PLASMA PROTEINS IN THE ASSESSMENT OF HAEMATEMESIS

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Of medical emergencies, none is more dramatic than severe upper alimentary haemorrhage and in none is more satisfaction to be gained from successful management and treatment. Yet few conditions call for more careful assessment or more discrimination in the application of the principles of therapy than massive haematemesis or haemorrhage which has persisted for days. Nor is the problem a small one and that it is one which is likely to become more frequent and more imperative in the future may be seen from a consideration of the changes which have been taking place in recent years in the nature of gastric and duodenal ulcer.

Tidy[1] has shown, from analysis of the Registrar General's mortality figures, that there has been a rapid increase in the incidence of peptic ulcer and especially duodenal ulcer, in men over the age of 40 yrs. since 1920. The increase has been progressive, but would appear to be levelling off in recent years. In males over 40 yrs. the increase in gastric ulcer between 1921 and 1931 was almost 90% and in duodenal ulcer 100%. Of particular interest, is that in Scotland as/

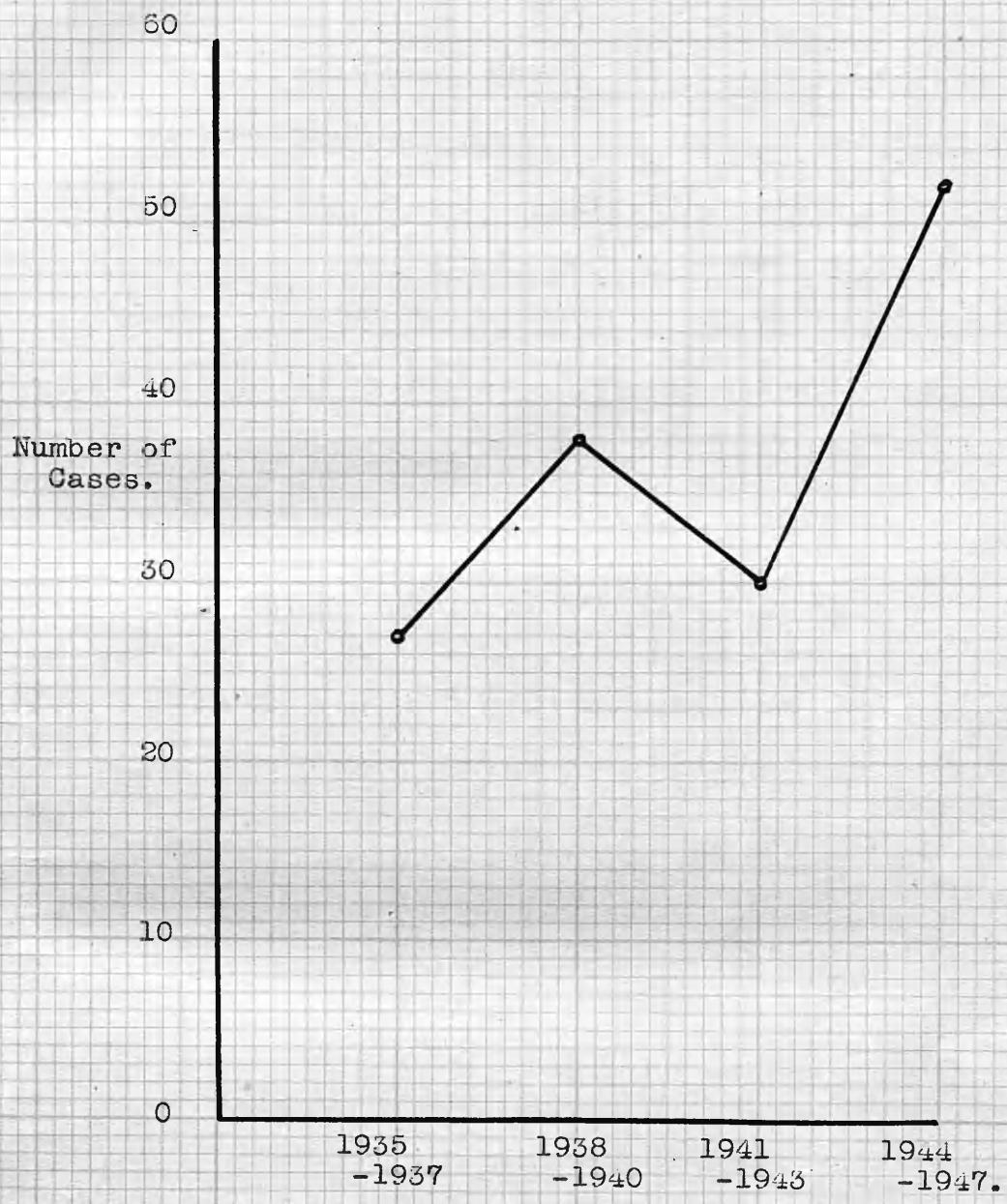
U.K.

as opposed to the rest of the country, the increase in gastric ulcer continued right up to 1938. The same trend is reflected in the increase of perforations in Glasgow reported by Illingworth, Scott and Jamieson[2]. The peak in this case came in 1940. Figures published by Avery Jones[3] for haematemesis over the period 1915 - 45 at the Central Middlesex Hospital are in agreement. The percentage of male admissions for haematemesis, who were over 40 yrs. of age, rose from 16% in 1915 to 50% in 1945.

In view of these facts one would expect that the actual incidence of haematemesis and melaena would be on the increase and an examination of the number of admissions over the past 14 yrs. to a medical unit in the Glasgow Royal Infirmary does bear this out. Considering only the cases of chronic ulcer of both sexes, and grouping the figures into 3 yr. periods in order to obtain numbers large enough to be significant, the following chart may be prepared.

CHART No 1.

ADMISSIONS FOR HAEMATEMESIS AND MELAENA.
1935-1947.



The importance of these figures lies in the fact that the increase is in patients in the older age groups because these groups, as might be expected, carry a much more serious risk than do the younger patients. In the same period, there were 146 cases of bleeding from chronic peptic ulcer. The age incidence and mortality rates are shown in the following table from which the very serious effects of advancing years on mortality can clearly be seen.

TABLE NO. I.

Mortality Rates in Chronic Peptic Ulcer
146 Cases (1935-47)

Age Yrs	Gastric Ulcer ♂		Duodenal Ulcer ♂		Gastric Ulcer ♀		Duodenal Ulcer ♀		Totals		%age Mortality
	No. Cases	Deaths	No. Cases	Deaths	No. Cases	Deaths	No. Cases	Deaths	Cases	Deaths	
21-30	4	0	14	0	2	0	3	0	23	0	0%
31-40	4	0	25	1	1	0	2	1	32	2	6%
41-50	11	2	23	2	5	1	3	0	42	5	12%
51-60	4	1	21	6	5	0	2	0	32	7	22.5%
61-70	3	0	9	1	3	1	1	0	16	2	(12.5%)
71-80	0	0	1	1	0	0	0	0	1	1	(100%)

The figures in the two oldest age groups are,

of/

of course, too small for reliability, but the seriousness of upper alimentary bleeding in the second half of life is amply illustrated and the conclusion justified that haematemesis and melaena will become even more serious problems in the future.

In assessing the prognosis of haematemesis or melaena in general, a consideration of the site and nature of the ulcer or the presence of some other pathology, is important. When one analyses the results according to final diagnosis, the position is as follows.

TABLE NO. 2.

		Compiled from the published figures of Avery Jones[3] Total Cases 615	Compiled from the present investigation Total cases 280
Chronic Ulcer Group	Gastric Ulcer	11.5%	12%
	Duodenal Ulcer	9%	10%
	Anastomatic Ulcer	3%	6.6%
Acute Ulcer Group		1.6%	0%
Portal hypertension		32%	30%
Carcinoma		30%	50%

It is clear that the Group comprising acute erosions, acute gastritis, "gastrostaxis" &c. carry a negligible/

negligible mortality. The high death rates in carcinoma and in portal hypertension are not surprising and in the present state of medical knowledge of these diseases, no improvement can be looked for, but numerically they form a small group. Attention is therefore directed to the chronic ulcer group, which carry a moderate mortality but owing to their numbers, provide the bulk of the fatal cases. In this group, gastric ulcer carries a higher death rate than does duodenal ulcer, but owing to the preponderance of duodenal ulcer in men, the deaths from duodenal ulcer out-number those from gastric ulcer; in this series by more than 2 to 1.

The factors which decide the outcome in any individual case are many, of which the prior state of nutrition of the patient, the presence or absence of complications in the stomach or duodenum and the presence or absence of complicating disease in other systems are of great importance, but Table I shows that in the chronic ulcer group, the age factor is probably the most important single factor of all. This is readily understood. The older patient will generally have had his ulcer longer. It will be more fibrotic and the bleeding vessel will/

will be rigidly held and incapable of contraction. The vessel itself will be the seat of a greater or less degree of arteriosclerosis. There will be a higher proportion of complications such as pyloric stenosis and of cardiovascular, renal or respiratory disease and even in the absence of any of these, the older patient will be less able to stand the shock and the metabolic upsets which follow sudden and severe blood loss.

The introduction of early feeding and liberal fluids as one of the principles of treatment has eliminated dehydration and uraemia as the common mode of death. Recent writers (Avery Jones[3], Gibson Graham[4]) stress the point that the usual P.M. finding is a chronic ulcer with a pouting, fixed vessel, from which bleeding could hardly be expected to cease spontaneously and that the actual cause of death is usually blood loss. Among the 17 deaths from chronic ulcer in the present series, P.M's. were obtained in 8 and in 7, such a vessel was found. It would appear, therefore, that the potentially fatal cases should be recognised clinically, apart from the fact that they are in an older age group and apart from any complication, by the presence of continuous or repeated/

repeated haemorrhage.

There are many criteria of severity of bleeding, all helpful but all with drawbacks of various sorts and only the pulse rate and blood pressure and the examiner's clinical observation of the patient give any indication of when bleeding is actually going on.

The presence of bright red blood in the vomitus is conclusive evidence of recent bleeding, but is not always available and is not evidence of the total amount of blood lost. The patient's or his friend's account of how much blood he lost in vomiting is notoriously unreliable. Even when no doubt exists that there has been a haematemesis, the extent of it cannot be judged with accuracy unless the entire vomitus can be inspected. A small amount of blood may readily colour a large amount of gastric content.

It is even more difficult to be sure of how much blood has been lost in melaena and when exactly the bleeding occurred. In the average case, there is a depression of alimentary function and the bowels may not move without enemata for several days, the first motion then showing no evidence of bleeding. In a severe case, it is not uncommon to find that there have been several large/

large loose stools of typical tarry colour and sometimes showing the reddish tints of incompletely altered blood, but this will depend on the rate of intestinal action in the particular case. The passage of blood may occasionally be so rapid that it is passed unaltered and the stool may suggest colonic rather than upper alimentary bleeding.

Schiff et al., quoted by Avery Jones, have shown, in normal individuals who were fed blood by stomach tube, that although it required 100 - 150 ccs. of blood to produce melaena, it required 1000 - 2000 ccs. in four hourly doses of 600 - 700 ccs. to produce a loose motion containing unaltered blood and the response was not predictable.

Objective evidence of bleeding is provided at the bedside by rising pulse rate, falling blood pressure and the signs and symptoms of shock. The production of these signs is related to the rapidity of blood loss rather than to its amount and ultimate severity. The type of case admitted in a state of extreme collapse which rapidly disappears with rest and warmth, is common experience and the subsequent course of the illness will show that the amount of blood loss was not great. At the/

the other end of the scale is the patient whose bleeding is slow but persistent. If restoration of blood volume by dilution can keep pace with the oozing, there may be no change in blood pressure and the pulse rate will only rise with the gradual development of anaemia. The pulse rate is also subject to too many other influences — the presence of cardiovascular or respiratory disease, whether the patient has just been sick or used a bed pan, whether he is worried about himself or by the presence, or it may be the absence, of his relatives, by the events around him on a busy receiving day and the unfamiliar surroundings of a hospital ward.

The blood pressure is subject to similar variations and more important, are the showings of Howarth & Sharpey Schafer[5] that the changes in blood pressure after haemorrhage are not constant. In a number of cases, there may be an actual rise in blood pressure and an increase in pulse pressure especially 24 - 48 hrs. after the initial bleeding.

It has been held by Witts[6] and others, that a pulse rate of over 120/min. and a systolic B.P. of less than 90 mms. Hg. are indicative of a severity justifying transfusion, but Dr. David Smith[7] records that/

that in his series many patients who died, never had a rapid pulse rate or only showed it terminally, whereas 80% of those with a pulse rate over 120/min. survived although not transfused. Similarly, 82% of those with a systolic pressure under 90 mms.Hg. survived without transfusion.

It is clear that the use of the pulse rate and blood pressure as guides to the continuance or resumption of bleeding is essential and very useful in the observation of a case, but that they are subject to limitations and that if other methods can be shown to provide confirmatory evidence, there is a place for them in the assessment of upper alimentary haemorrhage.

Two other commonly used criteria of severity are the haemoglobin percentage and the blood urea. It is now fully realised that the haemoglobin cannot give a true picture of the amount of blood loss until haemodilution is complete and the percentage haemoglobin has become stabilised. The process may take 24 hrs.or several days according to the amount and duration of the haemorrhage and to the amount of fluid available to the patient and his prior state of hydration. The position becomes more complicated where bleeding is continuing or has restarted. The/

The haemoglobin, therefore, is of very limited value in the detection of haemorrhage actually in progress and although it is true that once haemodilution is complete, the drop in haemoglobin gives a very fair idea of the severity of the haemorrhage, the knowledge would be more useful at an earlier stage in the progress of the case.

The course of azotaemia in upper alimentary haemorrhage has been exhaustively investigated by Black(8). He has shown that the rise in blood urea may be extremely rapid, a figure of 83 mg% having been found in one case two hours after bleeding, that the peak of the rise is usually 24 hrs. after the bleeding and that thereafter there is a rapid fall to about 50 mg%, which figure is maintained for about a week. If haemorrhage is very severe, the blood urea may continue to rise for several days and in a fatal case may go on rising till death. A secondary rise due to recurrent haemorrhage is often greater than the initial one.

He showed further that azotaemia was the result of three factors (1) the presence of large amount of blood in the bowel. A litre and a half of blood would produce 45 G. of nitrogen or the equivalent of 4 days normal diet/

diet. Clearly this factor would take some time to produce a rise in blood urea and cannot be the cause of the initial rapid rise.

(2) functional renal failure due to low blood volume and salt and water depletion or both. The mechanism involved is doubtless that demonstrated by Trueta and Barclay[9], whereby renal vasoconstriction affecting chiefly the interlobular arteries in that part of their course beyond the juxtamedullary glomeruli, causes a "shunt" of the renal circulation from cortex to medulla, with consequent oliguria and urea retention. This mechanism also, will not cause a rapid rise in blood urea.

(3) tissue breakdown. This is evidenced by loss of weight, excretion of inorganic sulphur and phosphorus, creatinuria and increased urinary potassium. These changes, Black believes, are due to loss of intracellular water by dehydration and by loss of sod.chloride with resulting upset of the colloid state of the intracellular protein. Anoxia he also regards as a contributing factor.

Summing up, one may say that the rise in blood urea will indicate by its degree and persistence, the/

the severity of the haemorrhage but that the figures will be influenced by latent renal disease or by prior dehydration and that the changes in blood urea will be too slow to be of value as a guide to the presence or absence of bleeding at a given moment. Black found no correlation between blood urea and pulse or blood pressure.

Finally, the presence of a leucocytosis has been used as indicative of bleeding. According to Whitby and Britton[10], the leucocytosis which follows haemorrhage "appears within a few hours". "It is usually maximal in about ten hours and lasts, in the absence of complications, for three to four days." Leucocytosis, therefore, does not give very precise information about bleeding. Its usefulness will be as additional evidence of bleeding in cases where that diagnosis is in doubt, rather than as a measure of the severity of bleeding. Whitby and Britton state that the magnitude of the leucocytosis is not proportional to the amount of bleeding. It is stated by Avery Jones not to occur in haematemesis due to hepatic cirrhosis but has been observed by him in cases of Banti's Syndrome.

The ideal guide to the presence or absence of bleeding/

bleeding is, of course, the blood volume estimation if done serially. The available methods, however, are rather laborious and time consuming. They are not suitable for routine use and their accuracy in the presence of haemorrhage and shock has been questioned. The best of the recent methods require the employment of a photo-electric colorimeter which is an expensive piece of apparatus and few hospitals will possess more than one. Usually it is kept in the Biochemical laboratory, where it tends to be inaccessible, especially during the night. "Evans blue" is now used in preference to Congo red on account of its slower disappearance from the blood and Crooke and Morris[11] have described a method with which a result accurate to ± 7% may be obtained using a Dubosq colorimeter and filter. This method was tried out as an aid to the assessment of progress in haematemesis and while it would be excellent where a single estimation was required, it proved quite unsuitable for serial estimations. It was found that two estimations on succeeding days, each requiring the administration of 5 ccs. of 0.70% Evans blue intravenously were sufficient to produce such a degree of blueness in the blood and tissues that the patients presented the appearance/

appearance of extreme cyanosis and circulatory failure, to the alarm of themselves and their relatives. Moreover, each estimation required 1½ - 2 hours time although this could probably be reduced with further practice. It was not possible to use a smaller quantity of dye because the shades of blue produced in the ultimate test solutions would have been too pale for accurate comparison in a Dubosq colorimeter.

It was felt that any method of investigation, to be of value, must be one which could be repeated as often and as soon as necessary, that it must be quick and simple, so that it could be done along with other work and that it should not be detrimental to the patient. Blood volume estimations were therefore discarded and other methods considered.

Of the constituents of the blood, the plasma proteins are most quickly restored after blood loss and might be expected to reflect most quickly the effects of haemorrhage. The present conception of the plasma proteins, initiated by Whipple[12], is that they form one part of a large pool of "mobile" protein, the other part consisting of available cell protein and being provided principally by the liver. The proportion of plasma/

plasma protein to the rest of the pool is estimated as 1 : 30, Elman & Lischer[13]. This pool may be used to furnish haemoglobin, plasma protein or cell protein as the occasion requires. It has been shown by many workers, that after experimental bleeding in animals, the plasma proteins are restored with remarkable rapidity from the mobile pool. In cats, Beattie and Collard[14] state that the movement of protein into the blood can be detected within 2 hours and Calvin[15] reports that in dogs 50% of protein lost by bleeding was made good in 4 hrs. Wallace and Sharpey Schafer[16], in experimental bleeding of convalescent patients of amounts up to 1150 ccs., declare that restoration is so prompt that little change can be detected in the plasma proteins. Drew, Scudder and Papps[17] also state that changes in plasma protein in either overt or concealed haemorrhage "do not approach the percentage loss of cellular elements as indicated by fall in whole blood specific gravity and haematocrit."

It seemed reasonable to hope, therefore, that serial estimations of the plasma proteins in cases of upper alimentary haemorrhage would show by a steady fall that bleeding was continuing and that cessation of bleeding/

bleeding would be reflected in a prompt rise, and further, that these conclusions could be drawn before they would be apparent by other methods. If one could say with certainty that no further bleeding was taking place, one could view with less concern a falling haemoglobin, knowing that it indicated only haemodilution and one could judge accurately from its fall the amount of blood loss. Conversely, if one could say that bleeding was continuing, a step would have been taken towards the early recognition of the possibly fatal case.

The plasma proteins can be quickly estimated by the Copper Sulphate Method, which lends itself ideally to the present purpose, Phillips, Van Slyke et al[18]. The principle of the method depends on the fact that a drop of blood or plasma allowed to fall into a solution of copper sulphate becomes instantly encased in a film of copper proteinate which causes the drop to remain discrete. The gravity of the drop remains constant for 15 - 20 seconds and its rise or fall during this period indicates whether it is of lower or higher specific gravity than the solution. Where the gravities are equal, the drop remains suspended. By letting drops of blood or plasma fall into a series of bottles of copper sulphate/

sulphate of graduated and known specific gravity, the specific gravity of the blood or plasma can readily be determined and in the case of the plasma, the plasma proteins can be deduced from the specific gravity by the charts supplied for the purpose. The size of the drop does not matter so that special pipettes are unnecessary and no temperature correction is needed, as in earlier specific gravity methods, because the expansion coefficients of the solutions is approximately that of blood and plasma. The commonly used ammonium-potassium oxalate mixture or heparin are both permissible as anticoagulants and the amount of plasma needed is very small so that it was found that the plasma in the haematocrit tube served the purpose, after the haematocrit had been read. It is possible from the specific gravity of both blood and plasma, by the use of line charts, to calculate the haematocrit provided the erythrocytes are normal in specific gravity and size, but for the sake of accuracy the haematocrits in this investigation were obtained in the usual way and the alternative method used only as a check on the accuracy of the specific gravities. It was found that the haematocrit values by the two methods always agreed to within 2%. As a theoretical consideration, it would seem that in extremely dilute/

dilute blood the lower osmotic pressure should result in slight increase in the erythrocyte size but evidently this is insufficient to prejudice the method. The authors state that in cases with normal erythrocytes the expected error would be 1.4%.

Provided the 3 : 2 mixture of ammonium and potassium oxalates is used in a strength not greater than 1 mg. per 1 cc. of blood, no correction is needed for its effect on plasma or blood gravities. No correction is needed for heparin.

The author's claim that plasma gravity can be determined to ± 0.0003 which corresponds to ± 0.1 G. protein per 100 ccs. blood. In average practice, however, the error, as compared with Kjeldahl methods, is 0 - ± 0.3 G per 100 ccs. This is a considerable error when small changes in plasma protein are considered. This error is due to non protein constituents of the plasma particularly urea and glucose, which raise gravity. Glucose is not likely to influence the result in cases of upper alimentary haemorrhage but the urea concentration will certainly do so and must be borne in mind. Since it will have the effect of raising the apparent plasma protein level, it may suggest cessation of haemorrhage before/

before it has actually occurred. Adams & Ballon[19] in 128 cases in which the plasma proteins were estimated by both methods found that 42% agreed within 0.3 G/100 ccs.

During the past year, estimations of the plasma proteins have been done as part of the routine observation of upper alimentary haemorrhage. Observations were made several times a day or daily as the circumstances of the cases seemed to warrant. In all, 25 cases were examined. Haematocrit, plasma proteins and blood urea were estimated on the same sample of blood each time, the latter estimation being done by the urease-Nesslerisation method using a Lovibond comparator.

On consideration of the results, the first striking fact is that the restoration of plasma proteins in the majority of the patients, while rapid, has not been nearly so prompt as experimental results in animals and man would have suggested. It was expected that plasma protein levels would have reached the lower limit of average normal within 24 hrs. of cessation of haemorrhage. Instead, in most cases and always in severe haemorrhage, it has taken several days and sometimes weeks to return to normality.

Whipple & Madden[12] have demonstrated that the/

the mobile pool of body protein can be depleted by repeated bleeding, after which the plasma proteins ceased to be restored, but in their animals this process of depletion required daily bleeding for 2-3 wks. This is a much more severe drain on body protein than occurs in natural haemorrhage from the upper alimentary canal except in cases of exceptional severity and duration. It seems unlikely that the human protein pool can be thus depleted by haemorrhage alone, but most cases of haematemesis cannot be considered healthy as the experimental subjects and Whipple's dogs were. They are suffering from a disease which may have reduced their food intake and particularly protein intake, so that their available protein may have been partially depleted before bleeding occurred.

Some support for this contention may be afforded by consideration of individual cases.

Case No. 17 was a woman aet 75 yrs., who had been taking aspirin in large amounts for the relief of pain due to rheumatoid arthritis. She was well nourished and her haematemesis, which was moderately severe, was preceded by minimal dyspepsia. Subsequent investigation showed no lesion to account for her bleeding, which

was/

was considered to be due to aspirin. She may be taken as representative of bleeding in a person with previously normal dietary and in her case, the plasma proteins fell but slightly and had attained their former level in 24 hrs.

The opposite extreme is found in case No. 21, a girl of 20 yrs., who, while not suffering from dyspepsia, had been living on a very inadequate diet principally of carbohydrate and had been under treatment for anaemia before haematemesis occurred from an acute ulcer. In her case the plasma proteins were still only 5.4.G.% a month after admission.

The possibility exists that slow restoration of plasma protein in such cases might be due to hepatic insufficiency and that this insufficiency might be due to or be aggravated by anoxia. The restoration of plasma protein is, in the first place, by replacement by albumin and plasma albumin is known to be low in cases of hepatic disease. At first sight, it seems unlikely that anoxia can play a part because the amount of reserve protein at the moment of bleeding cannot be affected by subsequent liver damage. Possibly anoxia might interfere with mobilisation of available protein/

protein, but it seems more probable that any hepatic damage present, existed before the bleeding and has resulted in a depletion of protein reserve.

Of liver function tests carried out in these cases as soon as the patient's condition became stabilised only the hippuric acid test has shown consistently low figures. In the following table, the cases have been arranged in order of severity of anaemia.

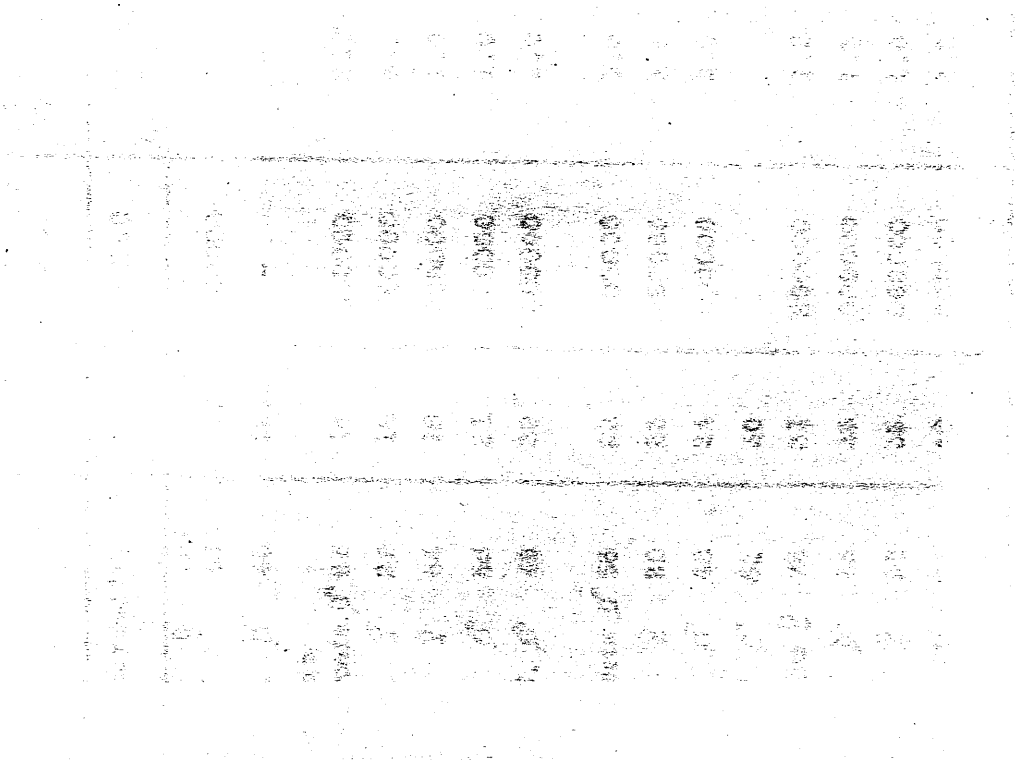


TABLE NO. 3.

	P.C.V.	Colloidal Gold.	Thymol Turbidity	Plasma Alkaline Phosphatase	Hippuric Acid	Plasma Proteins
Normality		000000	McLaggan 0-4 Units	Bodansky 1.5-4.0 Units	As Benzoic Acid 0.68-1.16 G.	6.3-7.7 G%
G.U. ♀ aet 42	11	200000	1.5	0.33	0.392 (163)	5.6
G.U. ♂ 45	12.5	-	-	1.8	-	5.0
Arterio-sclerosis ♂ 63	14	000000	2.75	1.37	0.27 (100)	-
Ac.U. ♀ 29	18	000000	4.3	0	0.26 (103)	5.8
D.U. ♀ 54	18	000000	1.0	0.11	0.646 (100)	6.7
D.U. ♂ 56	21	000000	1.0	2.2	-	6.2
Jej.U. ♂ 42	22	000000	1.0	0.11	0.288 (200)	-
Art.-sclerosis ♂ 56	22	100000	5.0	2.2	0.33 (150)	5.4
Ac.U. ♀ 20	23	320000	1.1	3.3	0.76 (776)	5.4
D.U. ♂ 42	24	000000	3.5	1.1	0.3 (136)	5.8
D.U. ♂ 63	30				0.641 (180)	6.5
Aspirin ♀ 75	31	200000	1.5		0.549 (202)	6.7
D.U. ♂ 23	36	000000	1.5	1.05	1.33 (225)	5.9
Ac.U. ♀ 20	39	000000	1.0			6.65
D.U. ♂ 63	40	000000	2.0	1.2	0.34 (180)	6.8

Figures in brackets after amount of benzoic acid represent urinary volume in ccs. Figures rendered invalid by too low a volume of urine have not been included.

Although not measuring any particular function of the liver, the hippuric acid test is recognised as giving a good "all round" indication of the state of liver function. Wide variation is recorded in the results of hippuric acid excretion in the normal individual, but in a recent review of the subject in the British Encyclopedia of Medical Practice, Medical Progress 1948[20], the lowest figures were recorded by Lippincott et al whose range was 0.225-1.16 G. Eighty per cent of normals, however, fell between 0.68 - 1.16 G. which figures are about those usually accepted as the normal range.

By these criteria, most of the above cases would seem to show a deficient synthesis of hippuric acid, but there is no close correlation between the degree of hepatic deficiency and the degree of anaemia or between liver function and the plasma protein levels.

When the figures for a series of dyspepsias without haemorrhage are examined, it is clear that there is no significant difference between the two groups.

TABLE NO. 4

* indicates duodenal ulcer with pyloric stenosis.

	Colloidal Gold	Thymol Turbidity	Plasma Alkaline Phosphatase	Hippuric Acid
Normality	000000	0-4 McLaggan Units	1.5-4.0 Bodansky Units	0.68 - 1.16G Benzoic Acid
D.U. ♂ aet 39	000000	1.0	1.11	0.411 (67)
D.U. ♂ aet 35	000000	1.5	1.05	0.531 (165)
D.U. ♂ aet 32	320000		1.84	
D.U. ♂ aet 40	200000	4	1.84	1.11 (224)
D.U. ♂ aet 57	000000	1.25		0.488 (96)
D.U. ♂ aet 42	000000	1.5	0.6	0.26 (115)
*D.U. ♂ aet 61	000000	2.7	1.1	0.26 (100)
D.U. ♂ aet 44	000000	0.7	1.2	
*D.U. ♂ aet 34	000000	1.0	0.6	0.23 (108)
D.U. ♂ aet 58	000000	0.5	2.5	0.47 (94)
*D.U. ♂ aet 42	000000	0.5	1.0	0.39 (124)
G.U. ♂ aet 67	210000	1.5		0.2 (108)
G.U. ♂ aet 36	200000	0.5		0.2 (110)
G.U. ♀ aet 66	000000	2		0.3 (176)
G.U. ♀ aet 36	200000	0.5	1.39	0.564 (112)
G.U. ♀ aet 48	220000	2.5		0.54 (110)
G.U. ♀ aet 44	220000	2.0	2.22	0.37 (180)
G.U. ♀ aet 60	100000	0.7	0.9	0.20 (100)
G.U. ♀ aet 65		2.5	1.5	0.2 (120)
Chr.gastritis ♀ aet 52	200000	1.5	1.39	0.53 (105)
" ♀ aet 65	200000	1.25		0.23 (76)
" ♂ aet 41	554220	2.0	1.88	0.456 (110)
" ♂ aet 32	100000	0.5		0.201 (116)

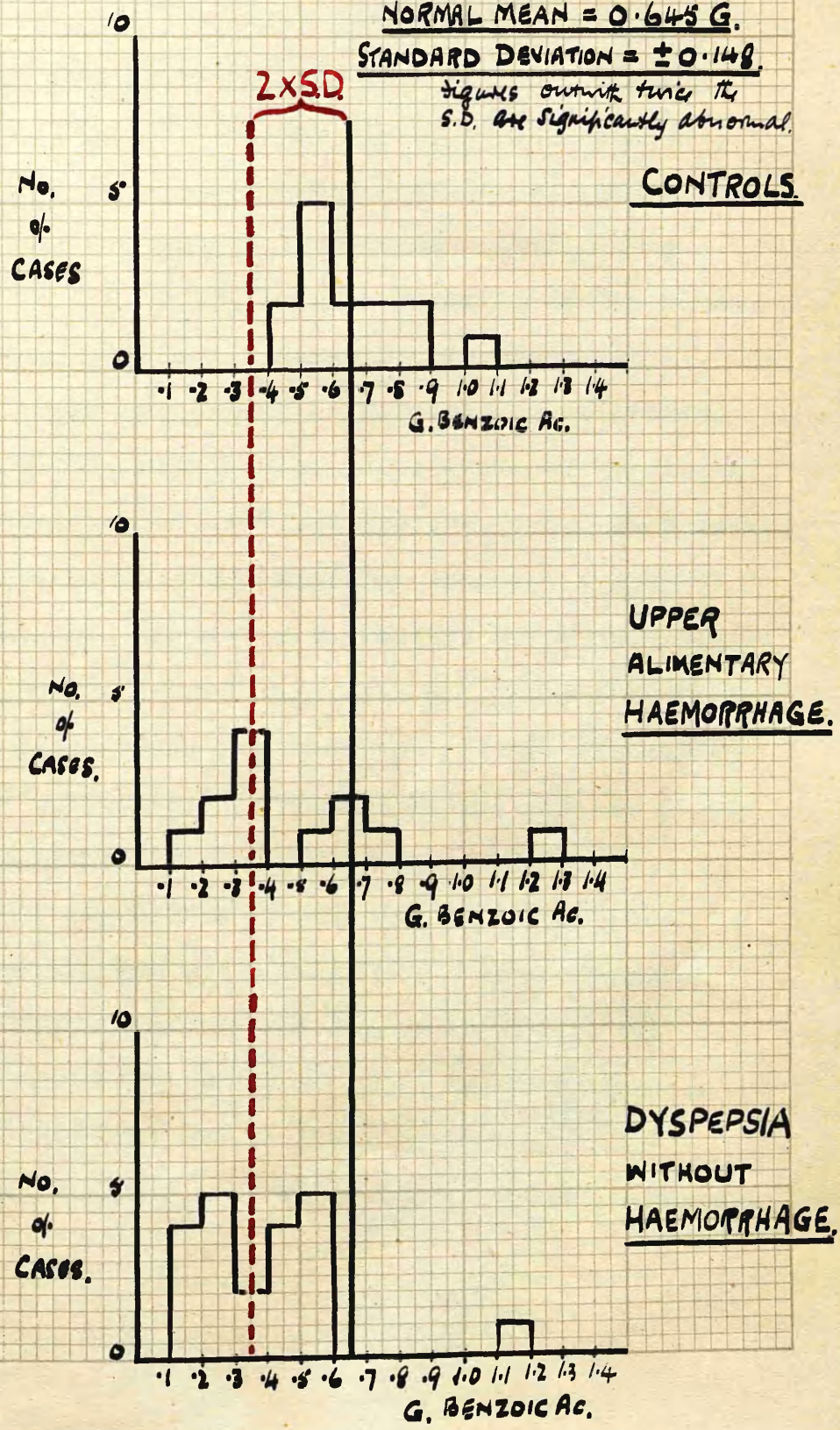
When comparison is made between these two groups and a series of normal controls, there is no doubt that genuine hepatic dysfunction exists in the dyspepsias.

TABLE NO. 5.

	Colloidal Gold.	Thymol Turbidity	Plasma Alk. Phosphotase	Hippuric Acid
Diss.sclerosis ♀ aet 45	210000	2.5	1.0	1.08 (186)
Diss.sclerosis ♀ aet 46	200000	0.5	1.77	0.645(130)
Diss.sclerosis ♀ aet 44	-	1.25	4.15	0.41 (180)
Diss.sclerosis ♂ aet 47	100000	6.5	1.9	0.73 (96)
Functional headache ♀ aet 33	320000	2.0	1.1	0.81 (233)
Sciatica ♂ aet 55	200000	3.2	1.6	0.72 (110)
Hysteria ♀ aet 45				0.535(124)
Dietl's crisis ♀ aet 35	320000	2.0	1.1	0.81 (104)
Normal students ♂	000000	5.5	1.3	0.46 (126)
"	000000	0.5	0.6	0.57 (90)
"	000000	1.75	1.1	0.54 (84)
"	100000	5.5	0.86	0.57 (128)
"	100000	5.5	1.06	0.58 (166)
"	000000	3.25	0.9	0.61 (108)

CHART No. 2.

ANALYSIS OF RESULTS OF HIPPURIC ACID TEST IN DYSPEPSIA WITH AND WITHOUT BLEEDING AND IN CONTROLS



The degree of hepatic dysfunction shown by the dyspepsia group is not always gross, but is very definite and can be shown most forcibly when the results are prepared diagramatically (Chart No. 2).

In interpreting these results, age and sex are factors which must be considered. Appreciably lower figures are to be expected normally in the older patient and in the female sex. To eliminate the influence of age and sex would require the collection of an impossibly large series which would be capable of suitable sub-division, but the present series is reasonably balanced as regards these factors. Among the controls 43% are males and among the dyspepsias 41%. The average age of the controls is 37 yrs., of the dyspepsias 46 yrs. It seems a fair assumption that age and sex variations will not be sufficient to invalidate the results of the groups when viewed as a whole.

One may fairly deduce from these results that the hepatic dysfunction shown by cases of upper alimentary haemorrhage is not due to anoxia in the average case, but is common to other cases of dyspepsia. Obviously a severely anoxic liver must be at a disadvantage compared with another less severely deprived but/

but the lack of obvious correlation between liver function and degree of anaemia seems to show that anoxia is not the principal factor determining dysfunction.

The source of this dysfunction provides interesting grounds for speculation. There are relatively few references to it in the literature, but Pollok[21] gives a brief review and adds his own observations on 68 ulcer patients using the oral hippuric acid test. He quotes Schnitker and Hass and Gordon and Manning, who both noted a high incidence of hepatic cirrhosis in ulcer patients post mortem. Boles et al. noted the association of hepatic lesions and acute ulceration. Various authors report an increase in serum bilirubin in ulcer cases. Johnson & Bockus[22] found it in 10% of 313 cases of duodenal ulcer.

Pollok himself, found 32 patients with a significantly low hippuric acid excretion test out of 68 male cases of peptic ulcer (i.e. 44%). These figures are in close agreement with the present investigation results, in which 6 out of 12 cases of alimentary bleeding and 10 out of 23 of dyspepsia without bleeding, making a total of 16 out of 35 cases (i.e. 46%) showed deficient/

deficient liver function by the intravenous hippuric acid test.

Pollok further showed that the deficiency was not permanent but improved with the response of the case to treatment and that even in individuals whose hippuric acid excretion was within normality on first testing, subsequent tests showed improvement after treatment.

Among suggestions put forward by Pollok to explain the hepatic deficiency are that it may be secondary to hepatic and biliary tract upset produced reflexly by ulcer pain, that hepatic dysfunction or metabolic inferiority may be part of the ulcer diathesis or that hepatic dysfunction may affect the resistance of the gastroduodenal mucosa.

Such suggestions must be purely speculative. A more probable explanation would seem to be, as already noted, that the ulcer patient tends to have a diminished food intake particularly of protein and the importance of protein intake to the wellbeing of the liver is now known. That this is the mechanism at work is difficult to prove, but there are many reasons for believing it to be true. First class protein in bland/

bland form - milk, eggs, white fish, rabbit, chicken &c. is expensive and often beyond the means of the poorer patient, especially after long periods of ill-health and absence from work. Meat is only obtainable in limited amount at present and its preparation and cooking to conform to the requirements of the bland diet is troublesome and imperfectly understood by the patient or his household. The vast majority of the patients of the hospital class at least, will readily admit that they do not adhere to their diet strictly and the reason given is usually the difficulty of doing so.

Anorexia or post prandial pain or fear of eating, all symptoms encountered in ulcer patients, will further tend to reduce food intake. One would expect a difference in this respect between gastric ulcer, which is more often associated with loss of appetite and fear of eating, and duodenal ulcer in which appetite usually remains and eating often gives relief. Such is the case. Of 8 gastric ulcers in the present series the mean figure for the hippuric acid test is 0.303G and for 17 cases of duodenal ulcer the corresponding figure is 0.55G. Worth noting too, are the/

the very low figures obtained in the three cases of pyloric stenosis, in which food intake and its absorption would both be affected. They were 0.26, 0.23 and 0.39 G.

Support for this view may be obtained from Bockus[23]. He states, "Hypoproteinaemia is not rare among the group of patients requiring gastric surgery. Many have been on a diet restricted in proteins; others because of obstruction and disturbance in absorption have been unable to assimilate properly various nutritive elements. Excessive vomiting has further depleted body stores and interfered with the retention of ingested proteins in many others. If acute or chronic diffuse liver disease co-exists with peptic ulcer, other causes for the development of hypoproteinaemia are present."

There is one further factor which should be mentioned in connection with the impaired liver function in peptic ulcer. Peptic ulcer is not alone as a cause of hepatic dysfunction in the absence of obvious hepato biliary disease. Such dysfunction is also found in many conditions, both acute and chronic, of which the common factor is infection, e.g. pneumonia, rheumatic fever/

fever, rheumatoid arthritis, syphilis &c. Hurst[24] emphasises the frequent association of chronic dental, tonsillar or nasal infection with ulcer and quotes Rosenow who isolated streptococci from the deep tissues of chronic ulcers removed at operation in 42 out of 54 cases and from adjacent glands 4 times out of 5.

The infective factor in peptic ulceration is one which tends to be forgotten in the light of more recent views on the aetiology, but is worth consideration as a possible additional factor producing hepatic dysfunction. It seems unlikely, however, that it is any more than that.

The practical application of these findings is twofold. In the first place it is now considered (Wiley[25]) (Bockus[23]) that hypoproteinaemia may cause delayed wound healing, increased susceptibility to toxaemia and to infection and decreased gastro-intestinal tract motibility. It seems a reasonable inference that hypoproteinaemia will also cause delayed ulcer healing and this may be one explanation of the success of Meulengracht's early and liberal finding in haematemesis. In the second place it emphasises the very great importance/

importance of diet in treatment of peptic ulcer and the need for taking time and care and patience in making sure that the patient understands his diet and the reasons for it.

To return to consideration of the plasma proteins, it is again difficult to prove that their slow return to normal after upper alimentary haemorrhage is due to hepatic dysfunction. The available figures do not show any close correlation between their rate of restoration and the liver function tests, but there are too many complicating factors such as severity and duration of haemorrhage, age, sex, duration of symptoms &c. to expect that the correlation would be obvious. When one reflects that the plasma proteins are derived in the first place from dietary protein and are manufactured in the liver, reduction of intake and hepatic dysfunction must at least be causes of major importance, in the slow restoration of the plasma protein. As has been mentioned, the restoration of plasma protein is primarily by albumin (Calvin[14]). The importance of good liver function in maintaining plasma albumin is so well known that Bockus hesitates to make a diagnosis of severe hepatic cirrhosis without finding a hypoalbuminaemia/

hypoalbuminaemia. It is fair to believe, therefore, that the degree of hepatic dysfunction shown to be present in cases of dyspepsia can be a cause of depleted protein reserve and of slow restoration where sudden large demands are made for plasma albumin. One visualises a vicious circle in which poor protein intake and liver dysfunction, on the one hand, and the ulcer on the other hand by mutually aggravating each other, ultimately produce deterioration in the patients general condition, in his protein reserve and in his ability to heal his ulcer. Attention to diet would be the simplest and most effective method of interrupting the circle.

The plasma proteins did conform to expectation in that the degree of fall was not nearly so severe as that of the cellular constituents of the blood as shown by the haematocrit. Since both constituents must be lost in the same proportion during haemorrhage, this fact in itself must mean that restoration of plasma protein commences immediately. It must start with and run a course very similar to that of restoration of blood volume, but where the protein pool is inadequate, as in the peptic ulcer case, restoration of plasma protein soon begins to lag behind the other. The work of/

CHART No 3.

The relative values for packed cell volume and plasma proteins in 21 cases, to illustrate the much less severe fall in the latter.

of Wallace and Sharpey Schafer, already quoted, would indicate that in the healthy person with adequate protein reserve no lag occurs and restoration of blood volume and plasma protein run parallel to completion. Hence their finding that little change can be detected in the plasma proteins. These workers bled their subjects of amounts up to 1150 ccs. which would represent a substantial natural haemorrhage, but where the loss exceeded this, it seems probable that even in a healthy individual some lag would occur.

In general the more severe the final degree of anaemia, the lower the plasma protein level reached, but the lowest figure recorded was 4.3 G%. This figure, which represents 62% of normal, was reached on three occasions in different cases and the haemoglobin levels recorded at the same time were 20%, 22% and 30%. None of these cases showed oedema. The absence of oedema with very low plasma protein levels after haemorrhage has been commented on before. No explanation has been offered, but it may be that the absolute level of plasma albumin tends to be maintained by rapid restoration in contrast to its low level in such conditions as the nephrotic syndrome or hepatic cirrhosis.

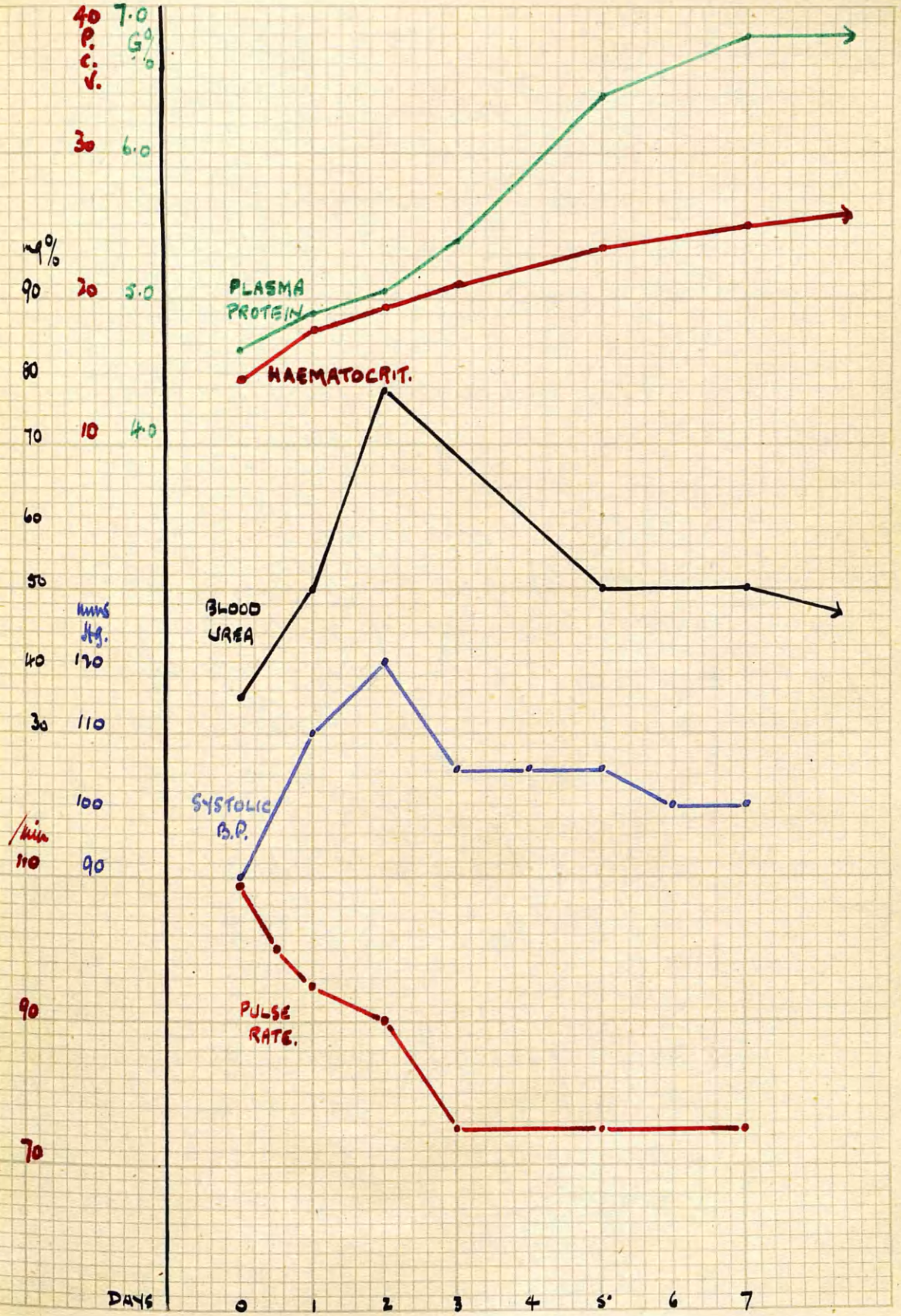
No/

No correlation was discovered between the severity of fall in plasma protein and its rate of restoration. The lowest levels reached did not necessarily mean the most rapid restoration, but rate of restoration is difficult to assess because with a restoration of blood volume occurring simultaneously even a stationary plasma protein level means that considerable restoration is going on.

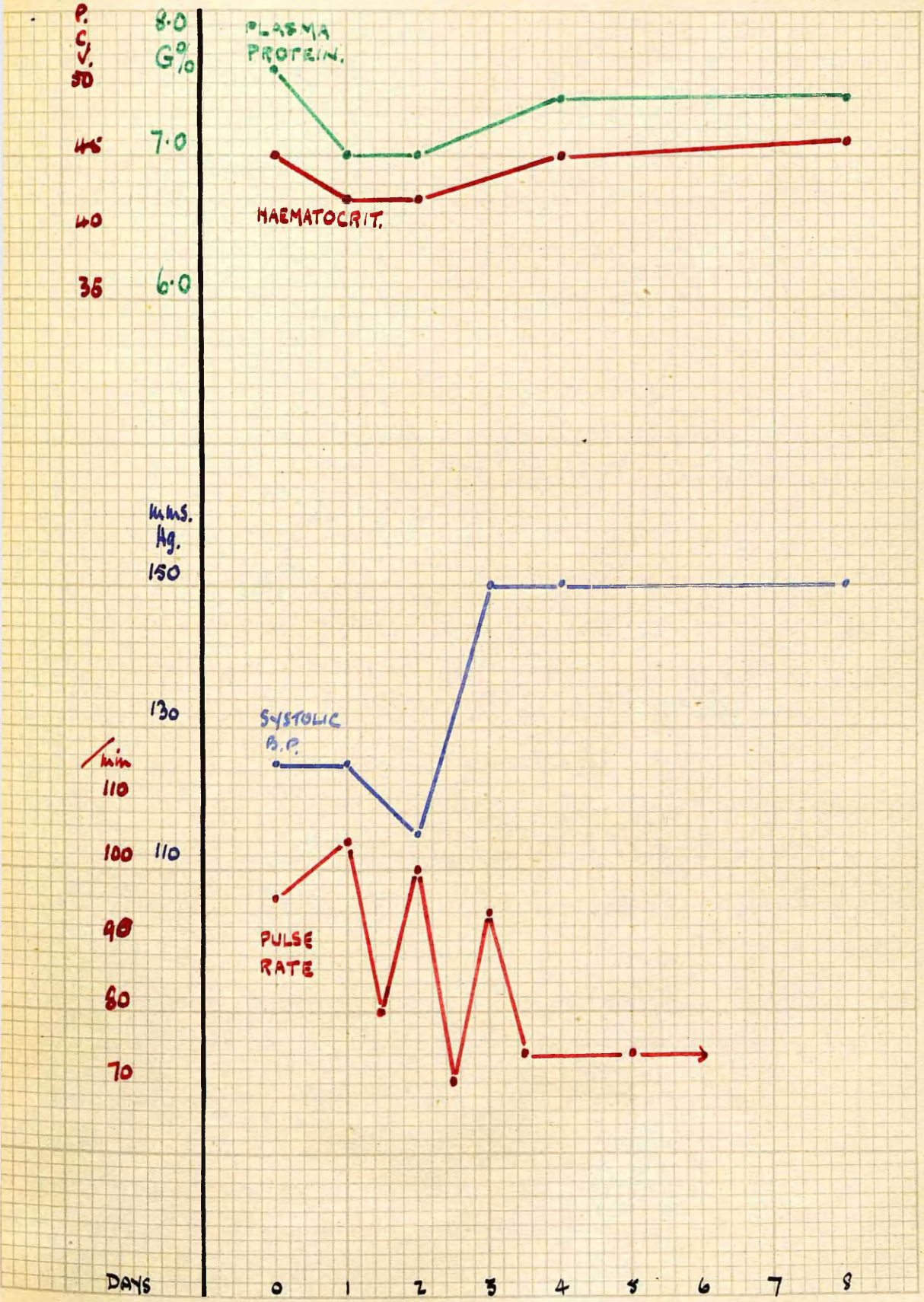
The cases examined fall naturally into three groups. In the first, the lowest plasma protein figure was obtained in the initial specimen and thereafter there was a steady rise, occasionally after a short stationary period. The behaviour of the P.C.V. and the progress of the cases in this group showed that bleeding had ceased either before or immediately on admission. Such cases seldom present any difficulty in assessment. Their clinical improvement is steady and it cannot be claimed that estimations of the plasma proteins help materially in following their progress. In a few cases, as in No. 2, where there was an unstable pulse rate or No. I in which the blood urea continued to rise for 48 hrs. after admission, some reassurance may be obtained from the plasma protein level.

There/

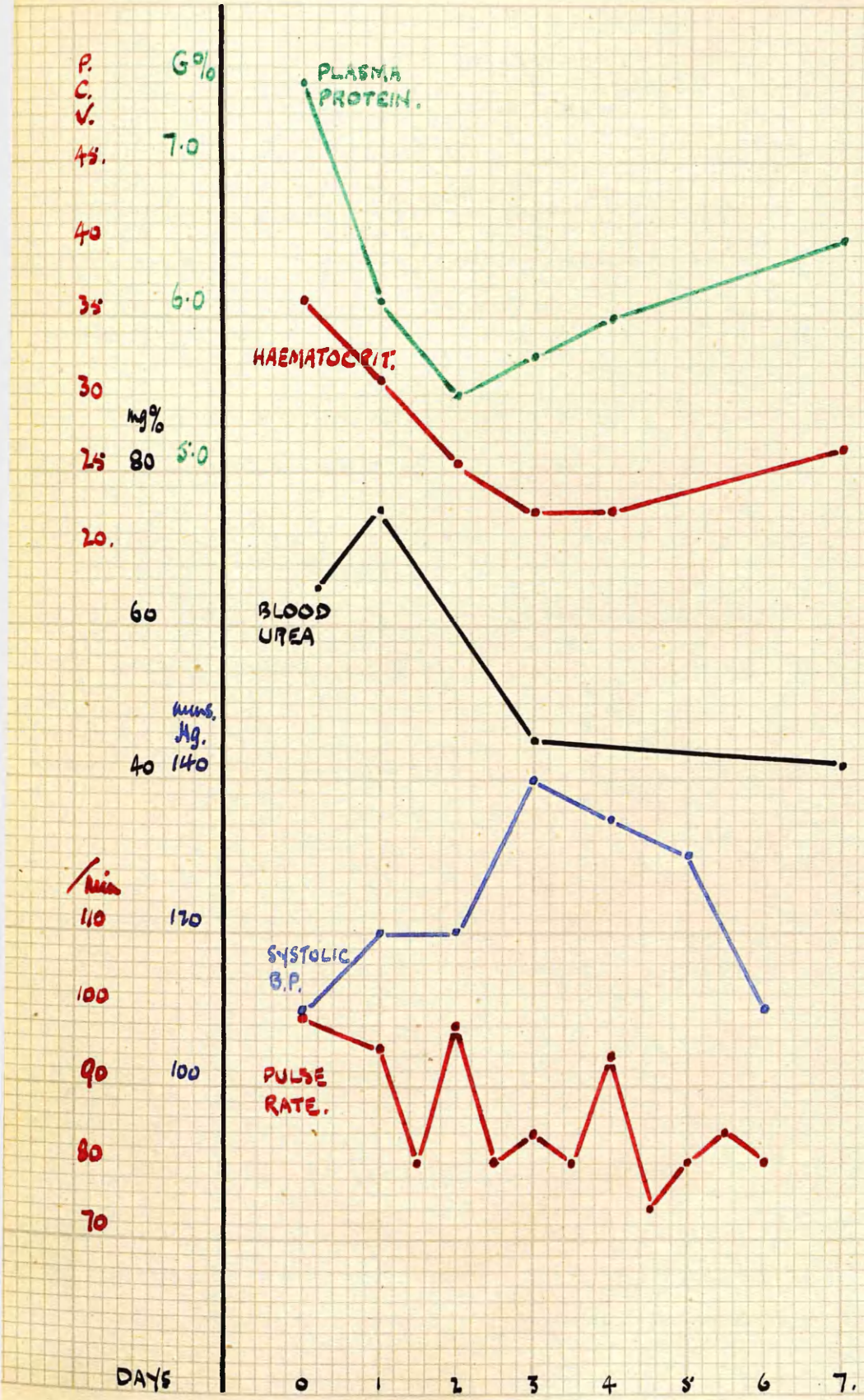
CASE NO. I ♂ aet 42. GASTRIC ULCER.



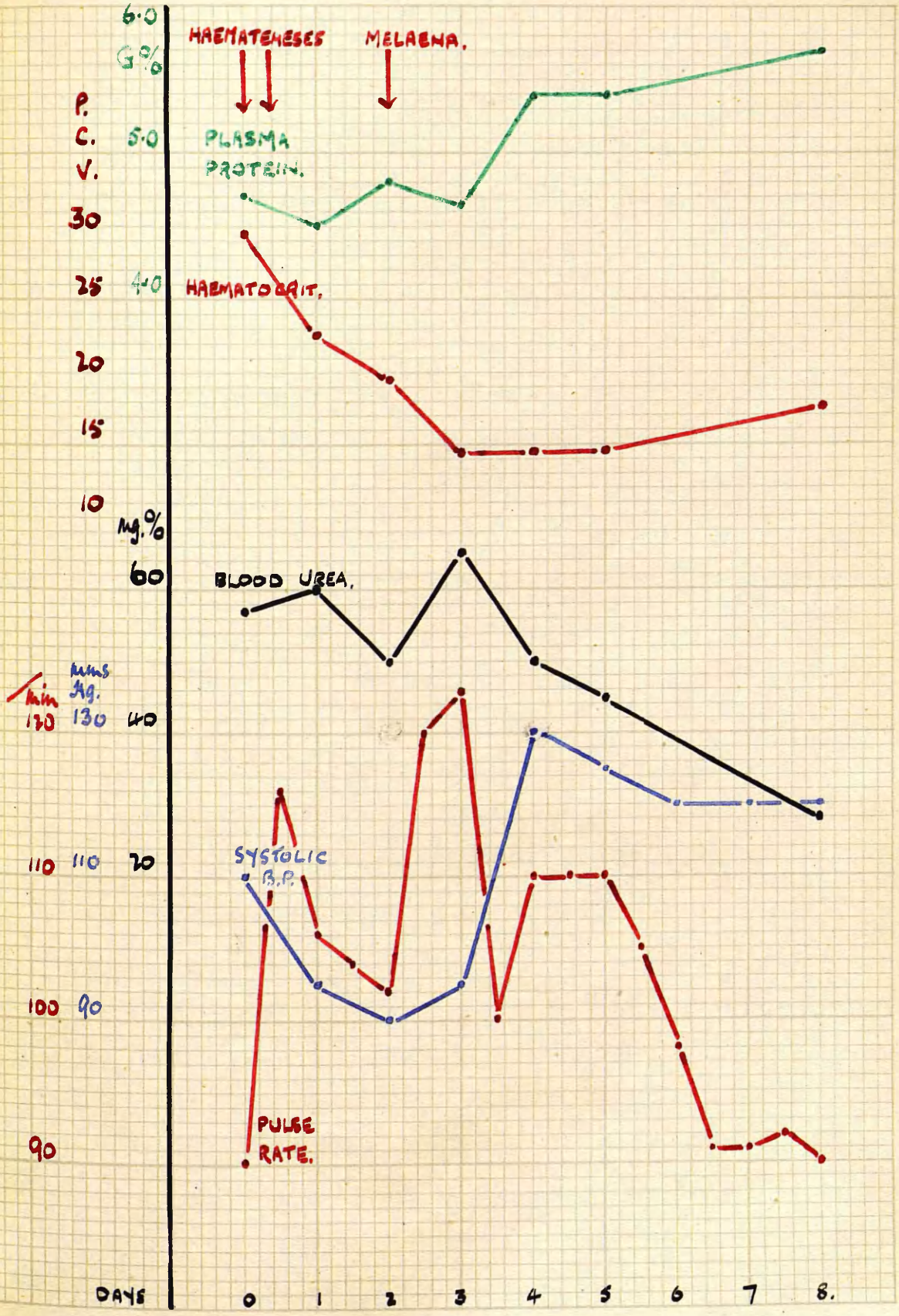
CASE No. 2. ♂ aet. 58. PSEUDOHAEMATEMESIS.



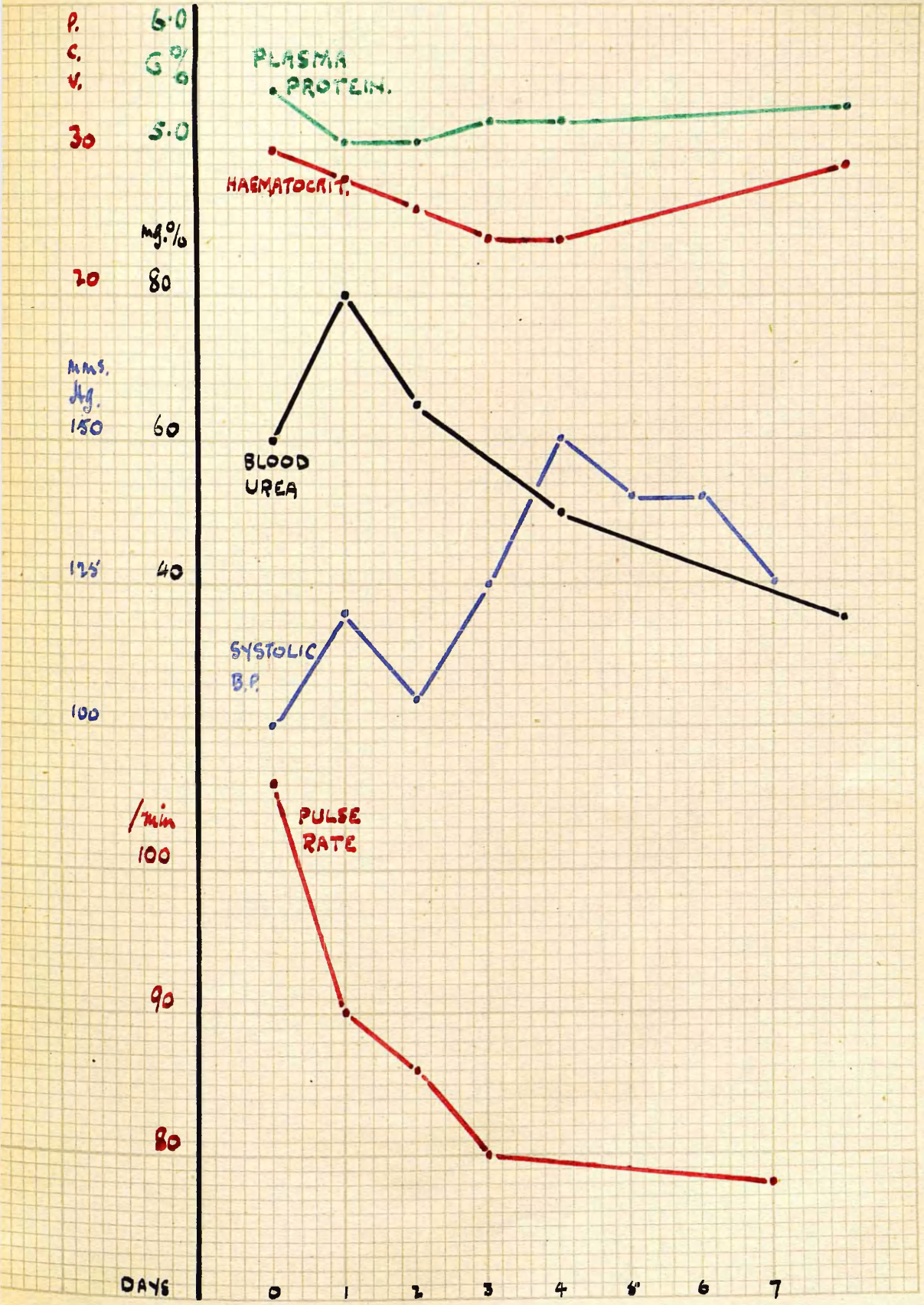
CASE No. 3. ♂ aet. 63. DUODENAL ULCER.



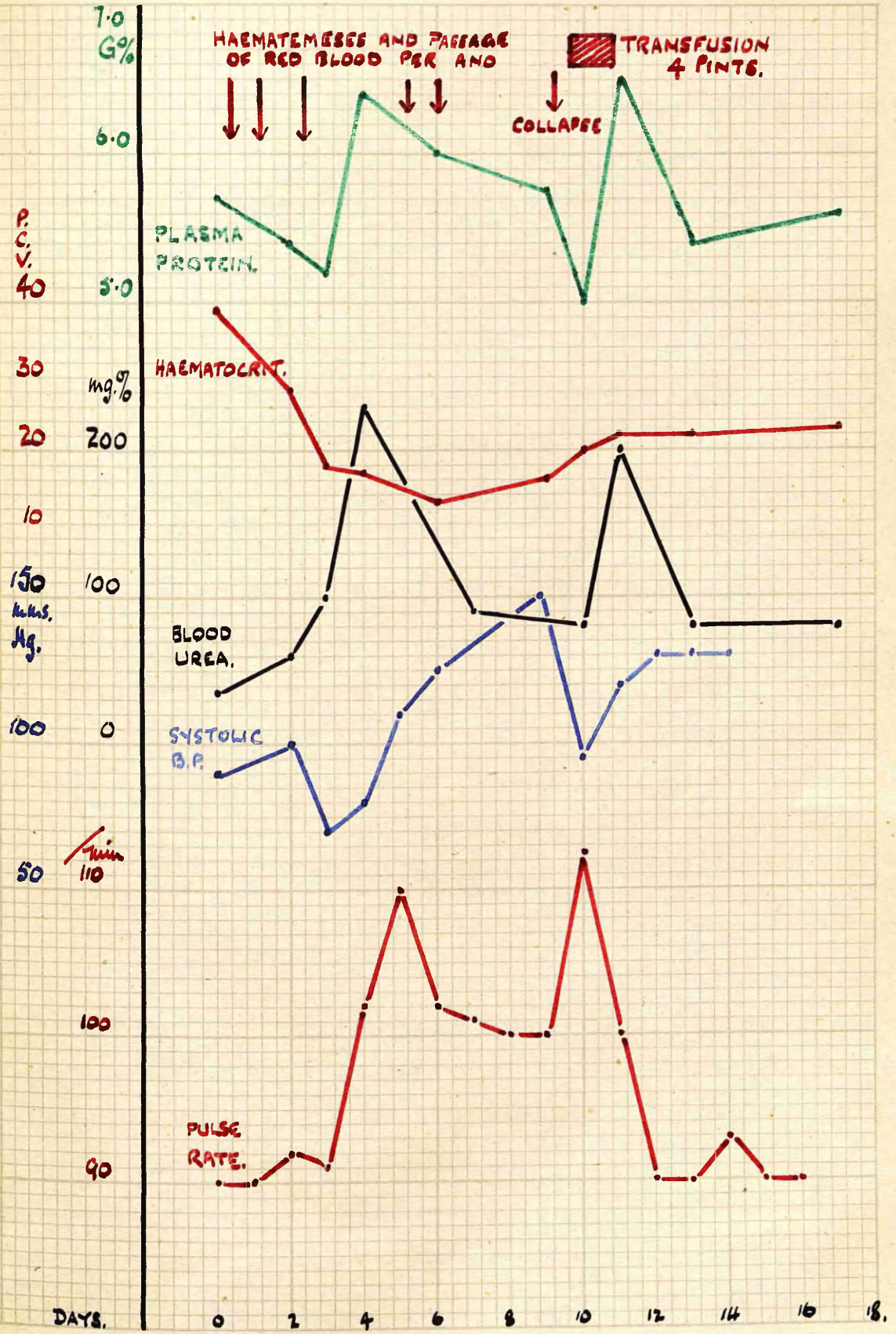
CASE No 4 ♂ Aet 26. DUODENAL ULCER.



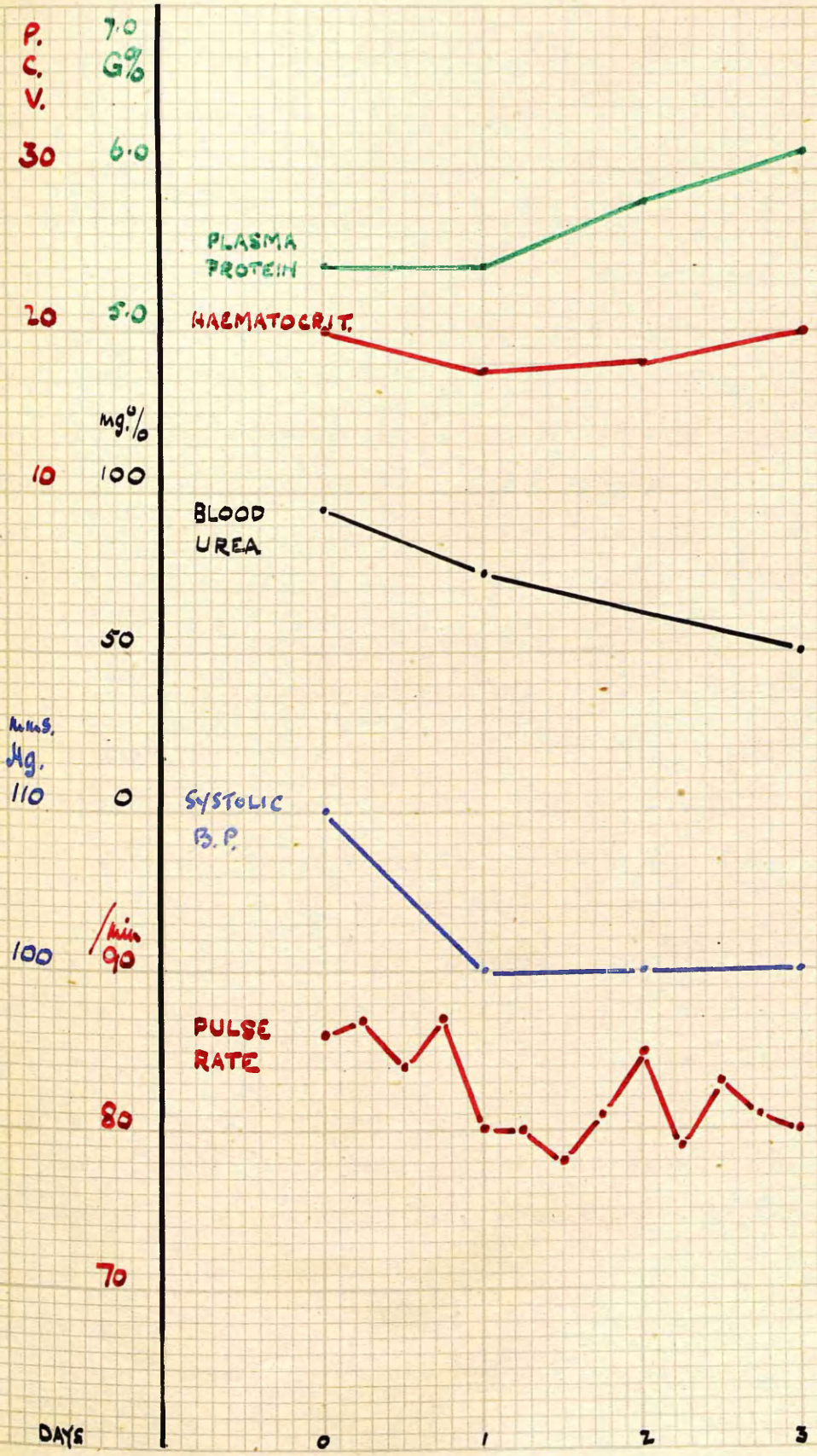
CASE No. 5. ♂ aet. 23. DUODENAL ULCER.



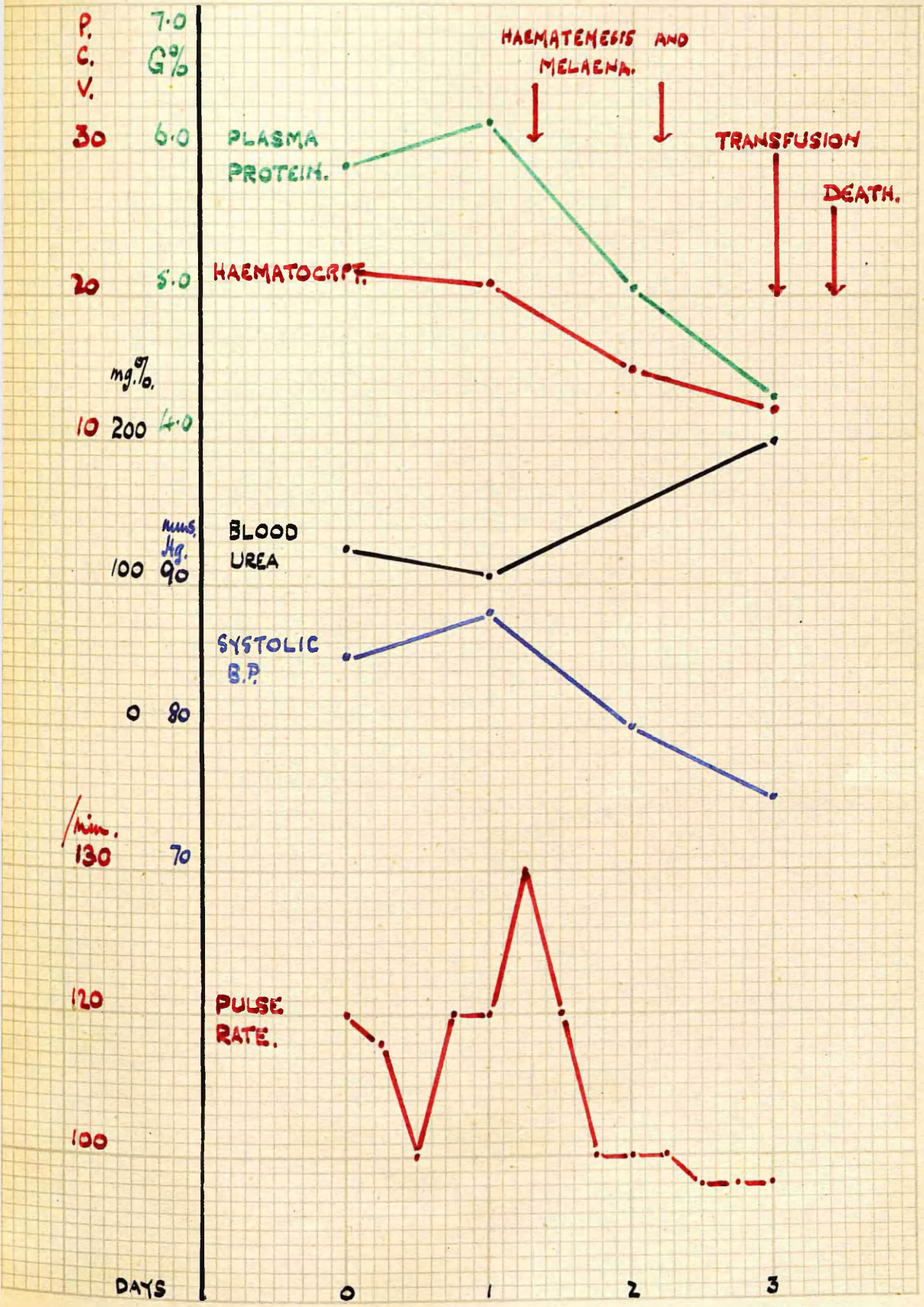
CASE No. 8. ♂ aet. 56. ACUTE ULCER.



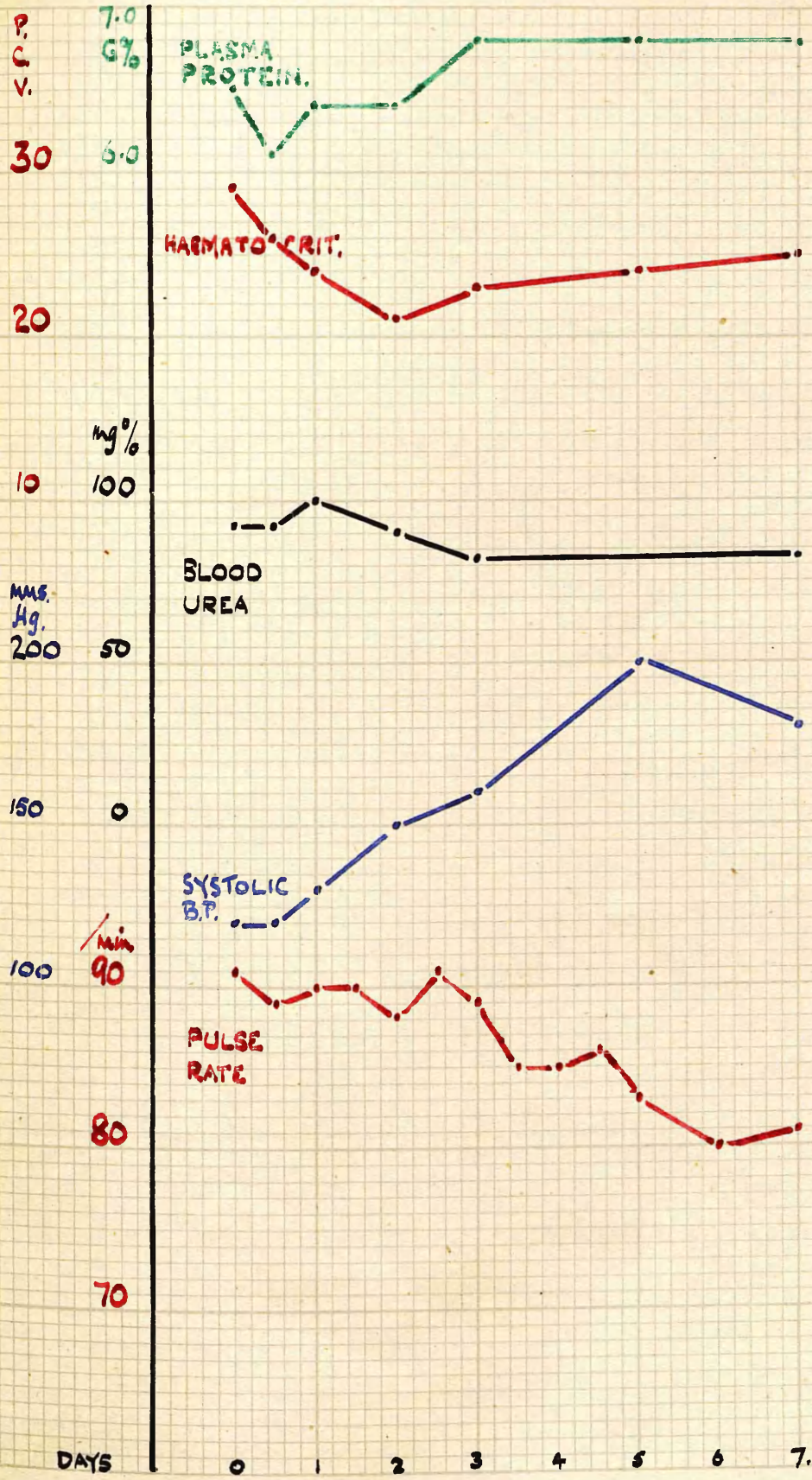
CASE NO. 10. ♂ aet 32 DUODENAL ULCER



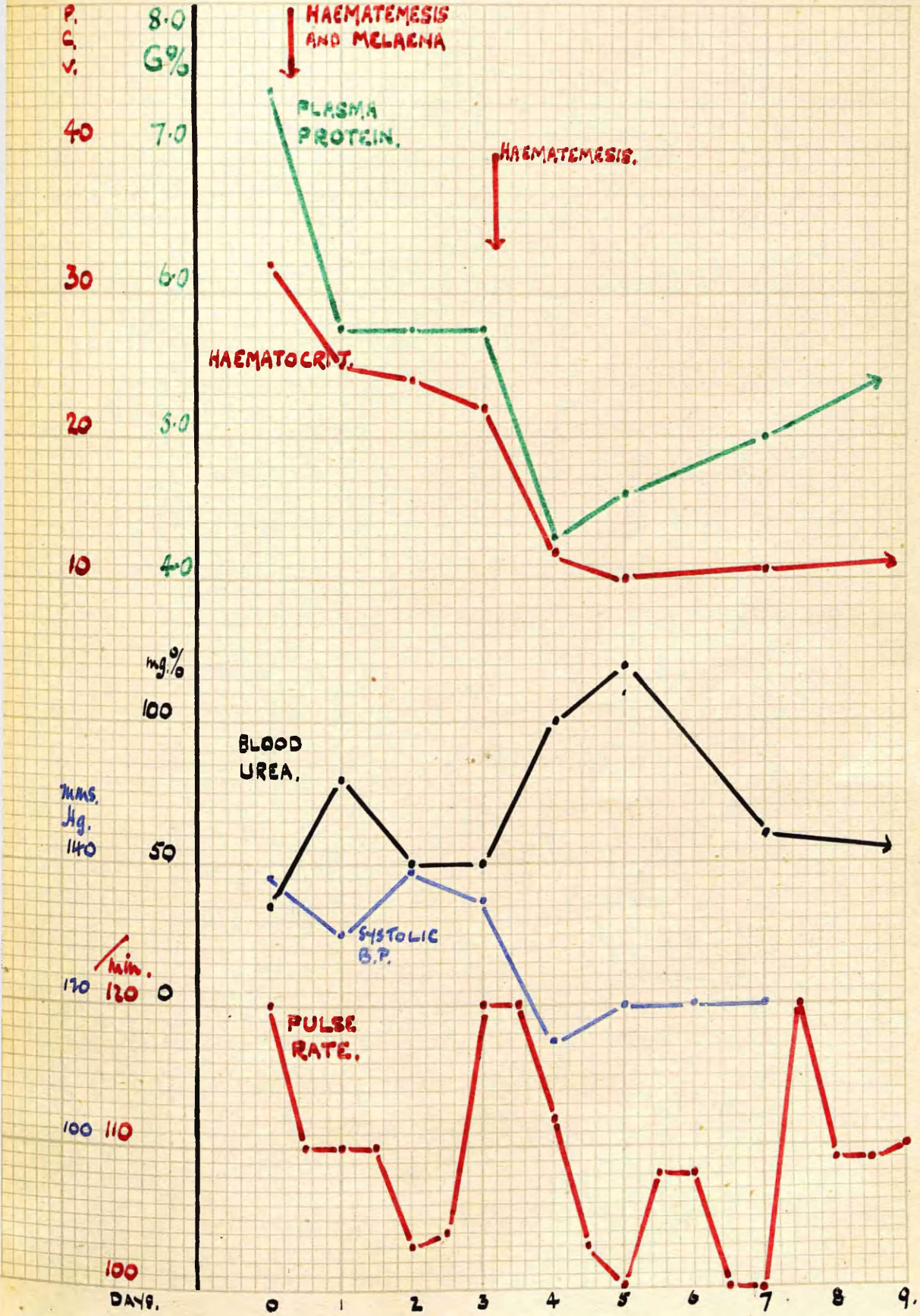
CASE No. 11. ♂ aet. 56. DUODENAL ULCER.



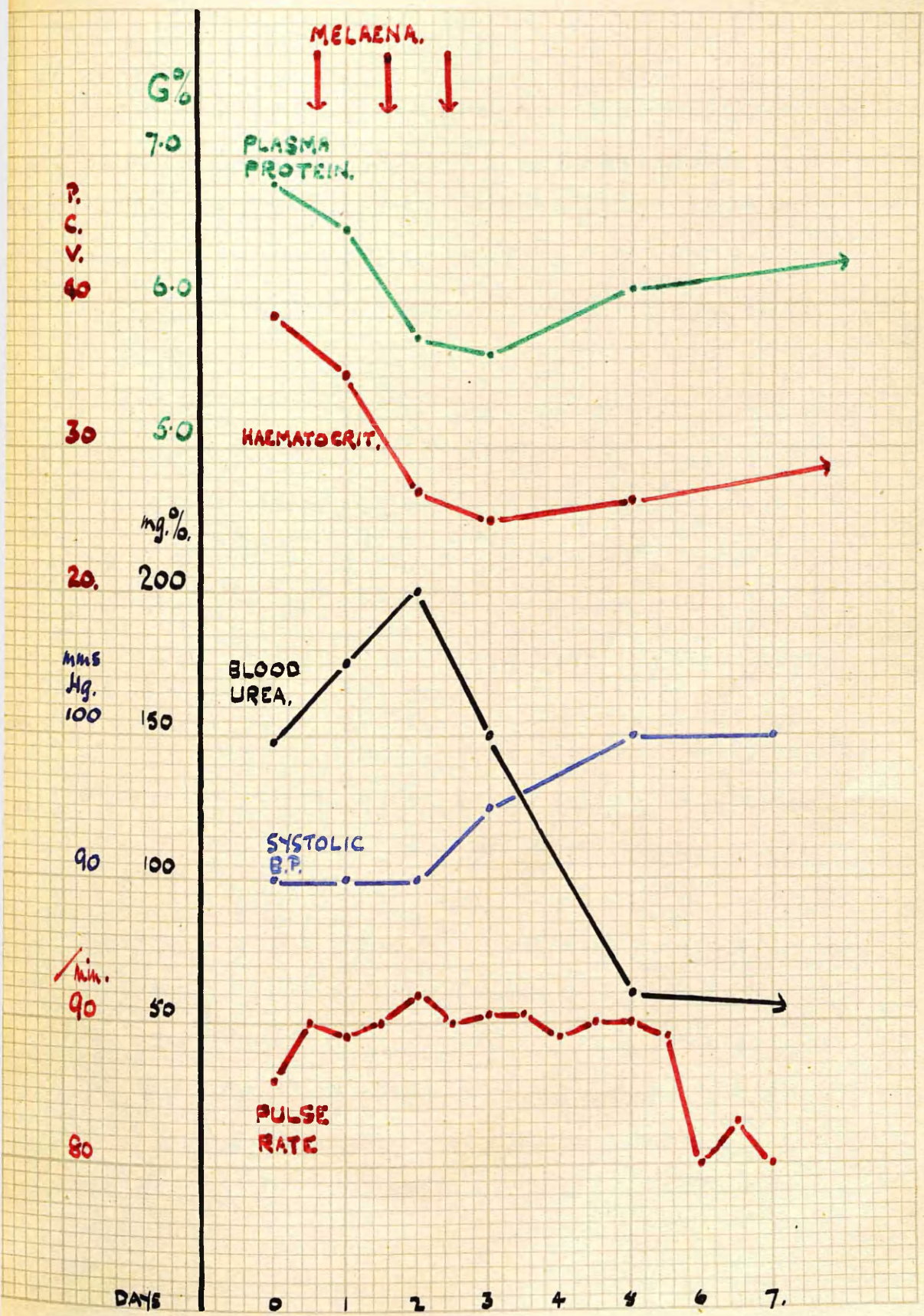
CASE No. 17. ♀ aet 75 ASPIRIN HAEMATEMESIS.



CASE No. 18. ♀ aet. 41. ACUTE ULCER.



CASE No. 22. ♀ aet. 51. DUODENAL ULCER.



There can never be certainty that bleeding will not occur and should this happen, knowledge of the plasma protein level before the recurrence will become significant.

Composite charts illustrating such cases are appended.

Ten cases fell into this group. The plasma proteins on admission ranged from 4.6 G% - 7.45 G%, three being under 5.0.G.% and eight under 6.0 G%. This means that the level of plasma proteins in the first specimen of blood examined has no prognostic value. A very low level does not mean that bleeding is still continuing.

In the second group, bleeding continued after admission. In all these cases, the plasma proteins show an initial fall and a subsequent rise, sometimes as in the first group, after remaining stationary for a short interval. The fact that restoration of plasma proteins is slower than had been anticipated has robbed the test of some of the value that was hoped for it, but the subsequent course of the cases in this group shows that at the point at which the plasma proteins ceased to fall, bleeding had ceased. The haematocrit meantime/

meantime continues to fall until haemodilution is complete and in these records, the plasma proteins have indicated cessation of bleeding at least 24 hrs. and sometimes several days before the haematocrit ceased to fall.

As would be expected, brisk haemorrhage is reflected in the pulse rate long before changes could be expected in the plasma. The majority of the cases illustrate this, particularly those in which there has been recurrent brisk bleeding, e.g. Nos. 4, 8, and 18. The plasma protein has shown occasionally that a rise in pulse rate has not been due to haemorrhage, by remaining steady or continuing to rise, as in Nos. 3 and 2.

In general, the blood pressure also reflects brisk haemorrhage promptly, but its response to slower haemorrhage is variable. Particularly deceptive is the tendency for blood pressure to rise on the day after the initial blood loss, the "hyperdynamic" response. In case No. 3, it can be seen that the blood pressure rose for the first 36 hrs. after admission during which the falling plasma proteins and haematocrit and rising blood urea, show that slow haemorrhage was continuing/

continuing. A similar state of affairs is seen in Nos. 5 & 8, at certain stages.

Thirteen cases come into this group and the initial plasma protein levels are, on the average, higher than in the first group. They range from 4.5 G% - 7.6 G%. Three cases had a first figure over 7.0 G% and eight were over 6.0 G%. These cases are those whose admission has followed promptly on their first bleeding and correspond to the cases with an initially high haemoglobin and in whom haemodilution has not had time to occur.

A normal plasma protein, therefore, in the first specimen of blood, cannot be taken to mean that haemorrhage has ceased and that the plasma proteins have been promptly restored. More commonly it means that haemodilution is yet to take place.

TABLE NO. 6

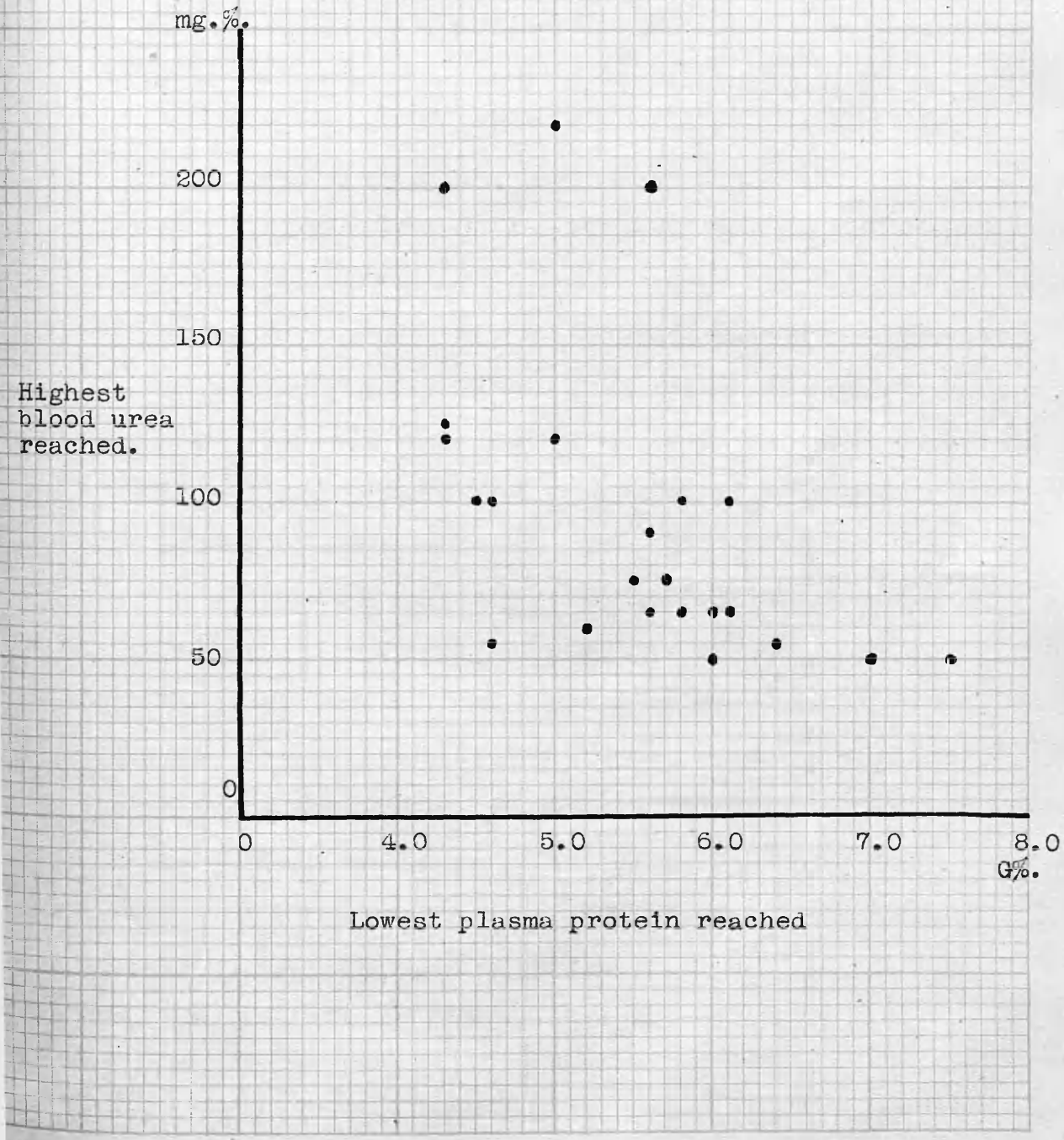
	Haemorrhage ceased on admission		Haemorrhage continued after admission	
	No. of Cases	% of Group	No. of Cases	% of Group
Initial Plasma Proteins 4-5 G %	3	30%	2	15%
5-6 G %	5	50%	3	23.5%
6-7 G %	1	10%	5	38%
7-8 G %	1	10%	3	23.5%

Death occurred in two cases. The first was a woman of 60 yrs. who was admitted in a moribund condition following haematemesis from oesophageal varices due to hepatic cirrhosis. She died 24 hrs. later. Three estimations of P.C.V. and plasma proteins during that time show that haemorrhage had not been severe and did not recur, but no conclusions can be drawn from the case. This is unfortunate, since no case of haematemesis with frank hepatic damage has been admitted since the investigation started. The behaviour of the plasma proteins would have been of great interest.

The second was a man aet 56 with chronic duodenal ulcer and a long history of dyspepsia. He has had a massive haematemesis followed by melaena two days before admission, but his condition then and for 24 hrs. thereafter was satisfactory, his plasma proteins rising and his pulse rate falling. On the second day there was a recurrence of bleeding which continued until death, in spite of transfusion on the third day. Points of interest in the present connection are that his pulse rate showed only a transient rise when bleeding recommenced and that the falling ~~of~~ plasma proteins gave a much truer picture of what was happening.

Plasma/

CHART No. 4.


mg.%.
200
150
100
50
0
Highest blood urea reached.
0
4.0
5.0
6.0
7.0
8.0
G%.
Lowest plasma protein reached

Plasma proteins, haematocrit and blood pressure all showed a steady decline until the point where transfusion was started. The very rapid and very high rise in blood urea, with resumption of bleeding in this case is in keeping with Black's statement that a secondary rise due to a repeated haemorrhage is often greater than the initial one.

Comparing blood urea levels with those of plasma proteins, in general, the more severe the bleeding, the lower the plasma proteins and the higher the blood urea, as would be expected, but the correlation is not very close as the accompanying chart shows.

The effect of a raised blood urea on plasma gravity has, in no case, been sufficient to confuse the picture. In case No. 8, the high values reached by the blood urea, may have contributed to raise the plasma specific gravity, but neither in this case nor in No. 11, in both of which the blood urea reached 200 mg%, did it disguise the fall in plasma protein due to bleeding.

When one compares the plasma protein levels reached with the commonly accepted criteria of severity - namely Haemoglobin less than 40% and Blood urea over 100 mg%, the following table can be prepared:-

TABLE/

TABLE NO. 7.

	Haemo-globin %	Blood Urea mg %	Plasma Protein G %	Plasma Protein in cases which fulfil criteria of severity.
1	75	55	6.4	
2	33	55	4.6 x	4.6
3	75	50	7.45	
4	30	65	5.6 x	5.6
5	40	80	5.6 x	5.6
6	65	50	5.95	
7	50	100	5.8 x	5.8
8	40	75	4.65 x	4.65
9	32	100	4.6 x	4.6
10	75	65	6.1	
11	20	200	4.3 x	4.3
12	90	50	7.0	
13	46	75	5.5	
14	22	100	4.5 x	4.5
15	45	60	5.05	
16	58	60	5.9	
17	25	228	5.0 x	5.0
18	21	120	5.0 x	5.0
19	55	75	5.7	
20	50	100	6.1 x	6.1
21	20	120	4.3 x	4.3
22	27	120	4.3 x	4.3
23	45	200	5.6 x	5.6
24	50	60	5.75	
			Average	4.9 G %

It would seem reasonable to accept a plasma protein level of 5.0 G% or less as indicative of a comparable severity to a haemoglobin of 40% or less and a blood urea of over 100 mg%. The correlation is fairly accurate with both. In the above table eleven cases had a haemoglobin of 40% or less and nine had a plasma protein of 5.0 G or less. Ten cases had a blood urea of 100 mg% or more and in seven the plasma proteins were 5.0 G or less. The use of this additional criterion of severity has certain advantages. The blood urea will tend to give first warning of danger in cases with latent renal damage or dehydration, the haemoglobin may pick out severe bleeding in an otherwise fairly fit individual, but the low plasma protein level will distinguish those with a previously poor nutritional state or latent liver damage.

CONCLUSIONS

(1) That restoration of lost plasma protein in cases of upper alimentary bleeding from ulcer is slower than in the normal individual and that this is probably due to previously poor protein intake and hepatic dysfunction.

(2) That a falling plasma protein indicates persisting or recurring haemorrhage.

(3) That a stationary or rising plasma protein indicates that haemorrhage has ceased.

(4) That the initial value for plasma protein after admission is of very limited prognostic value. A low value does not necessarily mean that bleeding is going on, nor a high one that it has ceased. More frequently the reverse is the case.

(5) That a plasma protein of 5.0 G% or less is indicative of a severity comparable to a haemoglobin of less than 40% or a blood urea of over 100 mg.% and that the plasma protein will tend to give first warning of that severity in cases of poor nutritional state or latent liver disease.

(6) That considering the simplicity and speed with which the estimation can be done, the plasma proteins are well worth while adding to the routine investigation of haematemesis and melaena.

CASE HISTORIES.

CASE NO. I.

Gastric Ulcer: Chronic. Admitted 30/12/47

J.S. Male - aet 42. Occupation: patternmaker.

The patient had a previous haematemesis on 1.3.47, which was preceded by only 10 days of mild dyspepsia. Thereafter he was completely well until 25/12/47, when slight post-prandial pain returned. He denies dietary or alcoholic excess, although his relapse occurred on Christmas Day. Dyspepsia persisted until haematemesis occurred on 30/12/47. He had noticed melaena two days earlier, 28/12/47.

Apart from appendicectomy when aged 5 yrs. he had no previous illness of note and there was no history of dyspepsia or other relevant feature in the family. He had had recent worry over the deaths of both parents and now lived alone, attending to his own needs. He smoked eight cigarettes per day and drank a half of whisky daily.

On admission, he was anaemic, but not shocked. Physical examination of the various systems was otherwise negative. Pain persisted until an antispasmodic mixture was given, but his progress was otherwise uneventful. Treatment was by bland diet, Aludrox and later/

later a bromide and belladonna mixture, iron and ascorbic acid.

Investigation: 17.1.48. Fractional Gastric Analysis: the acid curve shows definite hyperacidity but otherwise the result is normal.

5.2.48. Barium Meal examination shows a gastric ulcer on the lesser curvature of the stomach.

6.2.48. Gastroscopy: The ulcer is seen to have the typical appearance of a simple chronic ulcer.

21.2.48. Gastroscopy: The ulcer is smaller and the base granulating.

27.2.48. Barium Meal: The ulcer crater is no longer demonstrable.

12.1.48. Liver Function Tests:

Thymol turbidity Test - 0.5 McLaggan Units.

Serum Colloidal Gold Test - 000000

Hippuric Acid excretion Test - 0.13 G as Benzoic Acid.

	Haematocrit	Plasma Proteins G %	Blood Urea mg.%	B.P.
31/12/47	14.5	4.65	35.	90/50
1/1/48	18	4.9	50.	110/60
2/1/48	19	5.05	75.	120/65
3/1/48	21	5.4		105/65
4/1/48	24	6.4	60.	105/65
5/1/48	25	6.8	60.	105/65
7/1/48	29.5	6.8	50.	100/60
12/1/48	35.	6.8	50.	100/60
1/3/48		7.5		

sputum for a few days after admission showed blood tingeing. Treatment was with bland diet and ascorbic acid.

Investigation: X-ray of thorax 7.5.48, shows the presence of fibrotic tuberculosis in both lungs.

Sputa: repeatedly negative for tubercle bacilli.

Fractional Gastric analysis 15/5/49 shows a histamine fast achlorhydria with much excess mucus in all specimens.

Ba Meal Examination on 20/5/48 shows no abnormality of stomach or duodenum.

	Haematocrit	Plasma Proteins	B.P.
6.5.48	45	7.6	125/90
7.5.48	42	7.0	125/80
8.5.48	42	7.0	115/60
10.5.48	45	7.4	150/75
14.5.48	46	7.4	150/90

CASE NO. 3.

Duodenal Ulcer: Chronic. Admitted 21.5.48.

R.K. Male - aet 63. Occupation: Wood Sawyer.

The patient had had dyspepsia for the past 19 years. He was known to have a duodenal ulcer radiologically and had had haematemeses in 1931 and 1947. His symptoms had been severe for two months and he vomited blood on 18/5/48 and again on 21.5.48, the day of admission. Melaena had been present since 18/5/48.

He had had no illness other than his dyspepsia and, in the family, one brother was also dyspeptic. His circumstances were good. He smoked 2½ ozs. of pipe tobacco per week and drank very little alcohol.

On admission, he was noted to be a well nourished, well developed man, who was pallid and dehydrated but not shocked. Examination revealed no other systemic disease. Progress was uneventful. Treatment was by bland diet, Aludrox, iron and ascorbic acid.

Investigation: Fractional Gastric Analysis 12.6.48 shows a marked hyperchlorhydria but no evidence of delayed emptying.

Barium/

Barium Meal Examination 14/6/48 shows no abnormality of stomach. The duodenum shows scarring and an ulcer crater is present.

Liver Function Tests 7.6.48.

Hippuric Acid Excretion Test - 0.641 G as Benzoic Acid.

	Haematocrit	Plasma Proteins G%	Blood Urea mg.%
21/5/48	36	7.5	66.
22/5/48	31	6.1	75
23/5/48	25.5	5.5	-
24/5/48	22.5	5.75	45.
25/5/48	22.5	6.0	-
28/5/48	26.5	6.5	42.

CASE NO. 4

Duodenal Ulcer: chronic: Admitted 29.5.48

J.B. Male - aet 26 yrs. Occupation: Clerk

The patient had had remittent dyspepsia of ulcer type for the previous four years. Symptoms were aggravated following alcoholic excess on 22.5.48 and he had a moderately severe haematemesis on 28.5.48 at 11 p.m.

He had had no other illnesses and the family history was negative. His home circumstances were poor. He smoked 30 cigarettes per day and regularly drank whisky.

He was found to be a poorly developed young man, moderately well nourished. He was pale but not shocked - B.P. 110/70, pulse 96 per min. He was markedly tender in the epigastrium both to right and left of the midline but otherwise systematic examination was negative.

Haematemesis and the passage of loose tarry stools continued for three days after admission, by which time the patient was very anaemic but thereafter progress was steady and uninterrupted. Treatment was by bland diet, Aludrox, iron and ascorbic acid.

Investigation 20/6/48. Fractional Gastric Analysis showed a normal result.

24/6/48. Barium meal examination showed a normal/

normal stomach. There was a duodenal ileus with an ulcer crater in the second part of the duodenum.

	Haematocrit	Plasma Proteins G %	Blood Urea mg.%
29.5.48	29.5	4.7	57
30.5.48	22.5	4.5	60
31.5.48	19.5	4.8	50
1.6.48	14.5	4.65	65
2.6.48	14.5	5.4	50
3.6.48	14.5	5.4	46
6.6.48	17.5	5.7	-
9.6.48	22	5.4	28
13.6.48	24	5.4	-
17.6.48	26	5.75	28

CASE NO. 5.

Duodenal Ulcer: chronic: Admitted 6.5.48.

J. McN. Male - aet 23 yrs. Occupation - Hotel Handyman.

The patient had never had dyspepsia. He suffered a sudden bereavement by the death of his mother on 4.5.48 and on 5.5.48 he drank excessively. He fainted twice that night and on the following morning had a sudden moderately severe haematemesis.

He had had no other serious illness and the family history contained nothing relevant. He lived in the hotel in which he worked, smoked 10 cigarettes per day and drank usually 4 pints of beer per week.

On admission, he was seen to be a well built young man of good nutrition. He was markedly pallid and the tongue was dry and furred, but he was not shocked. B.P. 105/50, Pulse 100 per min.

His recovery was straightforward. The stools became negative for occult blood on 16.5.48. Treatment was with bland diet, iron and ascorbic acid.

Investigation: Fractional gastric analysis on 20.5.48 showed definite hyperacidity but the acid curve fell to normal levels at 2½ hrs. There was no delay in emptying.

Barium/

CASE NO. 6.

Peptic Ulcer: acute. Admitted 13.6.48.

J.M. Male - aet 33 yrs. Occupation: Miner.

This patient had no dyspepsia prior to the present illness. On 11th and 12th June 1948, he drank a considerable amount of alcohol, but not more than his usual. On 13.6.48, he felt faint and vomited blood. He then passed frank red blood by the bowel.

He had had no other illness of note and the family history was not relevant. He smoked 30 cigarettes per day and drank heavily of beer and whisky.

He was found to be a well built and well nourished man, who was not shocked. Beyond some epigastric tenderness, physical examination was negative. His recovery was uneventful. The stool became negative to occult blood on 19.6.48. Treatment was simply by bland diet and ascorbic acid.

Investigation: Fractional Gastric Analysis on 22.6.48 showed no abnormality.

Barium meal examination on 23.6.48 showed no abnormality of stomach or duodenum.

Gastroscopy on 25-6-48 was also negative.

CASE NO. 7.

Duodenal Ulcer: chronic. Admitted 9.11.48.

D. McC. Male - aet 42 yrs. Occupation: Crane Driver.

The patient gave a history of typical duodenal ulcer dyspepsia going back 20 yrs. He had a perforation in 1929 and an operation for "adhesions" in 1934. He had a haematemesis in 1940, and in 1946, while he was in hospital for treatment for his dyspepsia, a barium meal showed a duodenal ulcer.

On rising on 8/11/48, he felt faint and vomited about two pints of blood. He was admitted to another unit and transferred on 9.11.48.

He had had no other illness of note, but in his family, one brother had had a perforation and another dyspepsia.

On admission, he was seen to be a poorly nourished man, older than his years. He was not shocked and not grossly anaemic. Systematic examination was otherwise negative. His progress was steady. He received bland diet, iron and ascorbic acid.

Investigation: Fractional Gastric Analysis on 6.12.48 showed only a hyperchlorhydria.

Barium meal examination on 7.12.48

showed/

showed a small, very deformed duodenal cap with a large pseudo-diverticulum from the right fornix, the appearances being consistent with gross scarring.

	Haematocrit	Plasma Proteins G %	Blood Urea mg.%
9.11.48	24	5.8	100.
10.11.48	24	6.0	80.
11.11.48	24	6.0	60.
12.11.48	26	6.4	45.

Liver Function Tests, 11.11.48.

Thymol turbidity Test, 3.5 McLaggan unit.

Serum Colloidal Gold Test, 000000.

Plasma alkaline phosphatax Test, 1.1. Bodansky unit.

Hippuric acid excretion test, 0.3 G. as Benzoic acid.

CASE NO. 8.

Peptic Ulcer: acute; latent renal insufficiency.
Admitted 21.10.48.

J.A. Male - aet 56. Occupation: Civil Engineer.

The patient had no previous gastric trouble. For four days prior to admission he had a feeling of sickness and lack of appetite. This coincided with a business trip to London. After travelling back to Glasgow with the night train, the patient collapsed and had a severe haematemesis at 10.30 a.m. and another at 3.30 p.m. on 21.10.48, the day of admission.

He had had no previous illness of note and there was nothing relevant in the family history. He occupied a responsible post which involved a great deal of travelling to London and also overseas. He used tobacco and alcohol with moderation.

On admission, the patient was noted to be a well developed, well nourished man. He showed evidence of shock - B.P. 88/50, pulse 90/min. Physical examination of the systems was negative except for evidence of arteriosclerosis in the fundal vessels.

The patient had a very stormy passage. At first there was slight general improvement in his condition/

condition but on 23.10.48 he had a further haematemesis and became restless, shocked and showing cyclical respirations. Fluids were pushed orally and he slowly improved once more, but on 31.10.48, he had a sudden collapse with tachycardia, sweating and imperceptible pulse but without overt haemorrhage. It was decided that severe haemorrhage had restarted and blood transfusion was commenced. He received in all 4 pints of blood and 2 pints of glucose saline. Bleeding did not recur but he remained drowsy and weak, in a state suggesting incipient uraemia in spite of adequate fluids orally and an adequate urinary output. At this stage of the illness he was found to have developed a widespread cottonwool exudate in both fundi, which was attributed by the ophthalmologist to his anaemia. He also developed urinary retention without obvious cause and this was thought by the urologist to be due to bladder atony, the result of his poor general state. Renal function tests showed extremely poor function, but with gradual recovery this, the eye changes and the urinary difficulty all cleared up.

Investigation: Fractional Gastric Analysis 1.12.48 showed a moderate hyperacidity only.

Barium meal examination on 6.12.48 showed an irregularity/

irregularity of the mucosa in the region of the incisura but no definite ulcer was seen.

Gastroscopy on 11.12.48 showed a rugose but otherwise healthy mucous membrane. No lesion was seen.

10.11.48. Urea Clearance 10% and 14% of average normal.
Blood urea 47% mg.

30.11.48. Urea Clearance 28% and 34% of average normal.
Blood urea 50% mg.

28.1.49. Urea Clearance 40% and 60% of average normal.
Blood urea 49% mg.

4.3.49. Urea Clearance 73% and 100% of average normal.
Blood urea 32% mg.

Liver Function Tests. 2.12.48.

Thymol turbidity Test 5.0 McLaggan Units.

Serum Colloidal Gold Test 100000

Plasma alkaline phosphatase Test 2.2. Bodansky units.

Hippuric Acid excretion Test 0.33 G as benzoic acid.

	Haematocrit	Plasma Proteins	Blood Urea
21.10.48	39	5.7	35
23.10.48	28	5.4	60
24.10.48	18	5.2	100
25.10.48	17	6.4	228
27.10.48	13	6.0	
28.10.48			89
31.10.48	16	5.75	
	Transfusion		
1.11.48	20	5.0	80
2.11.48	22	6.5	200
4.11.48	22	5.4	90
8.11.48	23	5.6	80
21.11.48	30	6.1	60
14.12.48	41	6.0	50

CASE NO. 9.

Gastric Ulcer: chronic. Admitted 6.12.47.

J.B. Male - aet 45 yrs. Occupation: Barman.

The patient gave a history of dyspepsia for very many years, but not of typical ulcer type. He complained of epigastric discomfort at variable intervals after food, of flatulence and of nausea and sickness in the morning, when he brought up a little clear fluid.

He had haematemeses in 1929, 1934 and 1937. He had several haematemeses during the four days before admission.

There was no history of other serious illness or of dyspepsia in the family. He smoked 20 cigarettes per day and was adamant that he drank alcohol only rarely although he was a barman.

On admission he was very anaemic and shocked - B.P. 95/45, pulse 100 per min. He was a poorly nourished man, rather older than his years. For the first few days his progress was satisfactory, bleeding evidently having ceased. Further personal observation of this patient was not possible and the record is therefore incomplete. At a later date he had a recurrence/

recurrence of bleeding which was ultimately fatal, the autopsy finding being a chronic gastric ulcer.

	Haematocrit	Plasma Protein	Blood Urea
		G%	mg.%
6.12.47	11	4.6	40.
7.12.47	9	4.6	45.
9.12.47	12.5	5.0	40.

CASE NO. 10.

Duodenal Ulcer: chronic: Admitted 20.11.48.

J.K. Male - aet 33 yrs. Occupation - Painter.

The patient admitted to absolutely no previous dyspepsia. He felt faint and perspired freely on the evening of 16.11.48 and this state persisted on 17.11.48 and 18.11.48. He noticed melaena on 18.11.48 and it was still present on admission, 20.11.48.

He had had no other illness of note and there was nothing relevant in the family history. His home circumstances were good and he was a non-smoker and a teetotaller.

On admission he was seen to be a well developed and well nourished man. He was very pale, but not shocked, B.P. 100/50, pulse 72/min. Examination of the systems was negative. His recovery was completely uneventful. Treatment was by bland diet, iron and ascorbic acid.

Investigation: Barium Meal 15.12.48 - the stomach was normal. The duodenal mucosa was swollen and irregular and an ulcer crater was demonstrable.

Fractional gastric analysis on 1.12.48 showed

a/

a rapidly emptying stomach with definite hyperacidity and excess mucus in the first few specimens.

Liver function Tests 29.11.48.

Thymol turbidity tests 1.0 McLaggan units.

Serum Colloidal Gold Test 000000

Plasma alkaline phosphatase Test 0.7 Bodansky units.

Hippuric Acid Excretion Test 0.33 G as Benzoic acid.

	Haematocrit	Plasma Proteins G%	Blood Urea mg%
20.11.48	20	5.4	90
21.11.48	17.5	5.4	75
22.11.48	18	5.8	-
23.11.48	20	6.1	50

CASE NO. 11.

Duodenal Ulcer: chronic. Admitted 23.12.48.

R.G. Male - aet 56 yrs. Occupation: Tram Driver.

Prior to 1923, the patient was working in a shipyard and suffered from flatulent dyspepsia, but in that year he took work as a tram driver and his dyspepsia cleared up. In recent months, he had some flatulence if long without food, but paid little attention to it. At no time had he pain.

On 19.12.48, he felt sick, "as if something had disagreed with him". He had a massive haematemesis on 20.12.48 and noticed melaena since then. A smaller haematemesis occurred on 22.12.48.

Apart from a renal calculus, which he passed in 1934, he had been healthy and there was nothing of note in the family history. His home circumstances were good, but his work entailed varying shifts and irregular meals. He smoked 4 cigarettes per day and was teetotal.

On admission he was pale and shocked, B.P.85/45 pulse 120/min. He was poorly nourished and dehydrated. He improved for 24 hours after admission, but thereafter bleeding recurred and was severe and continuous. Death/

Death occurred on the fourth day in spite of transfusion.

Investigation:

Liver Function Tests 24.12.48.

Thymol turbidity Test 1.0 McLaggan Units.

Serum Colloidal Gold Test 000000

	Haematocrit	Plasma Proteins	Blood Urea
23.12.48	22	5.9	125.
24.12.48	21	6.2	115.
25.12.48	15	5.05	-
26.12.48	12.5	4.30	200.

Autopsy 27.12.48. showed a large chronic duodenal ulcer, measuring 1½" in its longest diameter with a fibrotic base and an eroded and pouting vessel. The ulcer base was adherent to pancreas.

CASE NO. 12.

Duodenal Ulcer: chronic. Admitted 27.1.49.

R.K. Male - aet 63 yrs. Occupation: Wood Sawyer

This individual is the same as Case No. 3. He was discharged well in June 1948 and adhered strictly to his regime. He had no dyspepsia. After a meal of steamed fish and potato on 26.1.49 he felt distended. He had a small haematemesis at 3 a.m. and another large one at 7 a.m. on 27.1.49.

He gave the impression of not being so well generally as on the previous admission. He appeared ill nourished in comparison. Blood loss was not severe and he was not shocked.

His recovery was uneventful on routine treatment.

Investigation: Liver function Tests 7.2.49.

Thymol turbidity test, 2.0 McLaggan Units.

Serum Colloidal Gold Test, 000000

Hippuric Acid excretion test 0.34 G as Benzoic acid

	Haematocrit	Plasma Proteins	Blood Urea
27.1.49	35	5.95	55
28.1.49	31	6.1	55
29.1.49	30	6.4	50
31.1.49	30	6.4	50
7.2.49	40	6.8	40

CASE NO. 13.

Duodenal Ulcer: chronic. Admitted 9.1.49.

J.B. Male - aet 22 yrs. Occupation: Radio Mechanic.

The patient had slight dyspepsia for many years, but for the past five years, he had epigastric pain two hours after food. He had remissions. A duodenal ulcer was diagnosed radiologically in August 1948. Since then pain had been very severe and associated with vomiting which afforded relief.

He had a small haematemesis on 7.1.49 and again on 9.1.49. Melaena started on 8.1.49.

He had had no other serious illness. He was an only child and his father suffered from duodenal ulcer. His circumstances were good. He owned his business, was a non-smoker and a teetotaller.

He was a well set up and well nourished lad, who was extremely pale but not shocked. Bleeding had evidently been going on insidiously for some time. The systems were negative. His recovery was uneventful on routine treatment.

Investigation: Fractional Gastric Analysis 10.2.49. There is a high fasting juice volume and acidity and a climbing type of acid curve, with slow emptying.

Barium/

Barium Meal Examination 12.2.49. The Duodenal Cap is spastic and deformed with associated pylorospasm and gastritis.

	Haematocrit	Plasma Proteins	Blood Urea
9.1.49.	19.5	5.6	80
10.1.49.	18	5.6	55
12.1.49	18	5.6	40
17.1.49	23	6	
26.1.49	31	6.4	40

CASE NO. 14.

Peptic Ulcer: acute. Admitted 21.1.49.

G. McG. Male - aet 56 yrs. Occupation: Dairyman.

The patient had no dyspepsia before the present illness. He began to feel epigastric pain before meals one week before admission. To relieve this, he took a dose of salts on 16.1.49 and on 18.1.49 he had a haematemesis which has been repeated daily since. He has noticed melaena since 20.1.49.

He had pneumonia and empyema in 1915 and had been subject to bronchitis since. There was nothing of note in the family history. He and his wife ran a small dairy business. The work was hard and the hours long. He smoked 3½ ozs. of pipe tobacco per day and had a glass of beer and a whisky regularly each day.

On admission he was found to be very pale, but not restless or uncomfortable. B.P.115/55, Pulse 110/min. He showed evidence of weight loss. Bleeding continued for the next 48 hours and he had a small "coffee ground" sickness on 23.1.49. Thereafter improvement was steady.

Investigation: Fractional Gastric Analysis 14.2.49. Showed/

Showed a definite hyperacidity but no other abnormality.

Barium meal examination, 22.2.49, showed "some elongation of the pylorus and a pressure effect, suggestive of some hypertrophy of the pylorus. There are rather coarse rugae in the descending part of the duodenum, suggestive of duodenitis".

	Haematocrit	Plasma Proteins	Blood Urea
21.1.49 a.m.	26	5.6	40
p.m.	22	5.4	60
22.1.49	18	5.0	120
23.1.49	14	5.5	85
26.1.49	16	5.75	75

Liver Function Tests. 28.1.49.

Thymol turbidity test, 2.6 McLaggan Units.

Serum Colloidal Gold test, 100000.

Plasma alkaline phosphatase test, 1.2 Bodansky units.

Hippuric Acid Excretion Test, 0.23 G.as benzoic acid.

CASE NO. 15.

Duodenal and Gastric Ulcers. Admitted 29.1.48.

M.C. Female - aet 54 yrs. Occupation: Cleaner.

The patient had dyspepsia since 1943. She did not associate her epigastric pain with food, but it was relieved by alkali and by vomiting. She had long remissions. There had been some weight loss. A Barium Meal examination in Dec. 1946 was negative.

Moderately severe haematemesis, causing her to faint, occurred on 28.1.48 and again on 29.1.48.

She had had an emergency appendicectomy in 1937 and a cholecystectomy, also as an emergency in 1938. She had had pneumonia in 1945 and in 1947 and suffered for some years from rheumatoid arthritis of hands and feet. Her father died of haematemesis. She did not smoke or take alcohol, but her hours were irregular and she required to carry her food.

On admission, the patient was extremely anaemic, dehydrated and somewhat shocked. She made a steady recovery, however, on routine treatment and without transfusion.

Investigation:

27.2.48. Barium meal examination showed a normal stomach/

stomach. There was deformity of the duodenal cap consistent with ulcer, but no crater was shown.

6.3.48. Gastroscopy showed a healing, shallow lesser curvature ulcer, probably an acute ulcer and the cause of her haematemesis.

Liver Function Tests:

9.2.48. Hippuric Acid Test - 0.646 G as benzoic acid

18.2.48. Serum colloidal Gold Test - 000000

Thymol turbidity Test - 1.0 McLaggan Units.

	Haematocrit	Plasma Proteins G%	Blood Urea mg%
29.1.48	15	5.6	45
30.1.48	18	6.5	65
2.2.48	18	6.5	-
4.2.48	18	6.7	55
16.2.48	20	7.5	50

CASE NO. 16.

Gastric Ulcer: acute. Admitted 26.2.48.

M.D. Female - aet 20 yrs. Occupation: Shop Assistant.

The patient had dyspepsia for one year but pain, which occurred ½ hour after food was not relieved much by alkali or by more food. She felt nauseated but was not actively sick. The attacks of dyspepsia each only lasted 3-4 days as a rule and she had long remissions. The present attack, however, had been more severe than usual and had lasted two weeks. The history was thought suggestive of recurrent acute ulcer. She had a haematemesis shortly after a visit to the outpatient department for investigation on 26.2.48 and on questioning it seemed likely that she had had melaena for a few days before.

She had had a ruptured appendix with peritonitis five years before, but otherwise her previous history was negative, as was the family history. Her meals were irregular and were carried. She smoked a few cigarettes per day but drank no alcohol.

She was a well nourished, well built girl. She showed no pallor on admission and was not shocked, B.P.105/55, pulse 76/min. Her recovery was uneventful.

Investigation/

CASE NO. 17.

Aspirin haematemesis. Admitted 26.2.48.

A.I. Female - aet 75 yrs. Occupation: at Home.

Eighteen months ago, the patient began to have nausea and flatulence after food, but they were not severe. She had no pain. In December 1946, she had a haematemesis and was admitted to this ward but investigation was negative. She awoke on 26.2.48 feeling sick and thirsty and shortly after, she vomited a fair amount of blood and passed several loose, melaena stools. Later she vomited a little "coffee grounds" and clots on several occasions. She had been taking aspirin in large doses for the relief of the pain of rheumatoid arthritis for some years. She had also been known to have a hypertension for some years.

There was no point of note otherwise in the past history, family history and social circumstances.

On admission she presented extreme pallor but B.P. was 120/80 and pulse 92 per min. She was a stoutly built woman who looked her 75 years. Beyond evidence of arteriosclerosis, systematic examination was/

was negative.

Her recovery was complicated by the onset of Auricular fibrillation on 6.3.48. This was controlled by digitalis and was still present on discharge. It was not considered advisable to attempt to restore normal rhythm with quinidine.

Investigation:

Barium Meal Examination 6.4.48 showed no abnormality.

Liver Function Tests, 19.3.48.

Thymol turbidity test, 1.0 McLaggan Units.

Serum Colloidal Gold Test, 000000

Hippuric Acid excretion test 0.337 G as Benzoic acid.

	Haematocrit	Plasma Proteins	Blood Urea
26.2.48a.m.	29	6.5	85
" p.m.	26	6.1	85
27.2.48	24	6.4	100
28.2.48	21	6.4	80
1.3.48	23	6.8	65
3.3.48	24	6.8	
5.3.48	25	6.8	
8.3.48	29.5	6.85	65
14.3.48	31	6.9	
18.3.48	31.5		
23.3.48	38		

CASE NO. 18.

Gastric Ulcer: acute. Admitted 1.4.48.

M.B. Female - aet 41 yrs. Occupation: Blocker in printing works.

The patient gave a history of remitting dyspepsia for 15 yrs. The attacks were short and she had no pain but a sense of fullness half an hour after food. The present attack had lasted three weeks.

She felt dizzy on the evening of 31.3.48 and during the night and the following morning she had loose tarry stools. A haematemesis occurred at 11 a.m. on 1.4.48.

She gave no relevant previous or family history and her social circumstances were favourable. She neither smoked nor took alcohol.

On admission, her general condition was good, B.P. 138/75, pulse 110 per min. Systematic examination was negative. Haematemesis and melaena persisted during the first day in the ward and recurred again 3 days later, but after that her progress was steady.

Investigation:

22.4.48 - Fractional Gastric Analysis gave a normal result.

30/4/48/

30/4/48 - Barium Meal Examination showed no lesion of stomach or duodenum.

5/5/48 - Gastroscopy showed a normal stomach.

15/4/48 - Liver Function Test:

Thymol turbidity 1.5 McLaggan units.

Serum Colloidal Gold, 200000

Hippuric Acid Excretion Test, 0.392 G as Benzoic Acid.

8/5/48 - Hippuric Acid Excretion Test, 0.591 G as Benzoic Acid.

	Haematocrit	Plasma Proteins G %	Blood Urea mg.%
1/4/48	32	7.4	35
2/4/48	25	5.75	80
3/4/48	24	5.75	50
4/4/48	22	5.75	50
5/4/48	12	4.3	100
6/4/48	10	4.6	120
8/7/48	10.5	5.0	60
11/4/48	11	5.6	55

CASE NO. 19.

? Hepato-lienal fibrosis. Admitted 23.12.48

J.P. Female - aet 65 yrs. Occupation: at Home.

Eight years ago the patient attended the Glasgow Royal Infirmary Dispensary with ulcer symptoms, but no ulcer was found radiologically. The symptoms cleared up and did not recur. Although still symptom free, she had a melaena in 1947 and was investigated, again with negative result, at the Southern General Hospital. She had symptoms referrable to hypertension for the past six months.

On 22.12.48, the patient attended an "Old Folks Treat" and the following morning she felt tired and without appetite. She took no food and slept most of the day. A haematemesis occurred at 5.15 p.m. on the 23.12.48 and haematemesis and melaena recurred frequently.

Previous illnesses, family history and social circumstances provided no relevant information.

When admitted, the patient was acutely ill, with greyish pallor, marked cyanosis and only semi-conscious. She had been given Morphine gr. ½ by her own doctor before admission. She was an obese woman and had/

had some oedema of the ankles. Pulse 84 per min. B.P. 80/50. There was cardiac hypertrophy, poor heart sounds and a systolic murmur at the apex. Examination of the abdomen was rendered difficult by obesity but hepatic and splenic enlargement were considered to be present.

Vomiting of coffee ground material and the passage of melaena stools continued although the blood findings suggested that no fresh bleeding was occurring. The patient became restless and very unco-operative and died on the day after admission.

	Haematocrit	Plasma Proteins G %	Blood Urea mg.%
23.12.48	41	6.1	60.
24.12.48 10 a.m.	36	6.45	65
" 1 p.m.	30	6.45	75

CASE NO. 20.

Duodenal Ulcer: chronic. Admitted 9.1.49.

C.S. Female - aet 46 yrs. Occupation: Clerkess.

The patient gave a typical duodenal ulcer history going back for 3 years. Symptoms were aggravated following a bereavement in October 1948 and became worse still following an attack of "influenza" commencing 30/12/48. On 6/1/49 she passed three large melaena stools and the stools remained black on the following days.

She had had no prior illness of note and her social circumstances were good. One sister had dyspepsia.

She was an obese woman, but was not acutely ill at any time. On admission, the pulse was 80 per minute and the B.P. 150/80. Her recovery was uneventful.

Investigation:

18.1.48. Fractional Gastric Analysis was quite normal.

25.1.48. Barium Meal examination showed no abnormality.

	Haematocrit	Plasma Proteins	Blood Urea
9.1.49	39	7.45	50
10.1.49	37	7.55	45
12.1.49	35	7.55	40
14.1.49	32	7.55	
17.1.49	33	7.55	
19.1.49	35	8.5	

CASE NO. 21.

Peptic Ulcer: acute. Admitted 23.9.48.

S.R. Female - aet 20 yrs. Occupation: Mill worker

The patient had been under treatment from her own doctor for a year for anaemia. She admitted to taking a very poor dietary, mainly of tea and carbohydrate. Three weeks before admission she began to have epigastric pain within five minutes of taking food, with a heavy feeling in the epigastrium. This was relieved by alkalies. She had a severe haematemesis at 7.30 a.m. on 23.9.48, the vomitus containing many large blood clots. Another occurred that evening before admission.

She had had no serious ill health otherwise and her family history and social history were clear. She was very pallid and slightly shocked on admission, B.P. 95/50, pulse 120 per min. Haematemesis recurred twice in the first 24 hours after admission, but thereafter slow progress was made. Her plasma proteins were particularly slow in their recovery in spite of a normal hippuric acid excretion. The serum colloidal gold reaction, however, was more abnormal than that of other cases in the series.

Investigation/

Investigation:

9.11.48. Fractional test meal showed a normal result.

29.10.48. Barium meal examination was negative.

Liver Function Tests 21.10.48:

Thymol turbidity test, 1.1 McLaggan units.

Serum Colloidal Gold Test, 320000

Plasma alkaline Phosphatase test 3.3 Bodansky units.

Hippuric Acid Excretion Test 0.76 G as Benzoic Acid.

	Haematocrit	Plasma Proteins G %	Blood Urea mg%
24.9.48	16	4.5	45.
25.9.48	12.5	4.3	66.
26.9.48	11	4.6	50.
27.9.48	11	4.8	50.
28.9.48	11	5.0	40.
30.9.48	12	5.0	35.
3.10.48	13	5.0	-
7.10.48	19	5.4	-
12.10.48	23	5.4	-

CASE NO. 22.

Duodenal Ulcer: Chronic. Admitted 18.3.48.

A.D. Female - aet 57 yrs. Occupation: at Home.

This patient had had a Gastric Ulcer 15 yrs. ago but symptoms disappeared and she had no further dyspepsia until three weeks before admission, when post.prandial pain returned. A sudden moderately severe haematemesis occurred at 7 p.m. on 18.3.48.

She was subject to chronic bronchitis which had rendered her unfit for work outside the home for the past two years. One brother suffered from dyspepsia. Her circumstances were good.

The patient was pale but not acutely ill, B.P. 90/70, pulse 88/min. Beyond obesity, systematic examination was negative. No further haematemesis occurred but melaena persisted and the blood findings indicated that oozing continued for three days.

Investigation: 4.4.48. Fractional Gastric Analysis showed an achlorhydria which was not histamine fast.

20.4.48. Barium Meal Examination revealed duodenal scarring but no active ulcer/

ulcer was demonstrated.

Liver Function Tests: 14.4.48.

Thymol turbidity test, 1.5 Units.

Serum Colloidal Gold Test, 200000

Hippuric Acid Excretion Test, 0.549 G as benzoic acid.

	Haematocrit	Plasma Proteins G%	Blood Urea mg%
18.3.48	39	6.8	148
19.3.48	35	6.5	175
20.3.48	27	5.75	200
21.3.48	25	5.65	150
23.3.48	26.5	6.1	60
31.3.48	31	6.7	50

CASE NO. 23.

Peptic Ulcer: acute. Admitted 9.1.49.

E.L. Female - aet 48. Occupation: at Home.

There was no history of previous dyspepsia whatsoever and no obvious precipitating cause for haematemesis. She suddenly felt weak and sick on the evening of 8.1.49, but did not have active sickness till 2 p.m. on 9.1.49. Haematemesis and melaena recurred during that day.

She had had considerable worry owing to the fact that her husband had been unfit for work for eighteen months with central nervous disease. Otherwise there was no relevant feature in previous health, family history or circumstances.

A thin, worried looking woman, she was not seriously ill on admission, B.P. 130/90, pulse 96/min. and her recovery was rapid and uninterrupted.

Investigation: 2.2.49. Fractional Gastric Analysis showed an acidity in the upper limits of normal.

31.1.49. Barium Meal examination showed a normal stomach and duodenum.

	Haematocrit	Plasma Proteins G%	Blood Urea mg.%
9.1.49	33	6.1	60
10.1.49	29	5.75	45
12.1.49	28	5.75	40
14.1.49	28	6.1	40
17.1.49	29	6.5	
19.1.49	29	7.0	

CASE NO. 24.

Peptic Ulcer: acute. Admitted 31.10.48.

M.S. Female - aet 29. Occupation: Engineer.

The patient had had no dyspepsia prior to 26.10.48 when she was sick without apparent cause. Thereafter she felt better, but had no appetite. Severe haematemesis occurred on the morning of 31.10.48 but bleeding had probably been going on insidiously for a few days.

She had had appendicitis and peritonitis in 1935. She had been a widow for two years and had a stepdaughter suffering from tuberculosis. Her work involved carried meals but she enjoyed it and did not find it heavy. Her living conditions were crowded but she had no financial worry.

Her general condition on admission was fairly good although she was pallid. B.P. 105/40. Pulse 104/min. Examination of the systems was negative and progress was rapid and uneventful.

Investigation: 5.11.48. Gastroscopy showed a healthy looking mucose and an active stomach, but there were two or three flecks of adherent/

adherent, coagulated mucus on the lesser curvature which showed faint areolae and were evidently healing erosions.

25.11.48. Fractional Gastric Analysis showed no abnormality

20.11.48. Barium Meal Examination showed no abnormality.

8.11.48. Liver Function Tests:

Thymol turbidity Test, 4.3 McLaggan Units.

Serum Colloidal Gold Test, 000000.

Hippuric Acid excretion test, 0.26 G as Benzoic acid.

	Haematocrit	Plasma Proteins	Blood Urea
31.10.48	16.5	4.6	35
1.11.48	17	5.4	55
3.11.48	17	5.8	45
4.11.48	17	5.8	40
6.11.48	18	5.8	40
9.11.48	20	6.0	35

CASE NO. 25.

Peptic Ulcer: acute. Admitted 18.11.48.

F.G. Female - aet 41 yrs. Occupation: Cash girl (part time)

The patient had experienced dyspepsia for one week only before admission. She then began to have a constant epigastric ache which was relieved for about half an hour by taking food. The pain gradually got worse until at 11.30,a.m. on 18.11.48 she suddenly vomited about a pint of fresh blood stained vomitus. Pain was immediately relieved. A further haematemesis occurred at 3.30 p.m.

In the past, she had a renal carbuncle operated on in 1941 and since 1937, she had had mild rheumatism in hands and feet.

There was no history of dyspepsia in her own family, but her husband suffered from a duodenal ulcer and the patient was much concerned about his health. She was a non-smoker and a teetotaller and her home circumstances were good.

The amount of blood loss must have been slight. Her condition was very good on admission. She had no recurrence of bleeding and no elevation of pulse rate or/

or reactionary pyrexia.

Investigation: 29.11.48. Fractional Gastric Analysis showed a slight hyperacidity. Maximal at 1¼ hours.

6.12.48. Barium Meal examination was completely negative.

	Haematocrit	Plasma Proteins	Blood Urea
18.11.48	37	6.4	55
19.11.48	37	6.6	40
20.11.48	37	6.8	35

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