis this morning. Had previous attack five months ago (case).
No indigestion or pain in the interval.

On admission:

Patient pale, cold and collapsed; skin moist.

Pulse 110. B.P. 95/55. Hb. 75%.

Treatment: Routine. Three pints of blood transfused.

Progress:

He continued to have attacks of haematemesis but his condition was never alarming. The recurrences were not severe and were compensated for by transfusion.

Fourth day Hb. 43%)
Seventh day Hb. 29%) Two recurrences in the interval.

Two and a half pints of blood transfused.

Ninth day Hb. 64%.

Sixteenth day Hb. 76%.

There had been no further bleeding. Discharged four weeks after admission.

Investigations:

X-ray report - No abnormality seen.

Gastroscopy. Stomach appeared normal.

Diagnosis:

1. Acute ulceration.
2. Haemorrhage, Type II.

Comments.

With the knowledge of the previous history and of the results of the investigations, no undue alarm was felt and no question of surgery arose.

Case 32. S.M. male, age 38.

Vomited two pints of blood this morning and then collapsed. History of abdominal pain and vomiting of dark green fluid for one week. No symptoms before this.

On admission:

Condition poor; patient pale and shocked, with dry furred tongue.

Pulse 100. B.P. 70/45. Hb. 70%

Treatment: Routine. Transfusion - three pints given.

Progress:

Continued to have attacks of stale melaena, probably not due to fresh bleeding. Fourth day. Haematemesis (about one pint). Three pints of blood given. After this Hb. was 58%.

Continued to have frequent melaena.

Ninth day Hb. 33% - two pints of blood given.

Eleventh day Hb. 38% - one pint of blood given.

Nineteenth day Hb. 48%

Twenty-sixth day Hb. 54%.

Weekly readings after this with iron administration showed steady rise, 67% - 78% - 85%.

Investigations:

Fractional test meal - sustained hyperchlorhydria.

X-ray report. Sharp, conical, penetrating ulcer crater at foot of lesser curve of pars media.

Diagnosis:

1. Chronic gastric ulcer.
2. Haemorrhage, Type II.

Comments.

Surgery not considered necessary as he was not collapsed after second attack of haematemesis and after this with transfusion and iron haemoglobin continued to climb.

Case 34. R.B. male, age 30.

Sudden haematemesis today - two pints.

Two days ago suddenly felt sick and weak. Had a gastro-enterostomy three and a half years ago. For three years before this he suffered from severe epigastric pain after food. A year after his gastro-enterostomy he had another operation - an emergency - an ulcer at the anastomosis had perforated.

On admission:

Collapsed and pale.

Pulse 100. B.P. 85/50. Hb. 60%

Treatment: Routine. One pint of blood given.

Progress:

Transfusion - 2 pints on following day

2 pints on third day after which Hb. was 67%.

No further recurrence; recovery uneventful.

Investigations:

Fractional test meal normal.

X-ray report - stomal ulcer present.

Diagnosis:

1. Stomal ulcer.
2. Haemorrhage, Type I.

Case 38. F.P. male, age 40.

Attack of melaena last night, did not produce weakness or fainting.

History of epigastric pain two hours after food, relieved by food and alkalis, for past twenty years. X-rayed seven years ago and duodenal ulcer found.

General condition good. No pallor or signs of shock.

Pulse 80. B.P. 105/70. Hb. 90%

Treatment: Routine. Discharged a week later to make room for war casualties.

Readmitted six days later with history of several severe attacks of melaena in past few days.

On examination: collapsed, skin cold and clammy.

Pulse 112. B.P. 95/55. Hb. 43%.

Treatment: Routine. Two pints of blood given immediately.

Progress:

Two days later he had a severe haematemesis.

Pulse 120. B.P. 80/30. Two pints of blood transfused.

He continued to bleed while drip transfusion was running and surgeon consulted with view to operation which was refused. Blood pressure rose to 100/60 after this transfusion and the condition temporarily improved but he had attacks of melaena on succeeding few days and received two pints of blood each day. His condition progressively deteriorated. Blood pressure fell again to 80/30 and pulse rate rose to 130-140 per min. He died on seventh day after admission.

Diagnosis:

1. Chronic duodenal ulcer.
2. Haemorrhage mild passing into Type III.

Post-mortem refused by irate widow who thought something should have been done to stop bleeding.

Comments.

This was at first a mild haemorrhage, which on readmission obviously belonged to the massive Type III class. In spite of large transfusions patient made no lasting progress.

The haemorrhage was more or less continuous and could not be made good by transfusion.

There is no doubt that the widow was right and that this patient should have had surgical treatment soon after re-admission.

Case 50. W.B. male, age 50.

Haematemesis yesterday, attacks of melaena since. Remained free from pain since discharge in 1943 (case) for about six months. Pain then recurred, one hour after food relieved by alkalies.

On admission:

Clinical condition poor. Appears very anaemic.

Pulse 80. B.P. 125/60. Hb. 22%.

Treatment: Routine. Two pints of blood transfused.

Progress:

Fairly severe attacks of melaena occurred about once daily for next six days and two pints of blood were transfused on each of the 3rd, 4th, 5th and 6th days. Haemoglobin was 30% on third day and 50% on sixth day - in each case estimation was prior to transfusion.

Eleventh day Hb. 76%

Recovery was uninterrupted.

Investigations:

Fractional test meal - normal.

X-ray - duodenal ulceration with marked deformity of cap.

Diagnosis:

1. Chronic duodenal ulcer.
2. Haemorrhage, Type I.

Case 53. J.W. male, age 48.

Vomited three pints of coffee coloured fluid two hours ago and then became unconscious.

History of epigastric pain half an hour after food, relieved by food and alkalies. Haematemesis three and a half years ago. Gastro-enterostomy thirteen years ago for duodenal ulcer with no subsequent improvement.

On admission:

Patient pale, cold and collapsed; dry tongue.

Pulse 52. B.P. 95/55. Hb. 83%.

Treatment: Routine. Two pints of blood transfused.

Progress:

On third day he had a further haematemesis; two pints of blood were given. Haemoglobin was then 66%. On fifth day another attack of haematemesis occurred and two more pints of blood were transfused. There was no further haemorrhage.

Sixth day Hb. 40%; two more pints of blood transfused.

Fourteenth day Hb. 69%

Thirtieth day Hb. 85%

Investigations:

Fractional test meal - histamine gast achlorhydria.

X-ray report - Stomal ulcer.

Diagnosis:

1. Stomal ulcer.
2. Haemorrhage, Type I.

Case 54. J.F. male, age 54.

Became suddenly giddy at work this morning and then vomited blood.

Previous attack fifteen years ago. No recent pain until six weeks ago when he developed pain across upper abdomen, unrelated to food.

On admission:

Patient pale, cold and collapsed with dry furred tongue.

Pulse 96. B.P. 100/60. Hb. 67%

Progress:

Much improved on next day but on fourth day he felt faint, weak and sickly and passed a black motion. Haemoglobin after this was 39% and B.P. 100/50 and a transfusion of three pints of blood was given. (It seemed he

must have been leaking.)

On sixth day melaena recurred.

Pulse 74. B.P. 90/50, and he received a further two pints of blood.

On eighth day there was another recurrence.

Pulse 84. B.P. 100/40. Hb. 27%

One and a half pints of blood were transfused followed by one, two, two, and one pints on successive days.

Thereafter no further haemorrhage occurred and on the seventeenth day the haemoglobin was 46%.

Twenty-first day Hb. 56%

Twenty-eighth day Hb. 80%

Investigations:

Fractional test meal - Histamine fast achlorhydria.

X-ray report. No abnormality seen.

Gastroscopy. Atrophic gastritis.

Diagnosis:

1. Gastritis. Recurrent acute ulcers.
2. Haemorrhage, Type I.

Case 55. J.S. male, age 43.

Vomited blood six hours ago. Previous attacks one week and two weeks ago. No indigestion until four weeks ago when he began to have pain, unrelated to food, in the left hypochondrium.

Drinks at week end only; about ten pints of beer and an occasional whisky.

On admission:

Patient cold, collapsed and sweating with dry tongue. He was very pale.

Pulse 100. B.P. 170/90. Hb. 22%.

Treatment: Routine. Transfusion - 2 pints.

Progress:

There was no further haemorrhage. Two pints of blood given on 2nd, 3rd

and 4th days.

Fourth day Hb. 51%

Eleventh day Hb. 51%

Fifteenth day Hb. 64%

On discharge at end of third week blood pressure was 190/90.

Investigations:

Fractional test meal - normal.

X-ray report - no abnormality seen.

Gastroscopy - normal.

Diagnosis:

1. Recurrent acute ulcer.

2. Haemorrhage, Type I.

Comments.

Although patient looked ill on admission, the blood pressure had fallen very little.

Case 62. W.R. male, age 53.

Vomited half a chamberful of blood today. Has felt weak for four days and has had a few attacks of melaena.

Gastro-enterostomy performed ten years ago.

On admission:

Patient shocked, mucous membranes pale, tongue dry.

Pulse 52. B.P. 90/60. Hb. 30%.

Treatment: Routine. Blood transfusion begun.

Progress:

There were a few recurrences of haematemesis and melaena on the first day and, in spite of continued transfusion, his condition deteriorated.

Laparotomy performed on second day. Large open artery from which there was profuse bleeding was found on the posterior lip of the stoma; it was caught in catgut sutures and the haemorrhage ceased. Transfusion continued during operation. Although there was no further haemorrhage his general

condition did not improve and he died on the third day.

Post Mortem report (T.C.) Incomplete.

Abdomen. No blood in stomach or bowel.

Lungs. Slight congestion of left lung base.

Heart normal.

Diagnosis:

1. Stomal ulcer.

2 Haemorrhage, Type III.

Operation 2nd day. Died 3rd day.

Comments.

Surgery decided on because after twenty four hours in hospital, despite transfusions, he was deteriorating. Death was probably due to irreversible shock - there was no blood in stomach or bowel at post mortem.

Delay in admission to hospital was perhaps a factor in determining degree of shock.

Case 63. J. McH. male, age 67.

Has had melaena for past 6-7 weeks and vomited dark blood last night.

Has had pain two hours after food for last eleven years.

On admission: Appeared very pale but did not look shocked.

Pulse 94. B.P. 150/70. Hb. 17%.

Progress:

Was somewhat improved on second day and two pints of blood were given to raise haemoglobin.

On fourth day Hb. 39% and patient improving. Later that day was seized with sudden severe pain in upper abdomen which became rigid and did not move on respiration. At same time he vomited twelve ounces of blood.

It was obvious that he had perforated and he was being prepared for operation when he had a further large haematemesis and died in spite of immediate and rapid blood transfusion.

A large ulcer, one inch in diameter, on the lesser curve of the stomach with a large open artery in its floor. Much fibrosis present. Microscopy showed this ulcer to be benign.

Diagnosis:

1. Chronic gastric ulcer.
2. Haemorrhage, Type II suddenly becoming Type III.

Comments.

1. Large fatal haemorrhage which was unpredictable. It is doubtful if a patient with so low a haemoglobin percentage, which had probably been present for weeks, could have survived an operation in the first forty-eight hours.

2. It is probable that the long delay in sending this patient to hospital and the prolonged anoxia associated with his anaemia contributed to his death.

Case 70. W.S. male, age 43.

One week ago suddenly became weak and vomited a chamberful of blood and passed a black motion. This was repeated last night.

On admission:

Very anaemic. Tongue dry. No evidence of shock.

Pulse 94. B.P. 110/50. Hb. 38%.

Treatment: Routine. Transfusion of three pints (for anaemia).

Progress:

On fourth day he suddenly became pale and weak and passed a reddish black stool; tongue dry and furred.

Pulse 120. B.P. 70/40.

Three pints of blood were given. Hb. was then 36%.

Fifth day. Three pints of blood given. Hb. 47%.

Seventh day. Two pints of blood given.

Eleventh day. Hb. 67%.

Eighteenth day. Hb. 81%.

Investigations:

Fractional test meal - Histamine fast achlorhydria.

X-ray report - no abnormality seen.

Gastroscopy - no abnormality seen.

Diagnosis:

1. Acute ulcer.
2. Haemorrhage, Type I.

Case 71. R.B. male, age 47.

Attack of haemorrhage today - dark red vomit with clots. Epigastric pain for one year, sometimes relieved by food and returning two hours later.

On admission: General condition good; not collapsed; no pallor.

Pulse 110. B.P. 130/80. Hb. 100%.

Treatment: Routine.

Progress:

On the second day he collapsed and vomited blood.

Pulse ? B.P. 75/40.

Two pints of blood were transfused and he improved after this.

On third day he vomited a large quantity of blood and became unconscious.

Pulse rose to 140 and became very thready in quality. B.P. 50/?.

Transfusion begun at once but he died a few minutes later.

Post mortem findings (Dr. T. Cooke)

Congested lung bases.

Patch coronary atheroma.

Stomach and intestine full of blood.

Large, deep, thickly fibrosed ulcer in first part of duodenum close to pylorus with large, open-mouthed artery in base.

Diagnosis:

1. Chronic duodenal ulcer.
2. Haemorrhage. Classified as mild on admission and as severe Type I on second day, later becoming Type III.

A sudden catastrophic haemorrhage on third day produced death almost at once. The death was unforeseen. If this case had occurred a little later in the series he would have been radiographed after his first recurrence or immediately a history of chronic ulcer had been obtained and operated upon before his fatal recurrence.

Case 79. W.C. male, age 72.

Vomited blood this evening and also yesterday. Has had much flatulence and upper abdominal pain brought on by food for past three years. Remissions have occurred but have never lasted for more than two weeks.

On admission:

Very anaemic. Pulse soft and rapid. Tongue dry and furred.

Pulse 110. B.P. 85/60. Hb. $40\frac{1}{2}$.

Treatment: Routine. Two pints of blood transfused.

Second day. Profuse haematemesis. Three pints of blood transfused.

Third day. Further haematemesis. Pulse 120. B.P. 80/50. Two and a half pints of blood given. Surgeon was now called in but refused to operate as "condition of patient was too precarious".

Fourth day. B.P. was 100/50. Pulse 95. Hb. 53%.

Fifth day. Had a recurrence of haematemesis and died half an hour later.

Post mortem (Dr. F.B. Smith)

Blanched, lean. Brain blanched. Early perihilar consolidation right lower lobe.

Capacious aorta, arch and trunk. Heart appears quite normal.

Stomach and ileum full of blood.

Chronic duodenal ulcer, 30 mms in diameter adjacent to pylorus; very deep and eroding into pancreas with a large artery perforated in base.

Liver quite normal. Small wasted adrenals.

Pale, athero-sclerotic kidneys. Normal prostate and bladder.

Diagnosis:

1. Chronic duodenal ulcer.

Comments.

Attacks of haemetemesis in this case occurred at fairly long intervals, but as each one was very severe, the clinical condition had undergone a definite deterioration, by the third day, in spite of transfusion. Surgical aid was sought. Absence of radiological evidence, absence of co-operative surgeon and death of patient regretted in retrospect.

Case 81. C.P. male, age 45.

Vomited a pint of thick blood about noon today and had subsequent melaena.

Epigastric pain 5-6 years, relieved by food and alkalies. Worse for last three months. Has had remissions.

On admission: Patient gravely ill and very shocked.

Pulse 130. B.P. 40/30(?)

Treatment: One pint of plasma given immediately, followed by blood.

Progress: Patient died within an hour of admission.

Post mortem findings.

Exanguinated, lean. Very marked, almost polypoidal lymphadenoid hyperplasia of tongue.

Chest. Gross emphysema with bullae of lungs; marked oedema of right lower lobe.

Abdomen. Blood in gastro-intestinal tract; 250 ccs with massive clot in stomach. Ulcer crater in posterior wall of stomach with pancreas as base. Source of haemorrhage not visible to the naked eye.

Spleen. A few scattered white foci up to 6 mms in diameter.

Liver. Indistinctive to naked eye but pattern not quite normal.

Kidneys. Appeared normal apart from pallor.

Blood stained peritoneal effusion. Considerable mass of pre-aortic lymph glands extending from coeliac axis to pelvic brim - discrete, white and moderately soft.

Microscopy of lymph glands and spleen. Reticulosis.

Diagnosis:

1. Chronic gastric ulcer.
2. Haemorrhage, Type I.
3. Reticulosis.

Comments.

Reticulosis probably contributed largely to the fatal issue and it is probable that it was responsible also for the haemorrhagic diathesis.

Case 86. G.M. male, age 42.

Had small haematemesis yesterday but, this morning vomited a large amount of blood and felt weak. Had a haematemesis two years ago. He had had epigastric pain for one and a half years and was radiographed one week ago by me and penetrating moveable ulcer on lesser curve was reported to his doctor.

On admission:

Severely shocked, very pale.

Pulse 135. B.P. 60/?. Hb. 60%.

Treatment: Routine. Two pints of blood transfused at once.

Progress:

Definite improvement observed on second day and blood pressure had risen to 110/60 mms Hg.

Third day. Haemoglobin 33%. Two more pints of blood transfused.

Fourth day. Another haematemesis - 2 pints.

Patient shocked and collapsed. Transfused - 2 pints.

A severe recurrence and his condition was deteriorating. Surgeon called in and was very willing to operate after he had seen films of large lesser curve ulcer.

Transfusion continued in theatre.

Partial gastrectomy performed.

Recovery progressive and uneventful.

Diagnosis:

1. Chronic gastric ulcer.
2. Haemorrhage. Type I - Type III.

Possession of previous radiographs persuaded surgeon to operate with confidence.

Case 87. W.S. male, age 37.

Collapsed on floor this morning and was violently sick. Vomit contained bright red blood. Has been vomiting thick brownish fluid for past few days.

History of pain related to food for 13 years. Remissions for months. Was in hospital a year ago with severe haematemesis.

On admission: Patient pale, cold and collapsed with dry tongue.

Pulse 112. B.P. 65/40. Hb. 22%.

Treatment: Routine. Blood transfusion - three pints.

Progress: Had a small haematemesis six hours after admission.

Second day. Somewhat improved. Pulse 116. B.P. 95/55.

Third day. Improvement maintained. Pulse 100. B.P. 106/60.

Was given small barium meal (4 ozs) at this point and large ulcer crater seen on posterior aspect of lesser curve.

Fifth day. Severe recurrence. Vomited about two pints of bright red blood.

Pulse 130. B.P. 70/40.

Surgeon called in at this point; he said he would operate as soon as systolic blood pressure had been raised to 100 mms Hg. (systolic).

Transfusion was begun and before going to theatre the blood pressure had been raised to 95/55.

Operation.

Right paramedian incision. Stomach which was very distended, opened through small incision and about two and a half pints of dark blood aspirated. Large ulcer on posterior wall of lesser curve was seen and a large artery in its base was bleeding freely. The artery was ligatured and the stomach and abdomen closed.

Seventh day. Mucous membranes are very good colour.

Patient much improved. B.P. 110/65.

96

Eighth day. Hb. 78%.

Ninth day. Severe haematemesis associated with shock and collapse.

Transfusion begun.

He continued to have frequent severe attacks of bleeding and died on eleventh day.

Post mortem. (Dr. F.B. Smith)

Lean and pale. Skull normal.

Chest. Small heart and lungs.

Abdomen. Enormous dilatation of very oedematous, thick walled stomach which contained 1 litre of dark brown, watery fluid with a large blood clot. Stomach lies against symphysis pubis.

Enormous excoriated lesser curve ulcer, 8 x 8 cms in area, in depth of which was a large clot pointing from lumen of a large artery.

Gall bladder thickened.

Great oedema of retro-serous tissue of upper abdomen.

Kidneys - very pale and surface slightly roughened and rather swollen.

Adrenals and pancreas - normal.

Ileum contains 10 ozs of fluid similar to that in stomach.

Colon contains many dark scybala.

Diagnosis:

1. Chronic gastric ulcer.
2. Haemorrhage, Type III.

Comments.

1. Surgeon should have been consulted sooner.
2. Ligature of bleeding artery only stopped the bleeding temporarily; it did not prevent recurrence.
3. Distension of stomach and oedema of wall due to prolonged secondary shock.

Case 88. H.B. male, age 62.

Had a large haematemesis this morning. Several years' history of indigestion becoming more acute recently. Liver and spleen impalpable.

On admission:

Moderately shocked, creamy pallor. Dry furred tongue.

Pulse 100. B.P. 85/60. Hb. 63%.

Treatment: Routine. Transfusion of two pints began at once.

Second day. Much improved and blood pressure had risen to 150/95. Hb. 40%.

Third day. Felt sick and faint, went pale and had copious melaena.

Pulse 110. B.P. 85/60.

Two more pints of blood transfused and was much improved.

Fourth day. Improvement maintained. Small barium meal given (6 oz).

Hamptons formula used but 2 ozs more water was added. This was given to determine whether the ulcer thought to be duodenal, was in the distal or proximal part of the cap. A large crater was readily seen on the lesser curve. It moved on respiration.

Fifth day. Another large melaena, patient shocked and collapsed.

Transfusion begun and surgeon agreed to operate as soon as systolic pressure recovered to 100 mms Hg. (systolic).

Taken down to theatre and transfusion continued.

Partial gastrectomy performed.

Recovery uneventful as in preceding case.

Diagnosis:

1. Chronic lesser curve ulcer.
2. Haemorrhage, Type III.

Case 89. J.S. age 59.

Last night vomited about a quart of blood and has vomited several small amounts since. Tongue furred but moist.

Abdomen moves with respiration; a little gaseous distension; no free fluid. Tenderness over duodenum but no guarding.

B.P. 165/90. Skin cold and clammy. P.R. 100 per min. Hb. 80%.

Has just again vomited a little brown fluid.

He began to vomit again and there was a swinging pyrexia.

Twenty-five days after his partial gastrectomy he developed tenderness, pain and fullness over his duodenum. The R. paramedian incision was re-opened and a small, foul smelling pocket of pus was found around duodenal stump with a small extension above liver. Pus and necrotic material evacuated and rubber drain put down.

Three days later bile began to discharge from drain evidently from a duodenal fistula.

Nine days later there was a well established fistula and the drainage was much less. Corrugated drain removed and sucker tube left in situ.

Eleven days later bile drainage by suction was discontinued and, eight days subsequently he was discharged home.

Diagnosis:

1. Chronic duodenal ulcer.
2. Haemorrhage, Type III.
3. Perforation.

Comments.

Simple ligature of bleeding point was again unsuccessful and bleeding was not permanently arrested until partial gastrectomy had been performed.

Case 92. A.N. female, age 67.

Had severe haematemesis today. History of pain after food for seventeen years.

On admission:

Not particularly shocked; moderate anaemia. Very obese. (21 stones)
Adiposis dolorosa. (Dercum's disease).

Pulse 110. B.P. 160/100. Hb. 42%.

Treatment: Routine.

Progress:

During succeeding three days she had repeated severe recurrences associated collapse and despite repeated transfusion died on fifth day.

Surgeon refused to operate because of obesity and poor condition of patient. No radiological examination carried out.

Post mortem. Refused.

Diagnosis:

1. Probable chronic gastric ulcer.
2. Haemorrhage, Type III.
3. Adiposis dolorosa.

Comments.

A case for surgical intervention although it was realized that this would have been technically difficult. Operation refused by surgeon as he considered her best chance was conservative treatment, on account of her general condition.

Case 120. W.N. female, age 34.

Vomited coffee ground fluid yesterday and this morning had severe haemorrhage with collapse and sweating. A year's history of indigestion with interval acute pain after food.

On admission: Pale and shocked; dry furred tongue.

Pulse 100. B.P. 80/50. Hb. 55%.

Cicatrices radiating from angles of mouth.

Treatment: Routine.

Two pints of blood transfused and condition much improved. Following morning six ounces of barium (modified Hampton meal) given and patient screened. Large ulcer crater seen fairly high up on lesser curve.

Two days later large haematemesis with collapse and pallor. Condition much deteriorated and giving rise to anxiety.

Pulse 120. B.P. 70/?. Hb. 40%.

Two more pints of blood transfused and condition improved again somewhat.

Further transfusion of two pints started and continued in theatre. High partial gastrectomy performed. Somewhat slow convalescence and ultimate recovery. Wassermann and Kahn reactions strongly positive.

Summary and Conclusions.

1. The literature has been surveyed and an analysis made of 120 cases of bleeding from the upper alimentary tract; in this group there were 10 fatalities due to haemorrhage (all chronic ulcer) - mortality 9%. Various aspects of the diagnosis and treatment of ulcer haemorrhage are considered with special reference to the massive type, of which there were 28 examples in the series.

2. The differentiation of ulcer from other causes of bleeding from the upper gastro-intestinal tract is discussed and attention is drawn to the possibility of missing a concomitant perforation in an ill, elderly patient who has had morphia.

Acute ulcer is defined; a relatively short history of pain, or its complete absence, was found to be the only reliable guide in distinguishing it from the chronic type.

3. A severe or massive haemorrhage is defined and the limitations of the criteria commonly employed in assessing severity discussed; those of Witts (excluding pulse and Blood Urea) were found reliable but the importance of the clinical impression is stressed. Recurrence was of paramount importance in prognosis, and the necessity for defining it further for clinical assessment, records and publication is pointed out.

Recurrence is divided into three types, depending on its severity, frequency, and the possibility of keeping pace with the blood loss by transfusion.

4. In accordance with the basic idea of the Meulengracht regime, the importance of adequate intake of fluids and electrolytes is stressed.

In severe cases these were given intravenously - three pints of normal saline followed by 4.1% glucose in one fifth normal saline. The advantages

5. Objections, often quoted by the older physicians and surgeons, to the use of liberal transfusions are refuted and the advantages in both conservative and surgical treatment, pointed out.

6. The use of Radiology in the stage of acute bleeding is discussed.

7. A critical analysis of the 10 fatal cases is made in retrospect. Some of these were preventable and the employment of surgery in cases of chronic ulcer with Type III haemorrhage, in the later part of the enquiry, was successful, partial gastrectomy being the operation of choice.

8. The shortcomings of the present method of admitting patients with ulcer haemorrhage to the medical ward and the benefits to be obtained by close co-operation of physician, surgeon and radiologist are outlined.

It is important, in a general provincial hospital, that the general surgeon, who may not have had extensive experience in this branch (the surgical registrar often deals with this emergency), should be a member of the team which is observing the case. This is especially so at the inception of such a team, before confidence and understanding have been established.

9. Four fatal cases were under fifty years of age, and post-mortem in three of these showed that the artery was encased in dense fibrous tissue; this is probably a more important factor than endarteritis in preventing retraction.

10. As a result of the enquiry a definite procedure resulted -

- (a) From the history and clinical examination on admission, cases were first divided into acute and chronic ulcer.
- (b) In cases which were not clearly acute ulcer, Radiology was employed during the emergency.
- (c) In the case proved to be chronic ulcer, type III recurrence was an absolute indication for immediate operation.

Appendix IISummary of case reports.

Key to abbreviations

Column 2

Column 3

Column 4

D.U. = duodenal ulcer

H. = Haematemesis

G.C. = general condition

G.U. = gastric ulcer

M. = Melaena

Hb. = haemoglobin

Chr. = chronic

Column 8

+ HCl = hyperchlorhydria

Acl = achlorhydria

Males.

Case No. and age.	Final Diagnosis	History	Clinical findings and progress.
1 (59)	Chr. D.U. Hypertension Atheroma.		See Appendix I for detailed notes.
2 (38)	Chr. G.U.	H. day of admission. Pain related to food 4 years	G.C. good. Pulse 72-74. B.P.136/80 Hb. 90%. Uninterrupted recovery.
3 (53)	Chr. G.U.		See Appendix I for detailed notes.
4 (40)	Chr. D.U.	H. & M. one hour ago. Pain after food, 4 yrs.	Collapsed. Skin cold and moist. Pulse soft, 84. B.P.90/60. Hb.70% B.P.(1 hr.later) 100/70 Hb.(2 days later 80%. B.P.(6 hrs later 120/70.
5 (36)	Chr. G.U.	H. a few hours ago - 2 pints.	Pt.cold but no other signs of shock Pulse 80. Hb.80% B.P.95/60. B.P. 8 hrs.later 100/60 24 " " 105/60 48 " " 110/60
6 (36)	Chr. G.U.		See Appendix I for detailed notes.
7 (42)	Acute ulcer	M.3 days ago. Vague indigestion 2 weeks. No pain.	G.C. very good. Tongue moist. Pulse 76. B.P. 125/75. Hb.85%
8 (48)	Chr. D.U.	H. day of admission. M. a few days ago. Pain related to food many years (1-5)	G.C. good. Pulse 80. B.P.130/80. Hb. 86%
9 (49)	Acute D.U.	H. four times in past 8 days. Dyspepsia 4 years. No pain.	G.C. good. Pulse 76. B.P. 135/85. Hb. 90%.
10 (47)	Acute G.U.	Severe H. producing coma.7 days ago. No history of pain or dyspepsia.	Pt. very blanched, otherwise good. Pulse 85. B.P. 115/80. Hb. 30% (Two pints for anaemia)
11 (29)	Banti's disease	Severe H. this morning.	Pt. collapsed at home, appeared moribund (report of own Dr.) On admission had recovered from shock. Pulse 124. B.P. 120/60. Hb. 63%. 2 days later had very profuse H. Died before transfusion could be given
12 (24)	Chr. D.U.	H. & M. one week ago, was pale and faint. Pain related to food, 6 yrs.	G.C. good apart from pallor. Pulse 88. B.P. 130/80. Hb. 38%. 21 days later Hb. 75%.

No. of recurrences.	Degree of haemorrhage.	Radiological findings	Fractional Test Meal.	Gastroscopy.	Remarks.	Blood transfusion (pints)
about 10	Severe	-	-	-	See Appendix I.	10
0	Mild	Positive	Sustained hyperchlorhyd.	-	-	-
	Severe	Positive	"	-	See Appendix I.	6
0	Mild	Positive	"	-	Shock in this case not oligæmic, probably psychological.	-
0	Mild	Positive	Histamine labile achlorhydria	-	-	-
9	Severe	R.hemithorax collapsed. Active.Subclav. focus L.side.	-	-	See Appendix I.	11
0	Mild	Negative	Climbing curve	No abnormality seen.	-	-
0	Mild	Positive	Normal	-	-	-
0	Mild	Positive	Normal	-	-	2
0	Mild	Negative	Sustained Hyperchlorhydria	Normal	Not admitted to hospital for 7 days after H. Dr.afraid to move him.	2
1	-	-	-	-	-	-
0	Mild	Positive	Sustained hyperchlorhydria	-	This pt.probably had small hæmorrhages which escaped notice.	-

Case No. and age.	Final Diagnosis	History	Clinical findings and progress.
13 (47)	Chr. D.U.	H. yesterday. History of pain related to food - 20 years. Previous haem. 4 yrs ago.	Condition good. Pulse 88. B.P. 140/90. Hb. 67%
14 (54)	Ac. ulcer (? alcoholic gastritis)	H. $\frac{1}{2}$ pint. Drinks 20 pints beer daily. Pain related to food, 10 years.	G.C. poor. "Muddy" look. Teeth black. Pulse 86. B.P. 150/90. Hb. 60%
15 (42)	Chr. D.U.	Collapsed yesterday. Vomited coffee grounds and clots. Pain related to food 7 yrs.	G.C. Good. Pulse 76. B.P. 120/80. Hb. 86%
16 (46)	Chr. G.U.		See Appendix I for detailed notes.
17 (44)	Anastomotic ulcer	H. yesterday. Gastro-enterostomy for D.U. 9 years ago. Pain recurred 4 years ago.	G.C. good. <u>No</u> shock. B.P. 120/70. No pallor. Hb. 100%.
18 (40)	Chr. D.U.	H. 1 pint today. Pain unrelated to food, began 10 yrs ago. Perforation 4 yrs ago then no pain until 2 yrs ago.	G.C. good. Pulse 80. B.P. 110/70. Hb. 72%
19 (41)	Acute ulcer	Large H. just before admission. <u>No</u> previous pain or dyspepsia.	G.C. poor. Tongue dry. Pulse 132. B.P. 90/50. Hb. 53%.
20 (50)	Chr. D.U.	5 days ago felt dizzy and later vomited dark clots. 2 days ago vomited bright red blood. Pain related to food 4 months.	G.C. good. Pulse 80. B.P. 115/70. Hb. 80%
21 (32)	Acute ulcer		See Appendix I for detailed notes.
22 (24)	Chr. G.U.	Yesterday severe H. 10 days ago H. 15 yrs ago gastro-enterostomy for D.U. <u>No</u> pain or dyspepsia since operation, but has had repeated melaena.	Pt. pale; otherwise well. Pulse 88. B.P. 115/76. Hb. 57%. 4 days later Hb. 67%
23 (36)	Chr. D.U.		See Appendix I for detailed notes.
24	Acute stomal	Yesterday suddenly became giddy. Today H. & M. with collapse. Gastro-enterostomy 26 yrs ago. No pain since operation.	G.C. good apart from pallor. Pulse 120. B.P. 125/65. Hb. 90%. Three days later - Hb. 80%. B.P. 130/60.

No. of recurrences.	Degree of haemorrhage.	Radiological findings	Fractional Test Meal	Gastroscopy	Remarks	Blood trans-fusion (pints)
0	Mild	Positive	Histamine fast ACl	-	-	-
0	Mild	Negative. No varices.	Normal	Patchy gastritis	-	-
0	Mild	Positive	Histamine fast ACl	-	-	-
0	Mild	Positive	Normal			
0	Mild	Positive	Normal			
0	Mild	Negative	Sustained + HCl	Negative	Low Hb. reading shortly after haem. probably due to low Hb. before. Had severe rheumatoid arthritis. Chronic invalid.	2
0	Mild	Positive	"	-	-	-
7	Severe	Negative	"	Hypertrophic gastritis.		
0	Mild	Negative	Normal	Ulver lesser curve, stoma normal.	-	-
0	Mild	Stomal spasm	Normal	-	-	-

Case No. and age.	Final Diagnosis	History	Clinical findings and progress.
25 (60)	Chr. peptic ulcer. Short oesophagus.	Pain p.c. 15 years ago. X-ray showed D.U. then. Last 3 years - indigestion and epigastric pain. Mod. Haematemesis yesterday.	G.C. good. No shock or collapse. Pulse 80. B.P. 110/70. Hb, 80%.
26 (77)	Chr. D.U. Perforation		See Appendix I for detailed notes.
27 (35)	Chr. D.U.		See Appendix I for detailed notes.
28 (53)	Probable Chr. D.U.		See Appendix I for detailed notes.
29 (33)	Acute ulcer		See Appendix I for detailed notes.
30 (64)	Acute ulcer	H. 1 pint just before admission. No history of indigestion or pain.	No pallor or signs of shock. Pulse 110. B.P. 130/80. Hb. 86% Two hours later pulse 110. Next day Hb. 70%
31 (55)	Chr. G.U.	H. today 1 pint. See case (14) same patient. Inter- mittent pain since last discharge.	G.C. good, better than on previous admission. Pulse 82. B.P. 145/80. Hb. 72%.
32 (38)	Chr. G.U.		See Appendix I for detailed notes.
33 (48)	Chr. D.U.	H. 1 hr. before admission. History of pain related to food 1 year.	G.C. good. Pulse 76. B.P. 125/80. Hb. 76%.
34 (30)	Stomal Ulcer.		See Appendix I for detailed notes.
35 (30)	Acute ulcer Atrophic gastritis.	H. just before admission. History of pain related to food - 6 months.	Condition good. Pulse 85. B.P. 130/80. Hb. 86%.
36 (34)	Chr. D.U.	H. yesterday and today. History of pain related to food - 8 yrs. Perforated 7 years ago.	G.C. good. Pulse 88. B.P. 160/95. Hb. 67%
37 (65)	Probable Chr. D.U.	H. 2 pints today. History of pain related to food - 1 yr.	Condition fair. Pulse 80. B.P. 160/95. Hb. 67%
38 (40)	Chr. D.U.		See Appendix I for detailed notes.

No. of recurrences.	Degree of haem.	Radiological findings	Fractional Test Meal	Gastroscopy	Remarks	Blood transfusion(pints)
0	Mild	Short oesoph. with ulcer. See section on Radiology Case I.	Sustained + HCl.	-	Haem. thought to be gastric by own Dr. who thought he had G.U. Also had rheumatoid arthritis.	-
0	Mild	-	-	-	Severity of case, due to perforation undiagnosed before death.	1
1	Severe	-	-	-		6
1	Severe	-	-	-	Trans. to E.M.S. hosp. before further investigations carried out.	2
8	Severe	-	-	-	Same case as 21. 5 months between admissions.	6
0	Mild	Negative	Normal	Normal	-	-
0	Mild	Positive	Sustained + HCl	-	No ulcer seen 1 yr ago by x-ray or gastroscopy.	-
0	Severe	Positive	Normal	-	-	-
0	Mild	Positive	Normal	-	-	-
0	Severe	Positive	Normal	-	-	-
0	Mild	Positive (gastritis)	Normal	-	-	-
0	Mild	Positive	Normal	-	-	-
0	Mild	-	-	-	-	-
8	Severe	-	-	-	-	12

Case No. and age	Final Diagnosis	History	Clinical findings and progress.
39 (55)	? acute ulcer	H. last night and this morning. History of pain related to food 2 months.	Condition good. Pulse 76 B.P. 120/70. Hb. 80% 3 days later Hb. 63% 4 days later Hb. 76%
40 (57)	Chr. G.U. Slight hypertension.	H. & M. 7 yrs ago. Pain related to food one year.	G.C. good. No pallor. Pulse 75. B.P. 150/100. Hb. 80% 2 days later Hb. 67%
41 (24)	Acute stomal ulcer.	H. this morning. Gastro-enterostomy for D.U. 4 yrs ago. No pain until 14 days ago.	G.C. good. Pulse 76. B.P. 100/70 Hb. 114%. Left hospital against advise 4 days later.
42 (49)	Chr. G.U.	H. 2 pints today. Epigastric pain related to food 30 yrs. Previous H. 23 years ago.	Pale, otherwise G.C. good. Pulse 96. B.P. 110/60. Hb. 29%. 2 pints blood given for anaemia. 5 days later Hb. 43% 13 days later Hb. 54%
43 (38)	Chr. D.U.	H. just before admission. Pain related to food 15 years.	Condition good. Pulse 82. B.P. 110/80. Hb. 77%.
44 (37)	Chr. D.U.	H. 2 pints yesterday. Pain related to food 14 years.	G.C. good apart from pallor. Pulse 80. B.P. 100/60. Hb. 50% 14 days later Hb. 54% 21 days later Hb. 58% 28 days later Hb. 70%
45 (47)	Chr. D.U.	H. 8 days ago. M. continuing since. Pain related to food 15 months.	Condition fair; moderate anaemia. Pulse 80. B.P. 145/85. Hb. 54% 7th day Hb. 47% (2 pints blood given) 8th day Hb. 49% -do- 17th day Hb. 72%
46 q (59)	Probable Stomal ulcer.	H. & M. yesterday. Gastro-enterostomy for gastric ulcer & pyloric stenosis 9 yrs ago. Free from pain until 2 yrs ago.	G.C. good. Pulse 76. B.P. 140/90. Hb. 89% 5 days later Hb. 88%
47 (44)	Stomal ulcer	H. this afternoon. Gastro-enterostomy for D. U. 12 yrs ago. Free from pain until 4 yrs ago.	Mod. pallor. Dry tongue. Otherwise G.C. good. Pulse 90. B.P. 120/65. Hb. 46%. Next day Hb. 43%. 2 pints blood given (for anaemia)
48 (47)	Acute ulcer	H. this morning. No history of pain or indigestion.	Condition very good. Pulse 75. B.P. 140/75. Hb. 110% Left hospital against advise.

No. of recurrences.	Degree of haem.	Radiological findings	Fractional Test Meal	Gastrosopy	Remarks	Blood trans-fusion(pints)
0	Mild	-	Normal	Normal	-	-
0	Mild	Positive	Sustained + HCl	-	-	-
0	Mild	-	-	-	-	-
0	Mild	Positive	Histamine fast A Cl	-	Initial low Hb. probably due to slow chronic bleed-ing. Slow response of marrow to iron therapy.	4
0	Mild	Positive	Sustained + HCl	-	-	-
0	Mild	Positive	Sustained + HCl	-	-	2
0	Mild	Positive	Sustained + HCl	-	-	4
0	Mild	Positive	Histamine fast A Cl	-	-	2
0	Mild	Positive	Histamine fast A Cl	Gastritis	-	-
0	Mild	-	-	-	-	-

Case No. and age.	Final Diagnosis	History	Clinical findings and progress.
49 (21)	Probable acute ulcer.	H. this morning. History of pain & dyspepsia 1 yr. Pain below and to right of umbilicus.	Condition good. Pulse 76. B.P. 117/75. Hb. 92%
50 (50)	Chr. D.U.		See Appendix I for detailed notes.
51 (51)	Acute ulcer.	H. yesterday with collapse. No past history of pain or dyspepsia.	G.C. fair. Moderate anaemia. Pulse 96. B.P. 105/80. Hb. 55% 10th day Hb. 65% Left hospital against advice (14th day).
52 (59)	Chr. D.U.	H. this morning. M. yester- day. Pain related to food 2 yrs.	Marked anaemia. Pulse 104. B.P. 105/90. Hb. 40%. 3rd day 2 pints blood given. 7th day Hb. 54%. 10th day Hb. 60%.
53 (48)	Stomal ulcer		See Appendix I for detailed notes.
54 (54)	Acute ulcer		See Appendix I for detailed notes.
55 (43)	Acute ulcer		See Appendix I for detailed notes.
56 (31)	Chr. D.U.	Severe M. yesterday. H. 3 weeks ago. Pain related to meals - 2 yrs.	Condition good. Pulse 76. B.P. 110/70. Hb. 87%
57 (61)	Chr. D.U.	H. 1 pint this morning & 3 days ago. Indigestion 4 weeks. No pain.	Marked pallor; not shocked. Pulse 100. B.P. 110/70. Hb. 37% 2nd day. One pint blood given. 4th day -do- 5th day Hb. 49%
58 (46)	Chr. D.U.	H. & M. last night. Today vomited brown, blood fluid. Pain related to food 9 yrs.	Patient very nervous. Mildly shocked. Pulse 96. B.P. 60/30% Hb. 72%. 1 hr. later B.P. 110/60% Next day Hb. 60%.
59 (60)	Chr. D.U.	H. & M. last night. Pain related to food 7 yrs. X-ray (6 yrs ago) D.U.	Rather pale. Pulse 80. B.P. 110/ Hb. 44%. 4th day Hb. 49% 5th day 2 pints blood given. 6th day Hb. 60%.
60 (56)	Chr. D.U.	H. last night. Pain related to food 5 yrs.	Moderate pallor. Slight shock. Pulse 100. B.P. 100/60. Hb. 55%.

No. of recurrences.	Degree of Haem.	Radiological findings	Fractional Test Meal	Gastroscopy.	Remarks.	Blood trans - fusion(pints)
0	Mild	Negative	Normal	-	-	-
	Severe	Positive	Normal	-		10
0	Mild	-	-	-	-	-
0	Mild	Positive	Normal	-	-	2
0	Severe	Negative	Histamine fast A Cl	-	See appendix I	6
4	Severe	Negative	Histamine fast A Cl	Atrophic gastritis	See appendix I	15
0	Severe	Negative	Normal	Normal	See appendix I	8
0	Mild	Positive	Normal	-	-	-
0	Mild	Positive	Normal	-	-	2
0	Mild	Positive	Histamine labile A Cl	-	Shock on admission probably psychological.	-
0	Mild	Positive	Sustained + HCl	-	-	2
0	Mild	Positive	Sustained + HCl	-	-	-

Case No. and age.	Final Diagnosis.	History.	Clinical findings and progress.
61	Acute ulcer	H. last night. Pain related to food - 5 months.	Mod. pallor. No shock. Pulse 78. B.P. 125/85. Hb. 80%. Slight attack of melaena each of next four days. 3rd day Hb. 53%. 17th day Hb. 70%.
62 (53)	Stomal Ulcer		See Appendix I for detailed notes.
63 (67)	Chr. G.U.		See Appendix I for detailed notes.
64 (51)	Acute ulcer	H. yesterday, about 2 pints. Pain related to food - 2 weeks.	Mod. pallor. Pulse 96. B.P. 95/60. Hb. 62%. Eight hours later B.P. 130/80. 5th day Hb. 48% - 1 pint of blood given. 21st day Hb. 96%.
65 (57)	Acute Ulcer (?cirrhosis)	H. & M. yesterday. No history of pain. Has drunk 6-7 pints daily for 40 years.	Obese. Telangiectasis. Slight enlargement of liver. Spleen impalpable. Pulse 76. B.P. 130/75. Hb. 57%.
66 (63)	Diverticulum of 2nd part of duodenum	H. this morning - 1 pint. Pain related to food 2 yrs.	Condition good. Pulse 68. B.P. 135/90. Hb. 120%.
67 (54)	Acute ulcer	H. today. No history of pain or dyspepsia.	Condition very good. Pulse 100. B.P. 135/80. Hb. 67%.
68 (58)	Acute ulcer (?cirrhosis)	Same case as 65. H. & M. this morning. No history of pain.	Obese. Telangiectasis. Liver two fingers breadth below costal margin. Pulse 80. B.P. 135/80. Hb. 67%.
69 (37)	Stomal ulcer	H. & M. today. Pain related to food 18 years. Gastro-enterostomy for D.U. 17 years ago. Pain no better. H. 6 years ago.	G.C. fairly good. Pulse 78. B.P. 120/80. Hb. 86%.
70 (43)	Acute ulcer		See Appendix I for detailed notes.
71 (47)	Chr. D.U.		See Appendix I for detailed notes.
72 (51)	Chr. D.U.	H. & M. yesterday. Pain 5 yrs. related to food.	Marked pallor. No signs of shock. Pulse 80. B.P. 95/60. Hb. 33%. 2nd day 2 pints of blood. 10th day Hb. 57%.
73 (64)	Chr. D.U.	H. 1 pint today. Pain related to food 1 yr. Perforation 15 yrs ago.	Moderate anaemia. Pulse 80. B.P. 140/90. Hb. 67%.

No. of recurrences.	Degree of Haem.	X-ray findings	Fractional Test Meal	Gastroscopy	Remarks	Blood trans- fusion (pints)
0	Mild	Negative	Climbing curve	Normal	-	-
Bleeding more or less continuous.	Severe	Negative	Normal	-	See Appendix I	8
12	Severe	Negative	Normal	-		7
0	Mild	Negative	Normal	-	-	1
0	Mild	Negative	Normal	Patch gastritis	History of alcoholism & clinical findings suggest cirrhosis.	-
0	Mild	Positive	Histamine fast ACl	-	-	-
0	Mild	Positive (gastritis)	-	Gastritis	-	-
0	Mild	Negative	-	Patchy gastritis	Observed for 2 years after discharge. No further bleeding or change in findings.	-
0	Mild	Positive	Histamine fast ACl	-	-	-
1	Severe	Negative	Histamine fast ACl.	-	See Appendix I	11
2	Severe	-	-	-	See Appendix I	2
0	Mild	Positive	Normal	-	-	2
0	Mild	Positive	High fast- ing acid	-	-	-

Case No. & age.	Final Diagnosis	History	Clinical findings and progress.
74 (39)	Chr. G.U.	H. & M. yesterday. Pain related to food 11 days, never before.	Moderate anaemia. Pulse 90. B.P. 110/70. 2nd day 2 pints of blood given. 4th day Hb. 44%. 2 pints blood given. 10th day Hb. 59%. 17th day Hb. 57%. Two pints of blood given. 22nd day Hb. 76%.
75 (40)	Chr. G.U.	H. & M. 5 days ago. Repeated 2 days later. Pain related to food - 6 years.	G.C. very good. Pulse 80. B.P. 145/90. Hb. 93%.
76 (51)	Stomal ulcer	H. this afternoon and on and off for past month. H. 8 & 6 years ago. Gastro-enterostomy 12 yrs ago. No pain since.	Condition fair. Moderate anaemia. Pulse 84. B.P. 125/75. Hb. 51%. 3rd day - slight melaena - no signs shock. 4 day Hb. 44%. 2 pints blood given. 8th day Hb. 64%, after further 2 pints on 6th day.
77 (76)	Acute ulcer	H. this morning & M. this past week and also four months ago. No pain or dyspepsia.	Marked anaemia. Pulse 124. B.P. 135/70. Hb. 35%. 2nd day 2 pints of blood transfused. 6th day -do- Hb. 55% 15th day -do- Hb. 60% 18th day -do- Hb. 78%
78 (27)	Probably Chr. D.U.	H. this evening. Pain related to food 1 yr.	Moderate anaemia. Condition otherwise good. Pulse 76. B.P. 120/70. Hb. 59%. Went out against advice on 6th day.
79 (72)	Chr. D.U.	See Appendix I for detailed notes.	
80 (46)	Probably Chr. D.U.	H. today. Pain related to food - 18 yrs, Perforation 20 yrs ago.	G.C. good apart from slight anaemia. Pulse 110. B.P. 130/90. Hb. 62%. Went out against advice on 5th day.
81 (45)	Chr. G.U. also reticulosis	See Appendix I for detailed notes.	
82 (53)	Ac. ulcer Thyrotoxicosis	H. today. M. 2 weeks ago. No pain or indigestion.	Moderate anaemia. Signs of thyrotoxicosis present. Pulse 98. B.P. 190/90. Hb. 58%.
83 (64)	Probable ac. ulcer. Essen-tial hyper-tension.	H. yesterday and during previous three days. No history of pain or dyspepsia.	G.C. good. Pulse 60. B.P. 220/95. Hb. 90%. Blood urea 35 mgms %.
84 (34)	Chr. D.U.	H. yesterday. A few attacks of M. during past week. Pain related to food 1 yr.	Mod. anaemia. No shock. Pulse 88. B.P. 120/90. Hb. 52%.

No. of recurrences.	Degree of Haem.	X-ray findings	Fractional Test Meal	Gastroscopy	Remarks	Blood transfusion (pints)
0	Mild	Positive (large ulcer lesser curve)	+ HCl	-	Note short history of pain.	10
0	Mild	Positive	Histamine fast ACl	-	-	-
0	Mild	Positive	Normal	-	-	4
0	Mild	Negative	Normal	Normal	-	6
0	Mild	-	-	-	-	-
3	Severe	-	-	-	See Appendix I	10
0	Mild	-	-	-	-	-
0	Severe	-	-	-	See Appendix I	1
0	Mild	Negative	Normal	Normal	-	-
0	Mild	Negative	Sustained + HCl	Normal	-	-
0	Mild	Positive	Normal	-	-	-

Case No. & age.	Final Diagnosis	History	Clinical findings and progress.
85 (31)	Chr. D.U.	A few attacks H. in past week. Pain related to food 5 years.	Condition good. Pulse 85. B.P. 130/80. Hb. 71%.
86 (42)	Chr. G.U.		See Appendix I for detailed notes.
87 (37)	Chr. G.U.		See Appendix I for detailed notes.
88 (62)	Chr. G.U.		See Appendix I for detailed notes.
89 (59)	Chr. D.U.		See Appendix I for detailed notes.
90 (56)	Chr. D.U.	Melaena today. 15 years history. Perforated 4 yrs ago. Better for 3 yrs but worse again for 1 yr, especially for last 2 months. Previous H. & M. 8 mths ago.	Pulse 78. B.P. 130/90. Hb. 78%. General condition good. Radiographed on next day and operated on 2 days after that.

No. of recurrences.	Degree of Haem.	X-ray findings	Fractional Test Meal	Gastroscopy	Remarks	Blood trans- fusion (pints)
0	Mild	Positive	Normal	-	-	-
2	Severe	Positive	-	-	See Appendix I	6
4	Severe	Positive	-	-	See Appendix I	12
2	Severe	Positive	-	-	See Appendix I	6
1	Severe	-	-	-	See Appendix I	4
0	Mild	Duodenal cap never seen. Organic pyloric stenosis with 24 hrs residue.	High night secretion.	-	Operation. $\frac{3}{4}$ gastrectomy. Bancrofts procedure. Duodenum grossly deformed by large D.U. adherent. Duodenum left in situ. Stenosis and previous history of haemorrhage decided operation here on Type I.	-

Case No. & age.	Final Diagnosis	History	Clinical findings and progress
91	Acute ulcer	M. a few days ago. Continued 2 day. Attacks 4 & 6 yrs ago. Pain related to food from 30 yrs ago until 12 yrs ago. None since.	Moderate anaemia. Pulse 80. B.P. 135/80. Hb. 43%.
92 (67)	Probable Chr. G.U. Dercum's disease.	See Appendix I for detailed notes.	
93 (58)	Chr. nephritis with hypertension. Sup. gastritis	H. 2 days ago followed by M. Pain related to food and also known hypertensive for 5 yrs.	Mild anaemia. Pulse 68. B.P. 105/100. Hb. 80%. Fundi. arteriosclerotic retinitis. Urine albumen - tract. Sp.Gr. 1010 (constant). Blood urea 35 mgms per cent.
94 (56)	Acute ulcer	H. & M. yesterday. No history of pain or dyspepsia.	Moderate anaemia. No signs of shock. Pulse 80. B.P. 120/75. Hb. 62%. 21 days later Hb. 96%.
95 (41)	Acute ulcer	H. last night. Pain related to food - 3 weeks.	G.C. good. Pulse 78. B.P. 130/70. Hb. 96%.
96 (27)	Chr. D.U. Essential Hypertension	H. today. Pain related to food - 9 yrs. Perforated D.U. 9 yrs ago.	No pallor or shock. Pulse 65. B.P. 250/130. Hb. 70%. Urine - no albumen. Sp. Gr. 1016. Blood urea 35 mgms per cent.
97 (63)	Portal cirrhosis	H. 3 pints this morning. Similar attacks 9 & 2 months ago.	Marked anaemia and oedema. Liver firm and two fingers breadths below costal margin. Spleen enlarged. Pulse 84. B.P. 120/50. Hb. 25%. Two pints of blood given immediately. Pulse rate continued to rise. Another large haem. 6 hrs later, further transfusion started but patient died.
98 (26)	Chr. D.U.	H. this morning and afternoon. Pain for 1 year.	G.C. fairly good. Pulse 108. B.P. 110/80. Hb. 76%.
99 (80)	Stomal ulcer	H. yesterday and today. Gastro-enterostomy for duodenal and pyloric stenosis 5 years ago.	Moderate anaemia. No signs of shock. Pulse 104. B.P. 100/60. Hb. 65%. 6 days later Hb. 43% 2 pints blood 8 days later H. with signs of shock. Pulse 87. B.P. 100/50. Hb. 53%.
100 (23)	Acute ulcer	H. & M. today and yesterday. No history of pain or indigestion.	Moderate pallor. No signs of shock. Pulse 87. B.P. 100/50. Hb. 53%.
101 (35)	Acute ulcer	H. today. Indigestion for past few days.	Moderate pallor. No signs of shock. Pulse 84. B.P. 110/80. Hb. 91%.

No. of recurrences	Degree of Haem.	X-ray findings	Fractional Test Meal	Gastroscopy	Remarks	Blood trans- fusion(pints)
0	Mild	Negative	+ HCl	Normal	Died 4 years later from cerebral haem. Had been under observation 1 yr. previous to this. B.P. being 190/140 during whole period.	-
8	Severe	-	-	-	See Appendix i	8
0	Mild	Negative	Normal	Superficial gastritis	Clinical & urinary findings diagnostic of chronic nephritis of which gastritis is a common feature.	-
0	Mild	Negative	Normal	Normal	-	-
0	Mild	Negative	Normal	Patchy atrophic gastritis	-	-
0	Mild	Positive	Negative	Sustained + HCl	-	-
0	-	-	-	-	-	2
0	Mild	Negative	Normal	Normal	-	-
0		Positive	Normal	-	-	4
0	Mild	Negative	Normal	Normal	-	-
0	Mild	Negative	Normal	Normal	-	-

Case No. & age.	Final Diagnosis	History	Clinical findings and progress	No re
102 (62)	Probable stomal ulcer	H. & M. 3 days ago. Gastro-enterostomy 14 yrs ago for pyloric stenosis. H. 3 yrs ago. <u>No</u> pain.	G.C. very good. Pulse 76. B.P. 140/90. Hb. 95%.	
103 (35)	Acute ulcer	H. this morning and this evening. Pain related to food 2 weeks.	Rather shocked. Skin cold & clammy. Pallor. Pulse 120. B.P. 100/50. Hb. 29%. 3 pints blood given. 2nd day Hb. 38%. 12th day Hb. 72%.	
104 (22)	Banti's disease	H. last night. Splenectomy 7 yrs ago. Five other attacks of H. in past 10 years.	G.C. good. Liver impalpable. No visible venous distension on abdominal wall. Pulse 75. B.P. 126/90. Hb. 53%. 6th day Hb. 57%. 2 pints of blood given. 10th day Hb. 76%.	
105 (50)	Acute ulcer (aspirin gastritis)	H. last night. No history of pain related to food or dyspepsia. Has taken 8 aspirins daily for past 3 wks.	No signs of shock. No anaemia. Pulse 80. B.P. 165/100. Hb. 90%.	
106 (30)	Chr. G.U.	H. & M. today. Pain related to food 3-4 weeks.	G.C. good. Marked pallor. No shock. Pulse 68. B.P. 125/75. Hb. 40%. 2 pints of blood given. 6th day Hb. 49%.	
107 (40)	Chr. D.U.	H. today. Pain related to food 5-6 years.	Moderate pallor. No shock. No anaemia. Pulse 80. B.P. 165/100. Hb. 90%.	
108 (37)	Chr. D.U.	H. & M. 2 weeks ago. Also $1\frac{1}{2}$ & 4 years ago. Pain related to food $4\frac{1}{2}$ years.	Marked pallor. Pulse 82. B.P. 115/75. Hb. 42%.	
109 (63)	Chr. G.U. Essential Hypertension. Thyrotoxicosis.	H. about $1\frac{1}{2}$ pints today. M. for past few days. Upper abdominal pain related to food 6 weeks.	Thyrototoxic signs. Condition otherwise good. Pulse 86. B.P. 200/100. Hb. 77%.	
110 (61)	Diaphragmatic hernia of stomach.	H. 3 & 2 days ago. Indigestion 7 years.	Marked anaemia. No shock. Pulse 78. B.P. 130/90. Hb. 45%.	
111 (45)	Chr. D.U.	H. yesterday. H. & M. 6 weeks ago. Pain related to food 1 yr.	Moderate anaemia. Pulse 96. B.P. 130/90. Hb. 72%.	
112 (59)	Portal cirrhosis	H. last night. 2 previous attacks of H. & M. Flatulence and epigastric discomfort.	Condition fair. Moderate anaemia. Abdomen very distended ascites ++ Dilated superficial veins. Spleen and liver enlarged. Pulse 112. B.P. 110/75. Hb. 53%. 2 pints of blood given. 15th day Hb. 57%. 21st day Hb. 75%.	

No. of recurrences.	Degree of Haem.	X-ray findings	Fractional Test Meal	Gastroscopy	Remarks	Blood trans- fusion (pints)
0	Mild	Negative	+ HCl	Not done	-	-
0	Severe	Negative	Normal	Normal	-	3
0	Mild	Negative	Normal	-	-	2
0	Mild	Negative	Normal	Day after admission haemorrhagic areas in upper part of stomach near lesser curve.	-	-
0	Mild	Positive	Normal	-	-	2
0	Mild	Positive	Normal	-	-	-
0	Mild	Negative	+ HCl	Pyloric antrum drawn up & to right suggest- ing D.U.	-	-
0	Mild	Positive	Normal	-	-	-
0	Mild	Positive	+ HCl	-	See section on Radiology (case 2.)	-
0	Mild	Positive	Normal	-	-	-
		Negative		-	-	-

Case No. & age.	Final Diagnosis	History	Clinical findings and progress.
113 (27)	Acute ulcer	H. this evening 2 pints. Flatulence past 6 days. No history of pain.	Moderate anaemia. No shock. Pulse 120. B.P. 135/80. Hb. 56%. 10th day Hb. 76%.
114 (49)	Acute ulcer	H. this evening 2 pints. Flatulence past 6 days. No history of pain.	Moderate haemorrhage. No signs of shock. Pulse 120. B.P. 135/80. Hb. 56%. 10th day Hb. 76%.
115 (65)	Chr. G.U. ? early cirrhosis	H. 2 nights ago. Abdominal distension for past 3 years. No pain. Heavy whisky drinker.	Abdominal distension moderate. Liver and spleen impalpable. Very anaemic and wasted. Pulse 96. B.P. 110/60. Hb. 25%. 2 pints of blood transfused. 6th day 2 pints of blood transfused. Hb. then 51%. 10th day 2 pints blood transfused. Hb. then 79%.
116 (29)	Chr. D.U.	H. yesterday and today. Pain related to food 2-3 months.	Moderate pallor. No signs of shock. Pulse 120. B.P. 115/75. Hb. 40%. 2nd day 2 pints of blood for anaemia 6th day -do- Hb. 65%.
117 (35)	Pyorrhoea alveolaris	H. a few times during past week. Abdominal pain unrelated to food - 6 months.	Patient nervous and anxious. Pyorrhoea of gums which bleed easily. Abdomen hypersensitive to palpation. No pallor or shock. Pulse 82. B.P. 130/90. Hb. 98%.
118 (25)	Acute ulcer	H. this evening & 3 and 1 days ago. Similar attack 5 yrs ago. No history of pain related to food or dyspepsia.	Moderate anaemia. No signs of shock. Pulse 120. B.P. 125/80. Hb. 41%. 10th day Hb. 46%. 2 pints blood given. 11th day Hb. 52%. 32nd day Hb. 92%.
119 (67)	Para-oesophag. hernia.	H. this morning. History of indigestion, pain in chest and down arms 4 yrs. History suggested angina also. Patient obese & of sthenic type.	No anaemia. No shock. Pulse 80. B.P. 130/100. Hb. 78%.
120 (34)	Chr. G.U.		See Appendix I for detailed notes.

No. of recurrences	Degree of Haem.	X-ray findings	Fractional Test Meal	Gastroscopy	Remarks	Blood trans-fusion (pints)
0	Mild	Negative	Normal	Normal	-	-
0	Mild	Negative	Normal	Normal	-	-
0	Mild	Positive	+ HCl	-	-	6
0	Mild	Positive	Histamine labile ACl	-	-	4
0	-	Negative	Normal	Normal	Blood vomit due to blood swallowed from sucking of gums.	-
0	Mild	Negative	Normal	Normal	-	2
0	Mild	Positive	Normal	-	See section on oesophageal hernia. (in Radiology)	0
1	Severe	Positive	-	-	See Appendix I.	6

REFERENCES.

1. Aird, J. (1948) Peptic ulcer haemorrhage, and its treatment.
B.M.A. Lecture, Preston.
2. Abercrombie, J. (1828). Diseases of the stomach. 13-46
Edinburgh.
3. Aitken, R.S., (1934) The treatment of profuse bleeding from
Stomach and Duodenum.
Lancet 1,839.
4. Åkerlund, A. (1926) Hernia diaphragmatica hiatus oesophagei vom
anatomischen und roentgenologischen gesichtsprunkt
Acta Rad. VI.3.
5. Allen, A.W. (1933) Bleeding duodenal ulcer.
New Eng. Med. J. 208.237.
6. Allison, F.R., Johnstone A.S., & Royce, J.B. (1943).
Short oesophagus with peptic ulceration.
J. Thoracic Surg. xii.432.
7. Alvarez, W.C. (1949) The discovery of a circulatory shunt in the
gastric wall which may explain the sudden
formation of peptic ulcers.
Brit. J. Rad. xxii.61.
8. Arnott, G.M., & Young, A. (1942)
Post-operative administration of fluids
to children.
Lancet 1.523.
9. Ashwell, H., (1836) Chlorosis and its complications.
Guys Hosp. Rep. i.529.
10. Baillie, M. (1793) Morbid Anatomy of some of the most important
parts of the Human Body. 140.
London.
11. Balfour, D.G. (1932) Surgical treatment of haemorrhage from duodenal ulcer.
J. Amer. Surg. 96.981.
12. Bannen, J.E. (1945) Investigation of free gas in the peritoneal cavity.
Brit. J. of Rad. 216.390.
13. Barclay, A.E. & Bentley F.H. (1949)
The vascularization of the Human Stomach.
Brit. J. Rad. xxii.61.
14. Bennett, T.J., Dow, J., Lander, F.P.L., & Wright, S. (1938)
Severe haemorrhage from stomach and duodenum.
Lancet 2.651.
15. Berg, H.H. (1926) Die Direkten Röntgensymptome des ulcers duodene
und ihre klinische bedeutung.
Ergebn. d. Med. Strahlenforsch. 2.249.
16. Black, D.A.K., (1942) Critical review. Azotaemia in gastro-duodenal
haemorrhage.
Quar. J. Med. 42.47.

17. Blalock, A. (1936) Shock and Haemorrhage.
Bull, New Yk. Acad. Med. 12.610.
18. Bonetus, T. (1700) Sepulchretum sive Anatomia Pratica.
Geneva. Lib.iii.Sec.xxi.observ.XXV.
19. Bohrer, J. (1941) Massive gastric haemorrhage with special reference
to peptic ulcer.
Trans.Amer.Surg.Ass. 59.296.
20. Bremwell, Byrom. (1901). Statistics of gastric ulcer with special reference
to gastric haemorrhage, its frequency and fatality.
Lancet 1.688.
21. Brinton, W. (1857) Ulcer of the Stomach. 95. 206.
London.
22. Browne E.G. (1921) Arabian Medicine III, 74.
23. Bucquoy, S. (1887) Étude clinique sur l'ulcère simple du duodenum.
Arch.Gen.de Med. 1.398.526.691.
24. Bulmer, E. (1932) Mortality from Haematemesis.
25. Celsus. (1478) De Medicina (A Stomachys doloribus) Florence. Leaf 68B
26. Chadwick, J. & Mann, W.N., (1950)
The Medical works of Hippocrates. 36.160.166.
27. Chiesman, W.E. (1932) Mortality of severe haemorrhage from peptic ulcers.
Lancet. 2.722.
28. Christiansen, T. (1935). On massive haemorrhage in peptic ulcer.
Acta.Med.Scand. 84.374.
29. Conybeare, J.J. (1935) Prognosis of gastric and duodenal ulcer.
Lancet 2.1017.
30. Cooper, A. (1827) The Lectures of Sir Astley Cooper, Bart.F.R.S.
on Principles and Practice of Surgery.
London.
31. Craig, J.D. (1948) The changing nature of gastric and duodenal
ulceration.
Post.Grad.Med.J. XXIV. 593.
32. Crohn, B.B. & Lerner, H.H. (1939).
Gross haemorrhage as a complication of peptic ulcer.
Amer.J.Digest.Dis. 6.15.
33. Crooke, A.C., & Morris, C.J.D. (1942)
Determination of Plasma Volume by Evans Blue method.
J.Physiol. 101. 217.
34. Cullinan, E.R., & Price R.K. (1932)
Haemorrhage following peptic ulcer;
prognosis and treatment.
Barts.Hosp.Rep.65.185.

35. Curling, T.B. (1842) On acute ulceration of the Duodenum in cases of burn.
Med.Chir.Trans. 25.260.
36. Curveilhier, J. (1835) Anatomie Pathologique du corps Humain à Paris. 1829 - 1835.
37. Donatus, M. (1586) De Medica Historia Mirabili.
Lib.IV.Cap.iii. 196.
38. Dunlop, D.M. (1947) B.M.A. Lecture, Preston.
39. Ebell, B. (1937) The Ebers Papyrus.
London. (Ox.Univ.Press).
40. Encyclopædia Britt. (14th Edition). ii.919.
41. Finsterer, H. (1939) Surg. Treatment of acute profuse gastric hæmorrhage.
J.Surg.Gyn.& Obstetr. 69.221.
42. Guthrie, D. (1946) A History of Medicine. 77.122.
43. Hempton, A.O. (1937) Safe method for Rontgen demonstration of bleeding duodenal ulcer.
Amer.J.Rontgen. 38.565.
44. Harrington, S.W. (1940) Diagnosis and treatment of various types of diaphragmatic hernia.
Amer.J.Surg. N.S. 50.381.
45. Hodgkin, T. (1840) Lectures on the Morbid Anatomy of the Serous and Mucous Membranes. ii.371.
46. Hurst, A.F. & Stewart, J. (1929) Gastric & Duodenal Ulcer.
London.
47. Hurst, A.F. & Ryle, J.A. (1937) Incidence, mortality and treatment of hæmorrhage in gastric and duodenal ulcer.
Lancet. i.1.
48. Ivy, A.C. (1920) Studies on gastric and duodenal ulcer.
J.Amer.Med.Ass. 75.1540.
49. Johnstone, A.S. (1943) Peptic ulceration of the oesophagus with partial thoracic stomach.
Brit.J.Rad. XVI. 192.357.
50. Jones, F.Avery, (1939a). Transfusion for Hæmatemesis and Melaena: observations on continuous drip transfusion.
Brit.Med.J. 1.915.
51. Jones, F.Avery, (1939b) Hæmatemesis and Melaena. Observations on salt and water requirements.
Brit.Med.J. 2.332.
52. Jones, F.Avery, (1947) Hæmatemesis and Melaena.
Brit.Med.J. 2.441.

53. Lewis, T. (1937) Diseases of the Heart. (London).
54. MacQuaide, D.H.G. & Mollison, P.L. (1940)
Treatment of anaemia by transfusion of
concentrated suspensions of red cells.
Brit. Med. J. 2.555.
55. Marriott, H.L. & Kekwick, H. (1935)
Continuous drip transfusion with records of
very large transfusions.
Lancet. 1.197.
56. McCance, R.A. (1936) Medical problems in mineral metabolism.
Lancet 1.823.
57. McMichael, J. (1942) Circulatory collapse and wound shock.
Edin. Post. Grad. Lect. 2.271.
58. Meulengracht, E. (1935) Treatment of Haematemesis and Melaena with food.
Lancet. 2.1220.
59. Miller, Glen, F., & Doub. Howard P. (1949)
Ulcer associated with diaphragmatic hernia.
Amer. J. Roentgen. 62.3.
60. Morley, J. (1949) Resectable carcinoma of the stomach.
Proc. Royal Soc. Med. XLii. 659.
61. Moynihan, B.G. (1905) Surgical treatment of non-malignant disease
of stomach.
London.
62. Needham, C.D. & McConachie, J.A. (1950-
Haematemesis and Melaena.
Brit. Med. J. 4671.133 & (b)
Editorial 4671.153.
63. Osler, W., (1892) Principles and Practice of Medicine.
London.
64. Papworth, M.H. & Lontit, J.F. (1943)
Gastro-duodenal haemorrhage treated medically
with enthusiastic blood transfusions.
Lancet. 2.469.
65. Portnoy, B. & Wilkinson, J.F. (1936)
The intra-dermal test for Vitamin C deficiency.
Brit. Med. J. 1.328.
66. Rawlinson, C. (1727) A preternatural perforation in the upper part
of the stomach.
Phil. Trans. Roy. Soc. Vol. vii. Part 111
507.
67. Robson, M. (1960) The Surgery of the Stomach.
Lancet. 1.683.
68. Samuel, E. (1948) Modern Trends in diagnostic Radiology.
London.

69. Sharpe, S. (1782) A Treatise on the Operations of Surgery.
London.
70. Sharpey-Schafer, E.P. (1944) Cardiac output in severe anaemia.
J.Chir.Sci. 5.125.
71. Tanner N.C. (1944) A critique of gastroscopy.
Brit.Med.J. 2.849.
72. Tanner, N.C. (1950) The treatment of haematemesis and Melaena.
Post. Grad. Med. J. 2.849.
73. Taylor Gordon G. (1938) The problem of bleeding peptic ulcer.
Brit. J. Surg. 25.403.
74. Templeton, F.E. (1944) X-ray examination of the Stomach.
Univ. Chic.
75. Tidy, H.L. Course of Death rate from peptic ulcer in
Great Britain.
Brit. J. Surg. 25.403.
76. Travers, B. (1817) On the Rupture of the Stomach from Ulceration.
Additional observations.
Med. Chir. Trans. viii. 232.
77. Trueta, J. Barclay, A.E., Daniel, P.M., Prichard, M.M.L. (1947)
Studies of the Renal Circulation.
Oxford.
78. Twining, E.W. (1933) Chronic hypertrophic stenosis of the
pylorus in adults.
Brit. J. Rad. ii. 644.
79. Vinson Porter, P. (1947) Diseases of the Oesophagus.
Illinois.
80. Wallace, J., & Sharpey-Schafer, E.P. (1941)
Blood changes following controlled
haemorrhage in man.
Lancet 2.395.
81. White, W.H. (1901) Are not patients said to be afflicted with
gastric ulcer really suffering from a
different disease?
Lancet. 1.1819.
82. Wilson, A.T.M. (1938) The psychogenic factor in haematemesis.
J. Ment. Sci. 84.1087.
83. Wilson, A.T.M. (1939) Psychological observations on haematemesis.
Brit. J. Med. Psychol. 18.112.
84. Witts, L.J. (1937) Haematemesis and Melaena.
Brit. Med. J. 1.847.
85. Wolf, G. (1928) Die Eikennung von Oesophagus varizen im Röntgenbilde.
Fortschi a.d. get.d. Rontgenstrahlen.
37.890.
86. Wolf, S., & Woolf, H.G. (1944) Human Gastric function.
Oxf. Univ. Press. 161.