BIOCHEMICAL PROBLEMS IN COLONIC OCCLUSION.

A THESIS

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BY

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I N D E X.

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BIOCHEMICAL PROBLEMS IN COLONIC OCCLUSION.

CHAPTER 1.

INTRODUCTION.

Intestinal occlusion is one of the commonest forms of surgical emergency and much has been written on its problems, but investigations tend to be divided into

(a) Work on intestinal strangulation,

(b) Simple obstruction below the Ampulla of Vater,

(c) Low ileal closed loop obstruction.

Very little seems to have been written about acute obstruction of the colon and the author has not been able to trace any literature concerning biochemical problems specifically dealing with this type of alimentary occlusion. Therefore he considered that the biochemistry of this condition was well worth some investigation, particularly in view of its relative frequency, and that the results might be compared with those obtained by other workers on blockage of the upper part of the alimentary canal.

In order to do this, 40 consecutive cases of obstruction of the colon admitted to Glasgow Royal Infirmary and Glasgow Victoria Infirmary were examined before operation and for three days after operation, and a chemical analysis of their blood made at approximately 24 hour intervals.

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CHAPTER 2.

INCIDENCE.

Colonic occlusion is a frequent form of intestinal blockage. Thus, out of a total of 474 cases of acute mechanical intestinal obstruction admitted to Croydon General Hospital during the years 1931-8 and to the Royal Sussex County Hospital, Brighton, during the years 1932-8, the author found 112 cases of colonic occlusion. These figures compare with the 951 cases of colonic obstruction out of 6,892 cases of acute intestinal occlusion investigated by Vick (156) from 21 of the largest voluntary hospitals in Great Britain.

	Total Cases	Colonic Obs.	%
Author	474	112	23
Vick	6,892	951	14

Of 3 other series investigated by the author, one was not sufficiently differentiated to be of service (9), one showed 8 cases of colonic obstruction out of a series of 25 cases of intestinal occlusion (48) and the third, a series of 241 cases of intestinal obstruction, did not include those with blockage of the colon "because the results were so bad" (51). Incidentally, the appalling death rate in obstruction of the colon has also been emphasised by Austin (9) and by Shier (140).

By far the most frequent cause of colonic obstruction is carcinoma, there being 106 cases in the author's series and 895 in Vick's series, and so, approximately 19 out of every 20 patients admitted to hospital with occlusion of the colon suffer from carcinoma. In the series under investigation, no patient suffered from any cause of obstruction other than carcinoma.

	Colonic Obs.	Ca. of Colon	%
Author	112	106	95
Vick	951	895	94

In clinical practice, there are only two other likely causes of occlusion of this part of the alimentary tract,

1. Congenital abnormalities.
2. Volvulus of the caecum or sigmoid.

As has been shown by the above figures, both of these conditions must be extremely rare and neither the author's nor Vick's figures show any cases of congenital obstruction of the colon. This may also be due to the fact that children are sent to special children's hospitals in most of Vick's cases, but it is not true of the cases at Croydon.

In regard to volvulus, it must be pointed out that this condition, although forming a block to the passage of faeces, causes symptoms of strangulation of the bowel and the pathology is quite different to that of ordinary obstruction of the bowel (2) (4) (5) (6) (100) (118). On the other hand, the

majority of patients who are acutely obstructed from a cancer of the colon are not ill from the carcinoma but from the mechanical effects of blockage of the lumen of the gut. (139).

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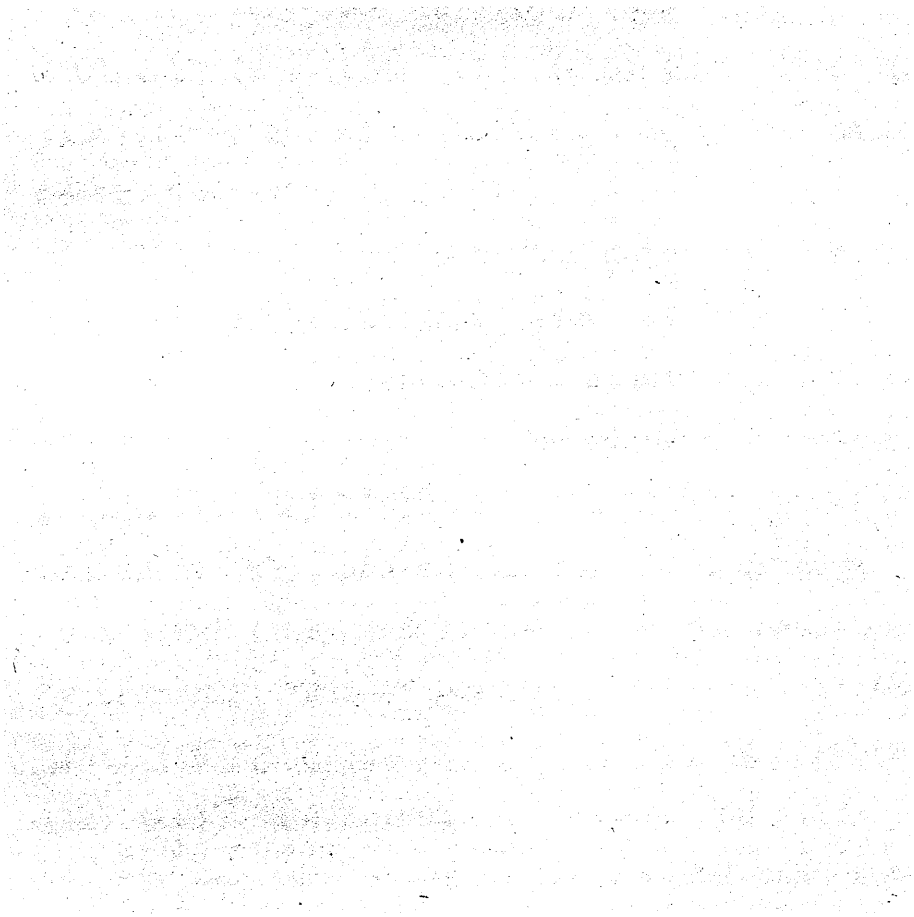

CHAPTER 3.

BIOCHEMICAL CHANGES IN INTESTINAL OBSTRUCTION.

(a) Dehydration.

Dehydration is one of the most important biochemical changes occurring in intestinal obstruction. This fact has been stressed by many authors (8) (10) (30) (50) (52) (56) (58) (63) (66) (67) (68) (69) (70) (75) (76) (79) (80) (91) (95) (104) (112) (113) (124) (146) (160) (164) (165) (168) and some think that it is the main cause of death. (56) (88) (154) (161).

It bears comparison with water loss occurring not only in the physiological state of sweating which may become serious under certain conditions (24) (25) (99) (109) (147), but also in the excessive vomiting of pregnancy, (40) (69) (81) (82), the cyclical vomiting of children, (87), acute vomiting and diarrhoea, (19) (23) (85) (132) (136) (137), and with that occasioned by the experimental loss of gastric (37) (42) (53) (86) (103) (132) (158), duodenal, (53) (157), pancreatic, (37) (46) (53) (86), and intestinal juices. (38) (93).

It has been shown by Church (22) that water loss up to 10% leads to serious illness and up to 20% to death. If the average volume of digestive fluids secreted by an adult man in 24 hours are as follows:- (105)

Saliva	....	1,500 c.c.
Gastric	....	2,000 c.c.
Bile	....	500 c.c.
Pancreatic	....	500 c.c.
Enteric	....	3,000 c.c.
		7,500 c.c.

then their complete loss would be much greater than the total blood volume, which may be taken as 6,000 c.c. in the average individual. (171). Fortunately, this total loss does not usually occur, for some of the juices seem to be reabsorbed and it appears probable that, as mechanical distension of the bowel causes a fall in blood pressure, (6) (59), there may be a lessened secretion of juices, although Herrin (93) says that increased intra-intestinal pressure causes a true succus entericus to be formed, thus aggravating the dehydration. That the pressure in the lumen of the gut is increased in intestinal obstruction has been shown by Owings and McIntosh (125). Even if there be an occlusion below the Ampulla of Vater at the lethal line of Draper Maury, (117), a considerable proportion of the intestinal juices may be reabsorbed at a lower level and thus death may be postponed.

Total loss may occur, however, not due to the obstruction but during its treatment. The action of contiuous duodenal or intestinal suction, beneficial in regard to the vomiting and to the distension of the bowel, may cause a serious negative water balance in the body. (1) (13) (14) (92) (126) (131) (159) (160).

This water loss tends to cause a blood volume reduction, but the fluid reserves in the tissues are able to counteract this for a time, (5) (134), and variation in the one gives an immediate response in the other. (134). These fluid reserves are very considerable and may amount to as much as 20%-25% of the body weight (36) (84) or 13 litres - more than twice the total blood volume. Thus dehydration is not necessarily accompanied by blood volume reduction, but, if it does occur, there may follow a lowered blood pressure with consequent lessened digestive secretions. The beneficial effect of isotonic salines (11) (15) (25) (32) (48) (57) (65) (67) (69) (71) (112) (140) (142) (159), is at least partly due to the fact that they increase the extra-cellular tissue fluids (89).

The matter is further complicated by the fact that water metabolism in the human body is also regulated, apart from fluid intake, by

1. The pituitary gland controlling its excretion.
2. Salt metabolism, which is intimately associated with that of water in order to keep up the very constant osmotic pressure of the blood.
3. The plasma proteins which are the main factors in controlling normal blood volume. (119) (143) (148).

(b) The plasma proteins.

As has been said previously, there is a definite relationship between the plasma volume and the plasma proteins, and any alteration in the latter will tend to cause a corresponding increase or decrease in the former, provided that there are ample fluid reserves in the tissues. As the extracellular tissue fluids are normally twice the blood volume in quantity, it is obvious that there is ample reserve for all normal contingencies.

It has been shown, however, that the plasma volume varies with relatively minor abnormal conditions. Gibson and Branch (60) have demonstrated that anaesthesia causes a decrease in the volume which runs parallel with the degree of elevation of the blood pressure, and that, by the end of an operation, the circulating volume is still further diminished, and may fall again at the beginning of the recovery period. Yet none of their cases showed the clinical signs of surgical shock.

Similarly, Keeley and his co-workers (96) noted a reduction in plasma volume in burns, but the blood pressure was maintained well above shock level. They and Weiner and Elman (162) maintain that the serum proteins are normal even in severe burns and that they are only diminished terminally. On the other hand, Cuthbertson and Tompsett (35) have shown that the plasma proteins fall in shock.

The problem may become even more confusing. Of the

three proteins present in the plasma, one may alter in proportion to its fellows. Thus fibrinogen is said to be raised in acute infections and its alteration is considered to be the main cause of alteration of the corpuscle sedimentation rate (171).

Cuthbertson and Tompsett (35) suggest that increased globulin is associated with healing processes, but they also say that trauma causes a rise in globulin and a fall in albumen. Weiner and Elman (162), although maintaining that the serum proteins are normal in severe thermal burns, have shown that the albumen-globulin ratio varied from 2.9 to 3.2, indicating a relatively greater loss of globulin to albumen, but that it returned to normal as the burn healed. It is therefore difficult to correlate the findings of Cuthbertson and Weiner because tissue destruction would appear to have been as great, if not greater, in Weiner's cases.

These results do not help in assessing possible changes in the protein ratio in colonic obstruction. Apart from alteration due to dehydration, there might be no change in the ratio of albumen to globulin, because tissue destruction is not marked in the bulk of cases and the only other possible cause is that based on the theory of proteose intoxication in intestinal obstruction put forward by Cooke, Whipple and Van Slyke (164) (165) (167). They suggest that in occlusion of the bowel the cause of death is due to excessive protein breakdown. That proteolysis may occur from dehydration has been

shown by McKay (111) and Marriott (115).

If proteins were set free in the blood stream, then one would expect the total proteins to be raised, but McKay (111), Orr and Haden (124) and Whipple and Cooke (165) all agree that proteins are broken down to the non-protein nitrogen state and, therefore, they would not alter the plasma protein level. Thus we are forced to conclude that if there be any changes in the values of the proteins they must be due to either severe dehydration or to some unknown effect of the obstruction.

(c) Non-protein nitrogen relationship.

The non-protein nitrogen of the blood has been shown to be raised in intestinal obstruction by many workers including Cooke (28), Cooper (30), Falconer and Lyall (48), Felty and Murray (50) and Haden and Orr and their co-workers (65) (68), in fact, Falconer and Lyall (48) maintain that it is the only constant biochemical change present in occlusion of the bowel. Levels as high as 400 mgm. per 100 c.c. of blood have been observed (68). The main cause of the high nitrogen figure is the increased blood urea and the latter has been noted many times in experimental obstruction of the bowel (10) (28) (86) (95) (104) (112) (113) (151) (166).

Theories as to the cause of the high N.P.N. (12).

1. Dehydration.

(a) According to McKay (111) and Marriott (115) dehydration is the cause of proteolysis

and this would lead to an increased blood urea.

(b) Teitelbaum (148) has shown that dehydration can cause a raised blood urea.

(c) Dehydration leads to an oliguria and this might lead to a retention of waste solids (39) (58) (85) (146) (173) (174).

These findings might be supported by other conditions in which dehydration and a raised blood urea are known to exist together such as after operation (39), haematemesis (135), and the vomiting of pregnancy (40) (64) (81), but Underhill and Carrington (153) say that there is no rise in urea in burns although dehydration is present.

2. Changes in the osmotic pressure of the plasma.

Another theory advanced for the raised blood nitrogen is that, owing to the loss of electrolytes in the vomit, urea is retained by the kidneys in an effort to keep up the osmotic pressure of the blood (87).

In favour of this is the remarkable improvement noticed in patients after the administration of sodium chloride. Against it is the fact that urea permeates all the tissues of the body and would not therefore regulate the osmotic pressure (105), and Hastings and Eichelberger (89) have shown that the extracellular tissue fluids contain all the chloride

and that muscle is not ordinarily permeable to chloride.

3. Alkalosis.

Owing to the loss of hydrochloric acid in the vomit, a state of alkalosis may exist. Felty and Murray (50) and Hastings (91) have demonstrated that, in obstruction of the upper bowel, the pH of the blood rises. A.M.Cooke (27) and Oakley (121) have shown that alkalosis causes a decrease in kidney function so that urea may be retained.

4. Transitory renal failure.

Brown and Eusterman (18) have suggested that there is a transitory renal failure, but Haden and Orr (68) and Whipple (166) say that this is not the case and that microscopic examination of the kidneys post-mortem shows no definite changes in them. Cooper (30) and McCance and Lawrence (106) say that the kidneys are not like those of a chronic nephritis and that, if the patient recovers from his obstruction, renal sequelae have never been recorded. However, there are factors which may help in causing renal failure and which cannot be passed over lightly.

(a) Lowered blood pressure.

It has been shown by Aird and Henderson (6) and Gendel and Fine (59) that distension of the bowel causes a fall in blood pressure and, in obstruction of the colon, this distension is well

marked, particularly in the caecum, (21) (57) (62) (129) (133) (159), and it occurs early in the obstruction (62) (128).

It is also recognised that intestinal strangulation, especially in its more severe forms, is likely to cause a fall in blood pressure because of depressor substances in the venous blood (100) (117) or in the peritoneal transudate (6).

The majority of patients suffering from colonic occlusion are over 60 and, although some may have high blood pressures, most of them tend to have low pressures, due to the onset of senile myocarditis.

Whether the lowering of the blood pressure is sufficient to cause renal failure is doubtful unless it is severe enough to cause greatly reduced glomerular pressure or virtual cessation of blood flow through the kidney, but in favour of the latter condition is the finding of Marriott (115) who says that reduced blood flow is present in obstruction. It should be borne in mind, however, in view of the warnings by Aird and Henderson (6) and Knight and Slome (100) that sudden relief of intestinal occlusion may set free depressor substances in the blood stream and that the blood pressure fall may bring about death although the obstruction is relieved.

(b) Dehydration.

If there is not sufficient water taken into the body there may not be sufficient blood to fill

the glomerular capillaries and so a smaller quantity of urine is passed. It has been calculated by Church (22) that at least 600 c.c. of water are required daily to act as solvents for the waste urinary solids and even then the urine would have a specific gravity of 1030. Thus dehydration may cause a reduction in urinary volume and solids may be retained in the system and signs of renal failure develop (39) (58) (85) (146) (174).

It has been shown by Hastings and Eichelberger (44) that hypertonic salines cause diuresis and this may account for some of the beneficial effects of salines. On the other hand, Rachmilewitz (132) says that the oliguria is not the cause of the high urea and there is ample evidence to show that urea may be raised in spite of polyuria (27) (106) (121).

(c) Plasma volume.

A fall in plasma volume causes a rise in plasma proteins and hence a rise in the colloidal osmotic pressure of the plasma. This in turn reduces the glomerular filtration unless the arterial blood pressure rises in compensation. From what has been said previously, this is not likely.

(d) Increased blood viscosity.

This condition may decrease the circulation rate owing to the number of red cells and so reduce the efficiency of the kidney. It is a well known finding in intestinal obstruction (3) (41) (75) (93) (110) (115).

(e) Alkalosis.

As has been said previously, A.M.Cooke (27) and Oakley (121) have shown that alkalosis causes a decrease in kidney function. The retained non-protein nitrogen increases the alkalosis and so a vicious circle is set up (12).

> "We may therefore conclude that signs of functional renal failure are common, but that excretion of urea, as judged by the height of the blood urea, may appear to be defective while other functions of the kidney are still relatively normal" (105).

5. Hepatic inefficiency.

This might be due to liver disfunction per se or brought about by the excessive vomiting.

6. Nervous control.

Little seems to be known about a possible nervous control of nitrogen metabolism but, in view of the relationship between the anterior pituitary and growth, there may be some control present.

7. Proteolytic.

It has been suggested that the cause of the high urea is excessive protein breakdown (111) (124) (165) and Haden and Orr and their co-workers (76) (80) (88) (124) maintain that there is a greater protein breakdown in intestinal obstruction than can be accounted for by starvation alone. The proteolysis may be aided by dehydration according to McKay (111) and Marriott (115).

(d) Chloride metabolism.

Possible alterations in the level of the blood chlorides in intestinal obstruction have been investigated by many workers including A.M.Cooke (27), Falconer and Lyall (48), Felty and Murray (50) and Haden and Orr (65), Hastings and Murray (91) McIver and Gamble (110) and McVicar (112) and all are agreed that there is a fall in the level. Bisgard (16) seems to be the only person who says that there is no alteration.

The hypochloraemia is chiefly due to the loss of chloride in the vomit, and all who have found this condition agree that the higher up in the alimentary canal the site of obstruction, the more profuse is the vomiting and the lower the level of the blood chlorides. It is thus well marked in pyloric stenosis and in occlusion of the upper intestine. A low blood chloride level is not confined to intestinal blockage, but may be found in other conditions in which vomiting is a feature such as the vomiting of pregnancy (40) (64) (81) (82) and the cyclical vomiting of children (87).

There is another factor which may aid in the loss of chloride from the body, namely, dehydration. It is now well established that the "cramps" of miners and stokers are due to excessive sweating, which brings about a chloride loss (45) (147), and that sodium chloride deficiency may occur in the tropics as a result of perspiration (101) (108). Animals may suffer as well as man, hence the reason for animals going to "salt licks" where

they may make good their deficiency. Keeley and Gibson (96), however, say that there is no alteration of the blood chloride level in burns although there is dehydration.

On the other hand, Darrow and Yannet (36) and Gamble and Ross (56) have shown that loss of sodium and chloride causes signs of dehydration, so it is possible for a vicious circle to be set up. A point of practical importance is stressed by McIver and Gamble (110) who say that the amount of hypochloraemia in intestinal obstruction is no guide to the degree of dehydration present.

The fall in chloride has been accounted for by Haden and Orr (65)(66)(67) (68) (69) (70) (71) (72) in their theory that not only is chloride accidentally lost in the vomit, but that the bulk of the chloride is used in some way as a protective agent to "fix" the toxins of the obstruction, and so render them harmless. They suggest that the following reaction may take place:-

$$\underset{\text{(toxic)}}{x} + NaCl + H_2CO_3 = NaHCO_3 + \underset{\text{(non-toxic)}}{x.HCl}$$

They offer in support of their theory their contention that the amount of chloride lost from the body cannot be accounted for by the loss through vomiting. Thus loss of chloride according to their view is not one of the possible causes of death but is an attempt by the body to prevent it.

This has influenced Scudder and his colleagues (138) (139) (173) to investigate the potassium level of the

plasma in view of the fact, first established by Amberg and Helmholtz (7), that sodium antagonises the action of potassium in the body. Falconer and Osterberg (49), however, have shown that there is no relationship between chloride fall and the potassium level of the plasma. Similarly, although there are usually alterations in chloride and non-protein nitrogen, there is no evidence that the one is clinically related to the other. This bears resemblance to the case reported by Kerr and Lendrum (98) in which a papilloma of the gall-bladder secreted sodium chloride but did not seem to affect the non-protein nitrogen level of the blood.

In regard to replacement of water and chloride in those suffering from intestinal obstruction, Falconer and Lyall (47) are of the opinion that rehydration is more important than the replacement of chloride. This is of interest because Haden and Orr discovered that the use of "large quantities of saline" relieved the symptoms of occlusion of the bowel, and some of the improvement may have been due to the use of fluid rather than of actual chloride solution. On the other hand, they state that glucose solution does not relieve the symptoms of intestinal obstruction and so they suggest that the use of sodium chloride is specific, but even then it does not entirely rule out the value of water.

(e) Potassium factors.

In the search for the toxin of intestinal occlusion

Scudder (138) considered the possibility of potassium as the cause of the symptoms, particularly because of its well-known depressant action on the heart, first shown by Howell in 1901 (95) and later verified by Carlson (20) and Mathison (116). He was also influenced by the similarity of the symptoms of acute intestinal obstruction and adrenal insufficiency. His colleagues, Zwemer and Sullivan (174), had shortly before suggested that potassium was regulated by the adrenal cortex and Thorn and Garbutt (150), Nilson (120) and Truszkowski and Zwemer (152) by their experiments, agreed with this view. His hypothesis was supported by the fact that sodium chloride, which is of great value in the treatment of intestinal obstruction, is directly antagonistic to the action of potassium in the human body (7).

As a result of his initial experiments on 8 cats, Scudder (138) came to the conclusion that the potassium of the plasma in intestinal obstruction rose to levels which were definitely toxic, and that the secretion of potassium into the gastric lumen, followed by vomiting, was a means whereby the body attempted to get rid of this poisonous element. He showed that the concentration of potassium in the vomit was much higher than that in the plasma and he associated the increased level in the plasma with the increased blood density.

His results, however, require further analysis. Of the 8 animals used in the experiments, one had an obstruction of the oesophagus, two had occlusions below the

Ampulla of Vater, one had a blockage in the middle of the transverse colon and the remaining four had strangulated loops. Thus his sweeping statements are based on observations on 3 animals with intestinal obstruction, for the first animal could not vomit and, as has been emphasised previously, the condition of strangulation is totally different to simple occlusion of the bowel. In addition, there was an initial fall in the plasma potassium in nearly all the cats and the rise seemed terminally. In the animal with colonic occlusion, the potassium fell for the first 5 days.

In a later series of 25 patients suffering from acute intestinal obstruction in which Scudder and Zwemer (139) investigated the potassium of the plasma, 5 were not investigated until after treatment had commenced and, of the remainder, it was raised in 7 cases only and was low in 5. Thus their results are quite indefinite and hardly justify their conclusion.

Since then Bisgard (16) has investigated the level of potassium in animals suffering from various conditions. He found it raised in approximately half the animals suffering from intestinal obstruction. He was not impressed with his results and he did not think that potassium was the toxic factor. He was of the opinion that changes in the potassium level were incidental to changes in the plasma volume as it was influenced by alterations in blood pressure produced by shock and anaesthetics. The latter has been confirmed by Larson and Brewer(102).

Bisgard agreed with Scudder's findings that there was a terminal rise in all animals.

Falconer and Osterberg (49) found a fall of plasma potassium in intestinal obstruction and said that their results did not correspond to any particular factor. They, too, thought that any alteration in the level was incidental to the other changes, although they recognised that potassium was lost in the vomit. As a result of their findings, they have suggested the use of Ringer's solution instead of normal saline for the treatment of intestinal occlusion in order to make good the loss of potassium as well as the loss of chloride.

It is, therefore, not surprising that a similar difference of opinion exists in the results obtained on the estimation of the plasma potassium in haemorrhage. Kerr (97), who investigated the problem in 1926, said that there was a rise, and so did Thaler (149) in 1935, but Stewart and Rourke (145) in 1936 said that there was no rise. Of interest, too, is the fact that Keeley and Gibson (96) say that there is no significant change in the plasma potassium level in burns although other chemical changes similar to those occurring in intestinal obstruction have been mentioned previously. In association with these remarks, it should be remembered that similar changes in the adrenal cortex have been observed in intestinal occlusion and in burns, so that if potassium were regulated by the adrenal cortex, then one would expect similar potassium changes in both conditions.

(f) Peculiarities of colonic occlusion.

As has been mentioned in the Introduction, most work on alimentary obstruction has been done on blockage of the duodenum and jejunum, but it must be pointed out that this type of obstruction is rare clinically (110). It is therefore necessary to consider factors in regard to the physiology of the colon and the pathology of its tumours which might influence the biochemistry of occlusion of this part of the intestine.

Although absorption of some substances may take place in the distal colon (33), the functions of the two halves of the colon are considered to be different for the following reasons:-

(1) The proximal part of the colon is developed from the mid-gut along with the absorptive small intestine, while the distal part is developed from the hind-gut.

(2) The proximal part of the colon is of larger calibre and its walls are thinner than the smaller thick walled distal portion.

(3) The prevailing type of movement in the proximal part of the colon, particularly at the caecum, is churning, whereas the prevailing type in the distal part is propulsion.

(4) The content of the proximal part of the colon is liquid, whereas that of the distal part is solid or semi-solid.

In regard to the water content of the colon,

Steggerda (144) found that the faeces became approximately 10% more concentrated in each of the four parts of the colon, namely, caecum, ascending colon, descending colon and rectum. He thought that there was a direct relationship between water absorption and the blood supply to the parts, but Bonoff (17) considered it to be related to the amount of mucus present.

The type of tumour in the halves is also different (9). In the caecum and ascending colon it is of the proliferative kind, whereas in the rest of the colon it is usually of the ring stricture variety. Therefore, owing to

(1) The greater calibre of the proximal colon,

(2) The fluidity of its contents,

(3) The type of tumour,

obstruction of the proximal colon is rare compared to the rest of the colon. (140).

Although obstruction be present, food, water and intestinal secretions may pass into the proximal colon, at least for a time, and increase the distension there (133). At this stage the valve action of the ileo-caecal orifice does not allow regurgitation. Thus vomiting is not an early feature in colonic occlusion and so dehydration and other chemical alterations may not be present during the first few days of the obstruction (133).

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CHAPTER 4.

METHODS.

In the investigations under consideration, certain chemical changes in the blood of the patient were estimated before operation and for three days after operation at approximately 24 hour intervals. Venous blood was used because not only is it easy of access but the concentrations of electrolytes in the various extra-cellular fluids are fairly closely predicted from the concentrations in venous serum (84).

As it was intended to calculate serum potassium and as the whole blood potassium is many times that of the serum potassium, it was decided to allow clotting of the blood to take place and to make all the calculations on serum. Fibrinogen was therefore lost from the total proteins but, as its calculation is said to be of little value clinically (83), and as Cuthbertson and Tompsett (35) say that oxalate reduces the total protein content of the serum, its loss was considered to be of little importance compared with the degree of accuracy likely to be obtained in the calculation of the serum potassium and the total and individual protein contents.

Before proceeding to the examination of likely abnormal bloods, determinations were carried out on 12 normal bloods in order to obtain accuracy and familiarity with the methods. In connection with the former, many of the bloods were re-examined twice and many of the abnormal bloods also were re-

estimated, especially those with marked abnormal figures.

The following estimations were carried out:-

1. The serum chloride concentration.
2. The serum potassium concentration.
3. The N.P.N. of the blood.
4. The total protein concentration of the serum.
5. The albumen concentration of the serum.
6. The globulin concentration.
7. The albumen-globulin ratio.

1. The serum chloride concentration.

This determination was carried out after the manner of Van Slyke's modification of Whitehorn's method (155). Owing to the hygroscopic action of the thiocyanate used for titration, a control with distilled water was carried out daily.

2. The serum potassium concentration.

The method used for this was the platinic chloride micro-method of Shohl and Bennett (141) in which the potassium of the serum is first converted into potassium chloro-platinate and then, by the addition of potassium iodide, into the wine-coloured iodo-platinate. This is then titrated with thiosulphate.

This method has the advantage that it is based on a precipitate of definite and constant composition and its accuracy is such that 0.1 mgm. of K (the amount in 0.5 c.c. serum) can usually be determined with an error within ± 4%, and 0.4 mgm. with an error within ± 2% (141). Controls were carried out daily. The end point is also quite definite. This is not the method used by Scudder and his colleagues, nor by Falconer and Osterberg.

3. The N.P.N. of the blood.

This was estimated by a micro-Kjeldahl method (83). The digestion was done by sulphuric acid and the catalyst employed was selenium. This made the digestion much quicker, and no alteration could be found in results checked with samples using the older and slower method of copper sulphate as the catalyst.

4. The total protein concentration of the serum.

For this investigation the total nitrogen of the serum was estimated by the micro-Kjeldahl method, selenium being used as the catalyst. From this result the non-protein nitrogen figure obtained by the previous determination was deducted. This gave the nitrogen figure for the proteins of the serum and from it the amount of the total proteins was calculated. This procedure is much more accurate than refraction or colorimetric methods.

5. The albumen concentration of the serum.

6. The globulin concentration.

In these determinations the globulin was precipitated by 22% sodium sulphate and the albumen filtered off. It is very important to use a very fine filter paper otherwise globulin will come through in the filtrate. The albumen was digested by sulphuric acid and its amount estimated from the nitrogen figure obtained after making allowance for the N.P.N.

The globulin figure was obtained by subtracting the albumen result from the total protein figure.

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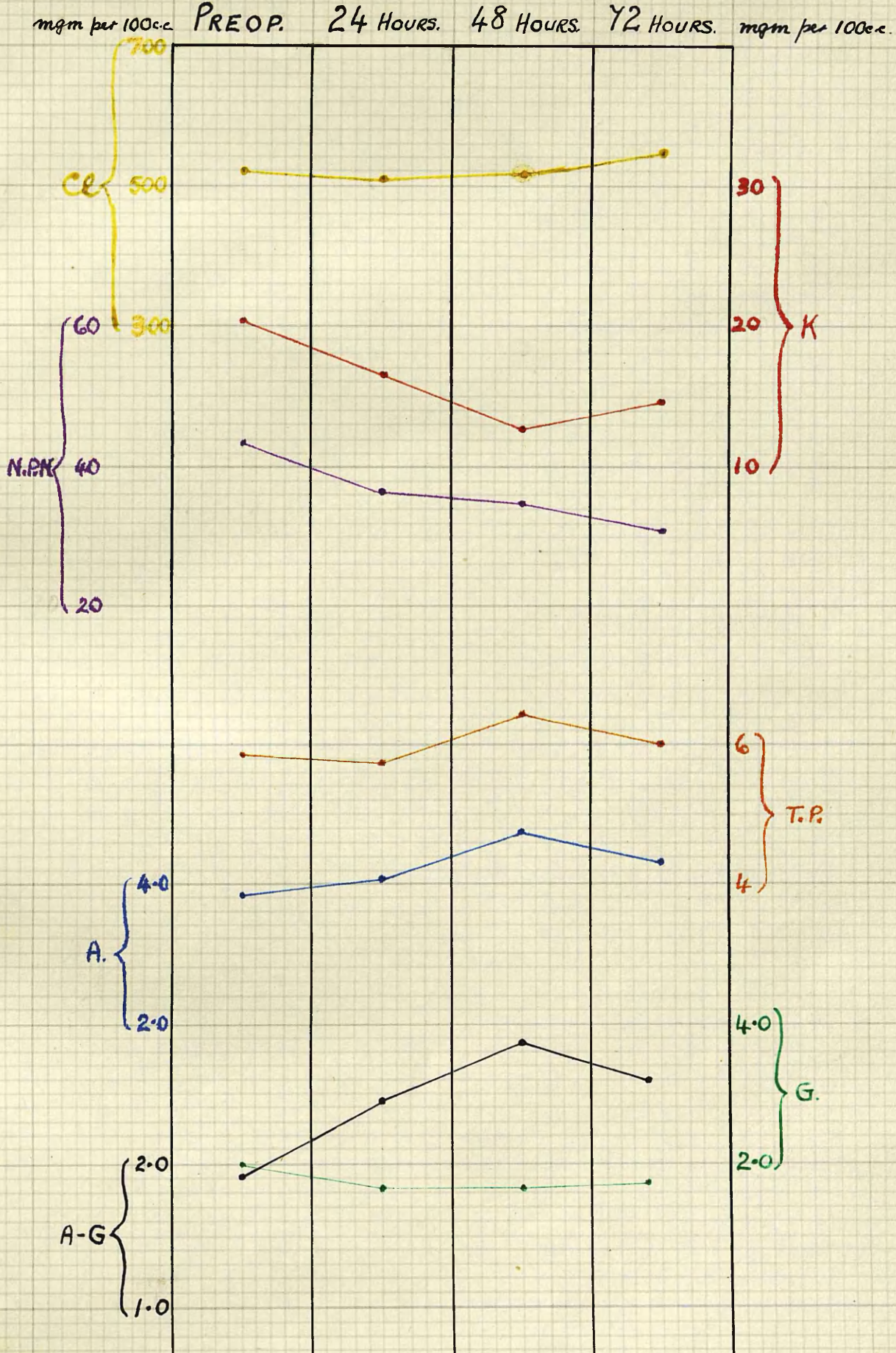
CASE 1.


mgm per 100c.c.
PREOP.
24 HOURS.
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CHAPTER 5.

CASES.

Case 1.

Man, aged 73.

Constipated 5 days. Abdominal pain. Vomiting slight.

Carcinoma of splenic flexure.

Operation - caecostomy.

Death 4 days after operation.

	Preop.	24 Hours.	48 Hours	72 Hours
Chlor.	520	510	520	541
Potass.	20.33	16.8	12.9	14.86
N.P.N.	43.68	36.4	35.0	30.8
T.P.	5.85	5.77	6.43	6.02
Albumen.	3.85	4.10	4.78	4.35
Globulin.	2.0	1.67	1.65	1.67
A-G Ratio	1.92	2.46	2.89	2.60

Case 2.

Man, aged 74.

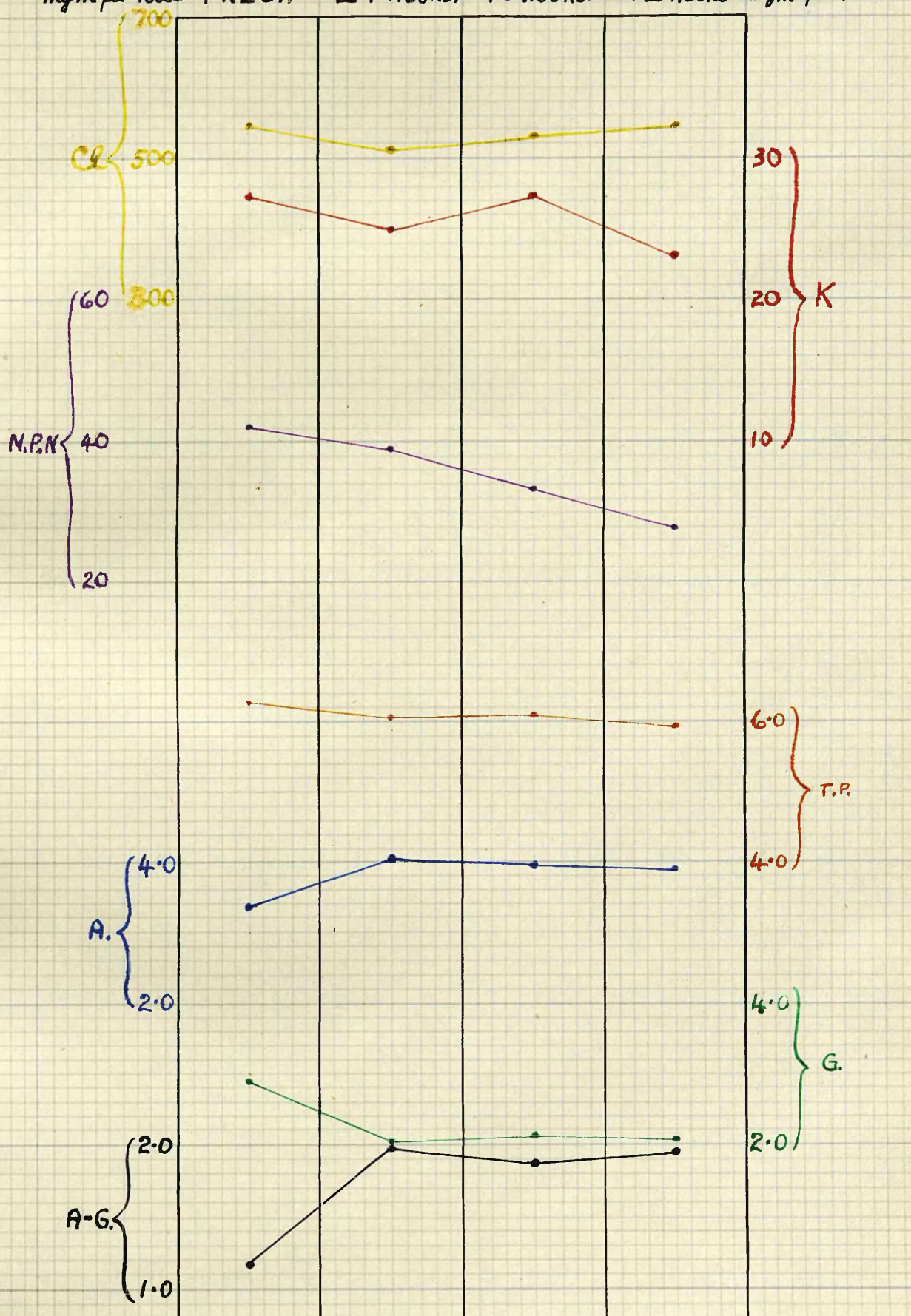
constipated 4 days. Abdominal pain. Vomiting slight.

Carcinoma of sigmoid.

Operation - descending colostomy.

Recovery uneventful.

CASE 2.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mgm per 100 c.c.
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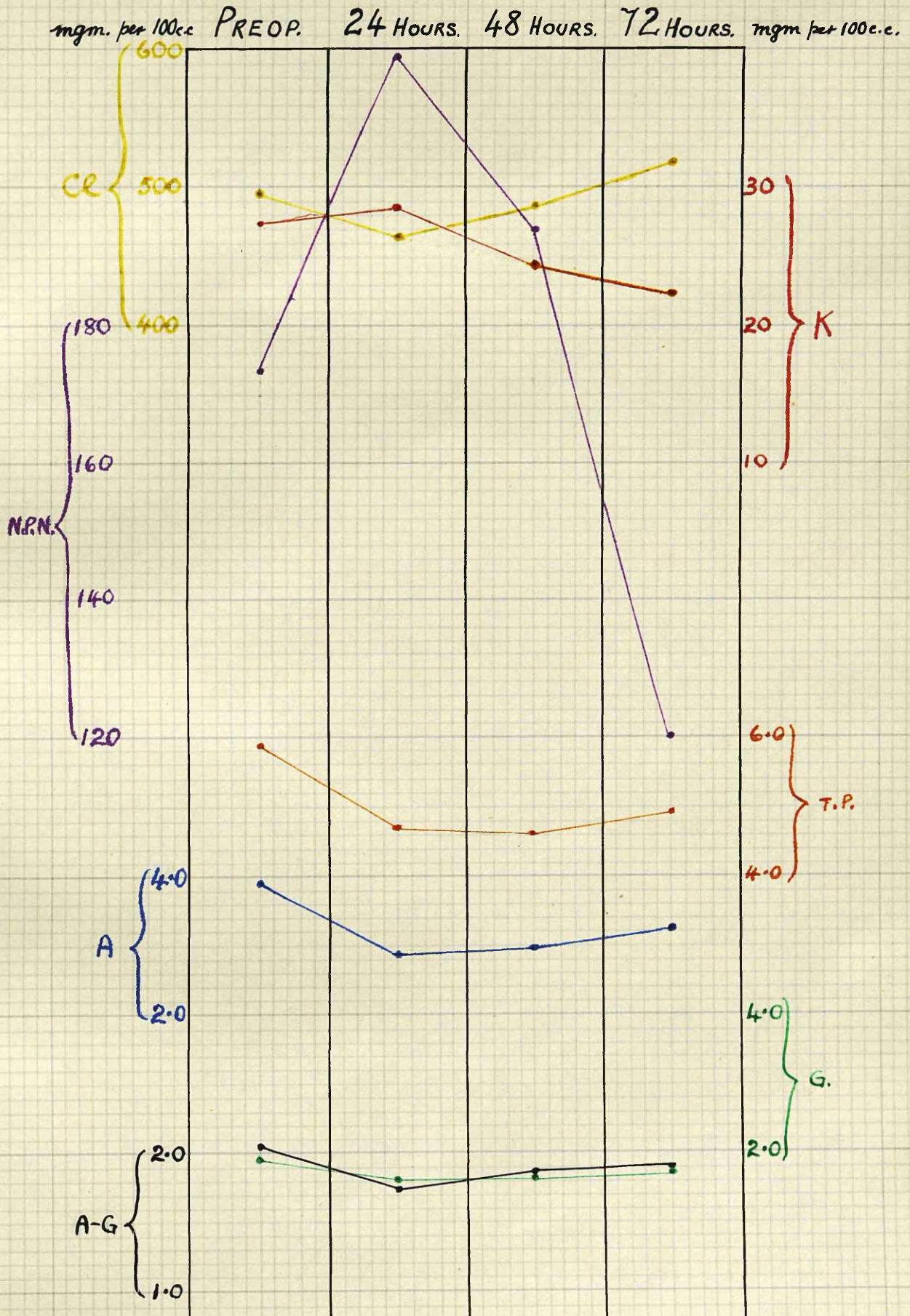
CASE 3.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PREOP.
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72 HOURS.
mgm per 100 c.c.
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	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	510	530	541
Potass.	27.14	25.0	27.37	23.07
N.P.N.	42.0	39.2	33.6	28.0
T.P.	6.26	6.06	6.09	5.95
Albumen.	3.37	4.05	3.98	3.93
Globulin.	2.89	2.01	2.11	2.02
A-G Ratio	1.17	2.01	1.89	1.95

Case 3.

Man, aged 58.

Constipated 6 days. Abdominal pain. Vomiting marked.

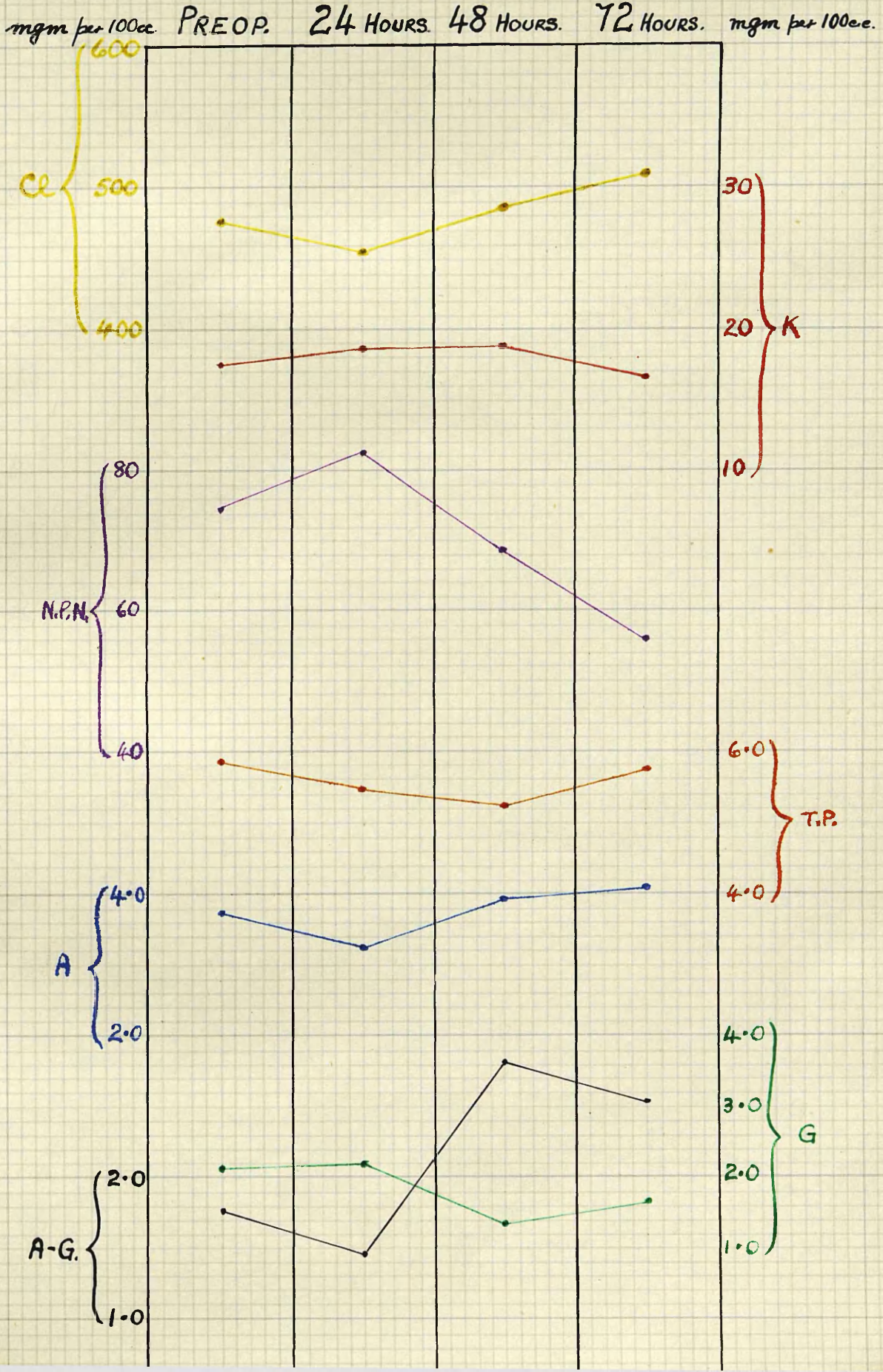
Carcinoma of sigmoid.

Operation - descending colostomy. Small intestine greatly distended.

Recovery after stormy convalescence.

	Preop.	24 Hours.	48 Hours	72 Hours
Chlor.	496	466	488	519
Potass.	27.37	28.5	24.6	22.3
N.P.N.	173.6	218.4	194.0	120.0
T.P.	5.85	4.70	4.61	4.97
Albumen.	3.93	2.87	2.98	3.24
Globulin.	1.92	1.63	1.63	1.73
A-G Ratio	2.05	1.76	1.83	1.87

CASE 4.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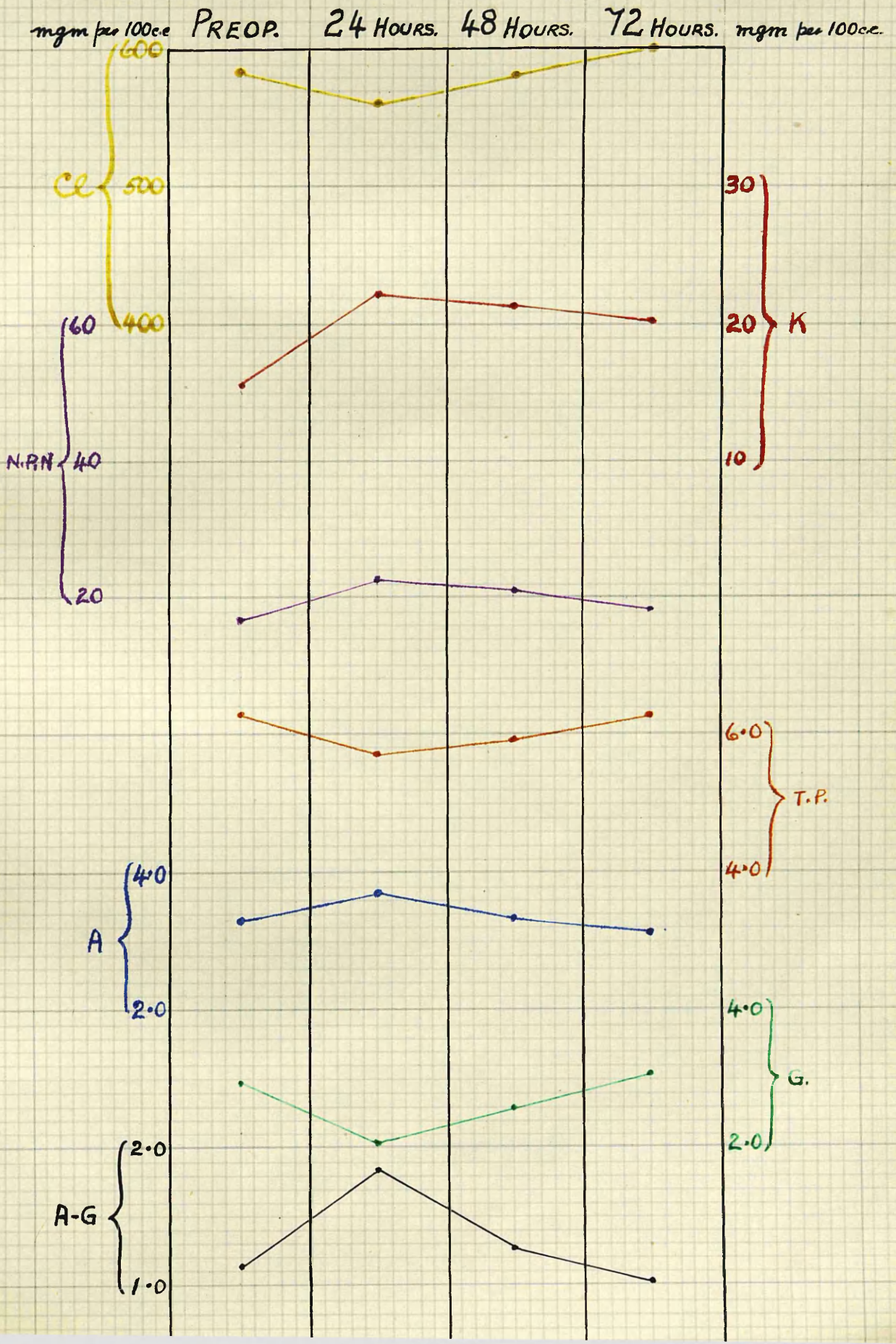
CASE 5.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Case 4.

Woman, aged 65.

Constipated 6 days. Abdominal pain. Much vomiting.

Carcinoma of sigmoid.

Operation - descending colostomy.

Recovery slow.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	477	456	488	510
Potass.	17.59	18.77	18.9	16.8
N.P.N.	74.2	82.6	68.6	56.0
T.P.	5.84	5.46	5.23	5.75
Albumen.	3.74	3.25	3.86	4.12
Globulin.	2.10	2.21	1.37	1.63
A-G Ratio	1.78	1.47	2.82	2.53

Case 5.

Man, aged 69.

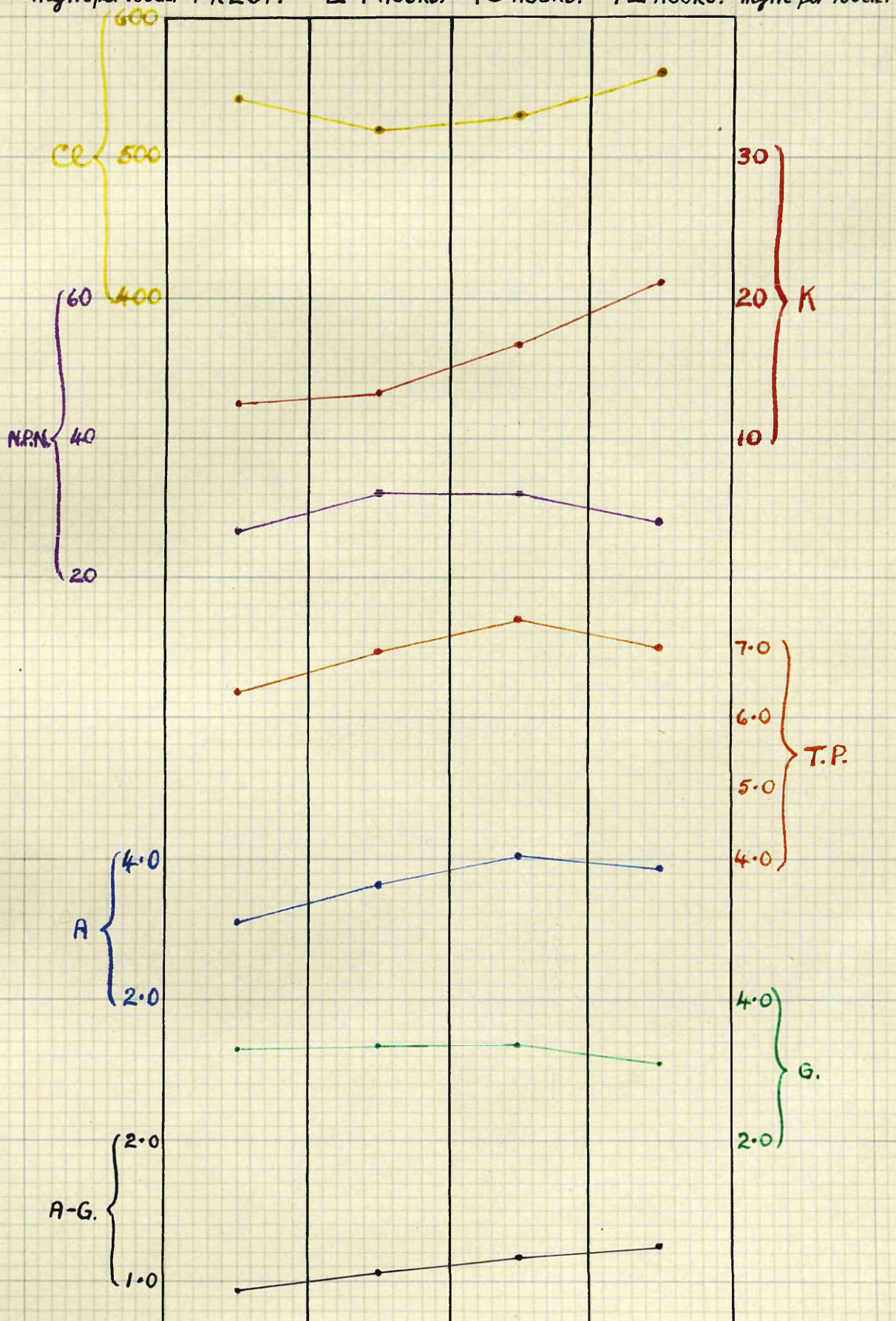
Constipated 4 days. Abdominal pain. Vomited once.

Carcinoma of descending colon.

Operation - caecostomy.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	583	562	583	603
Potass.	15.64	22.3	21.5	20.3
N.P.N.	16.8	22.4	21.0	18.2
T.P.	6.28	5.72	5.94	6.27
Albumen.	3.32	3.71	3.34	3.15
Globulin.	2.96	2.01	2.60	3.12
A-G Ratio	1.12	1.85	1.28	1.01

CASE 6.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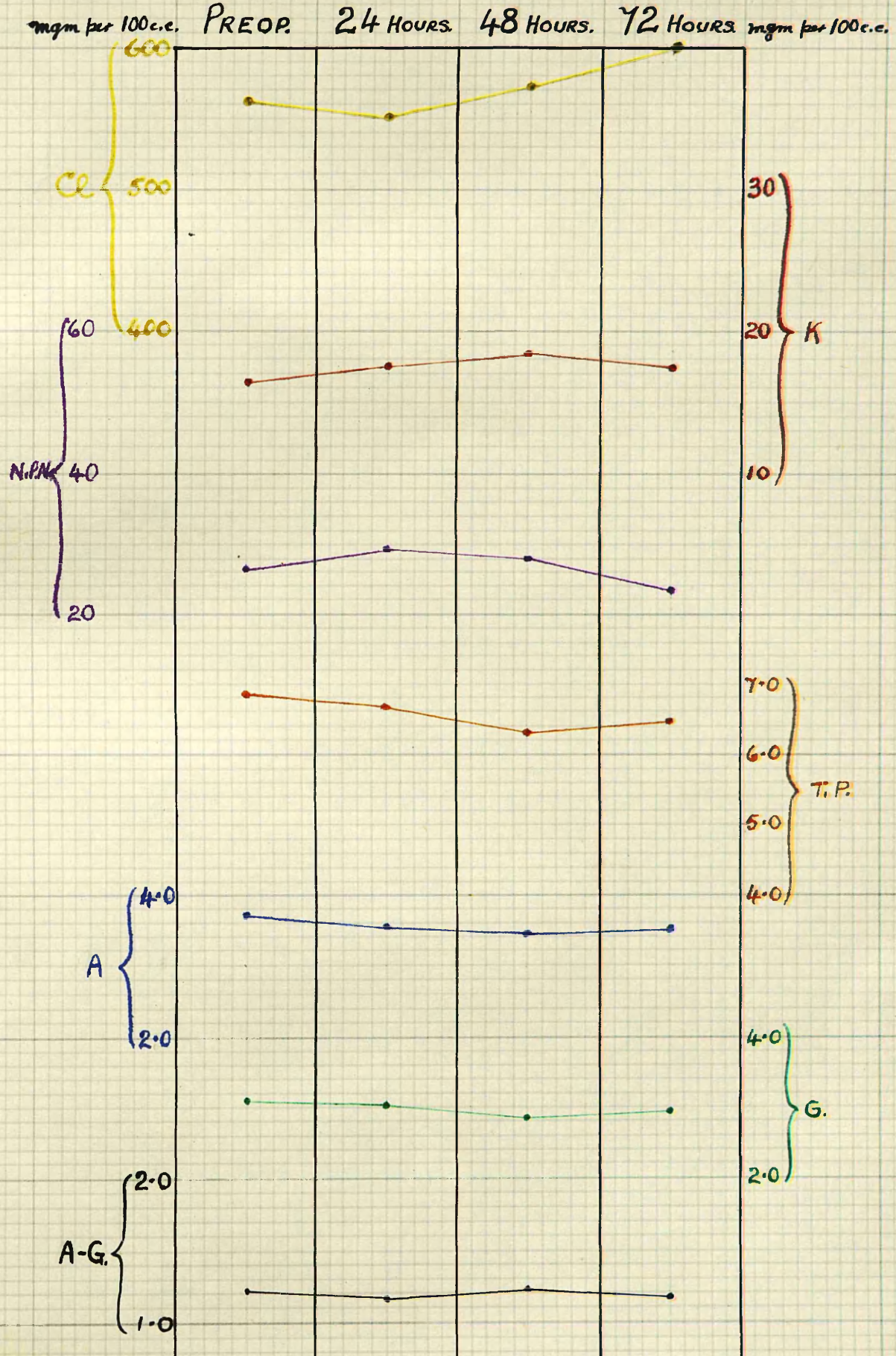
CASE 7.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Case 6.

Woman, aged 59.

Constipated 5 days. Abdominal pain. Vomited slightly.

Carcinoma of hepatic flexure.

Operation - caecostomy.

Recovery uninterrupted.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	520	530	562
Potass.	12.52	13.3	16.8	21.1
N.P.N.	26.6	32.2	32.2	28.0
T.P.	6.39	6.97	7.41	7.00
Albumen.	3.09	3.60	4.03	3.89
Globulin.	3.30	3.37	3.38	3.11
A-G Ratio	0.94	1.07	1.19	1.25

Case 7.

Man, aged 58.

Constipated 4 days. Abdominal pain. Vomited once.

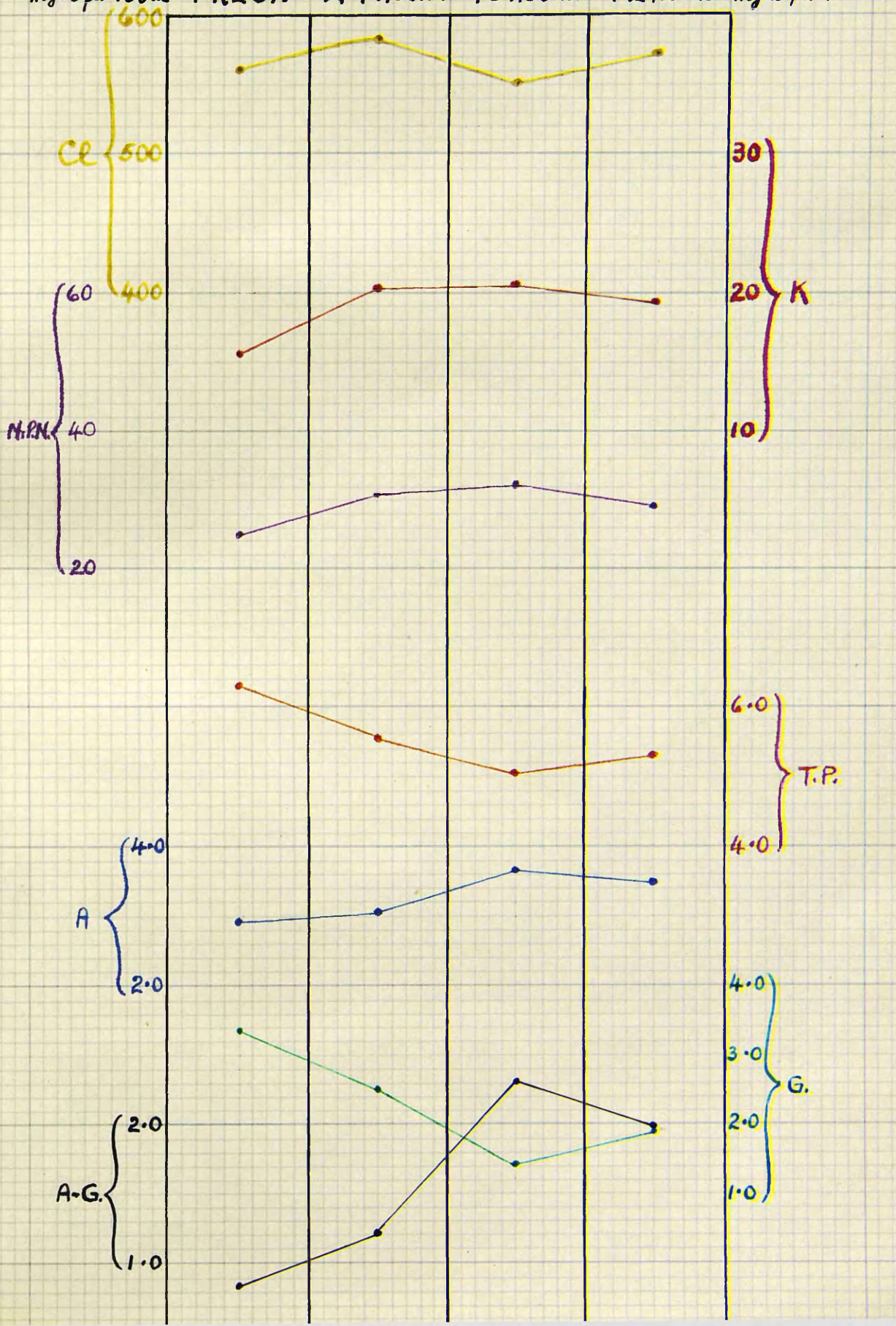
Carcinoma of rectum.

Operation - descending colostomy.

Recovery uninterrupted.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	562	551	572	603
Potass.	16.4	17.6	18.4	17.6
N.P.N.	26.6	29.4	28.0	23.8
T.P.	6.83	6.65	6.31	6.49
Albumen.	3.74	3.59	3.46	3.52
Globulin.	3.09	3.06	2.85	2.97
A-G Ratio	1.21	1.17	1.21	1.19

CASE 8.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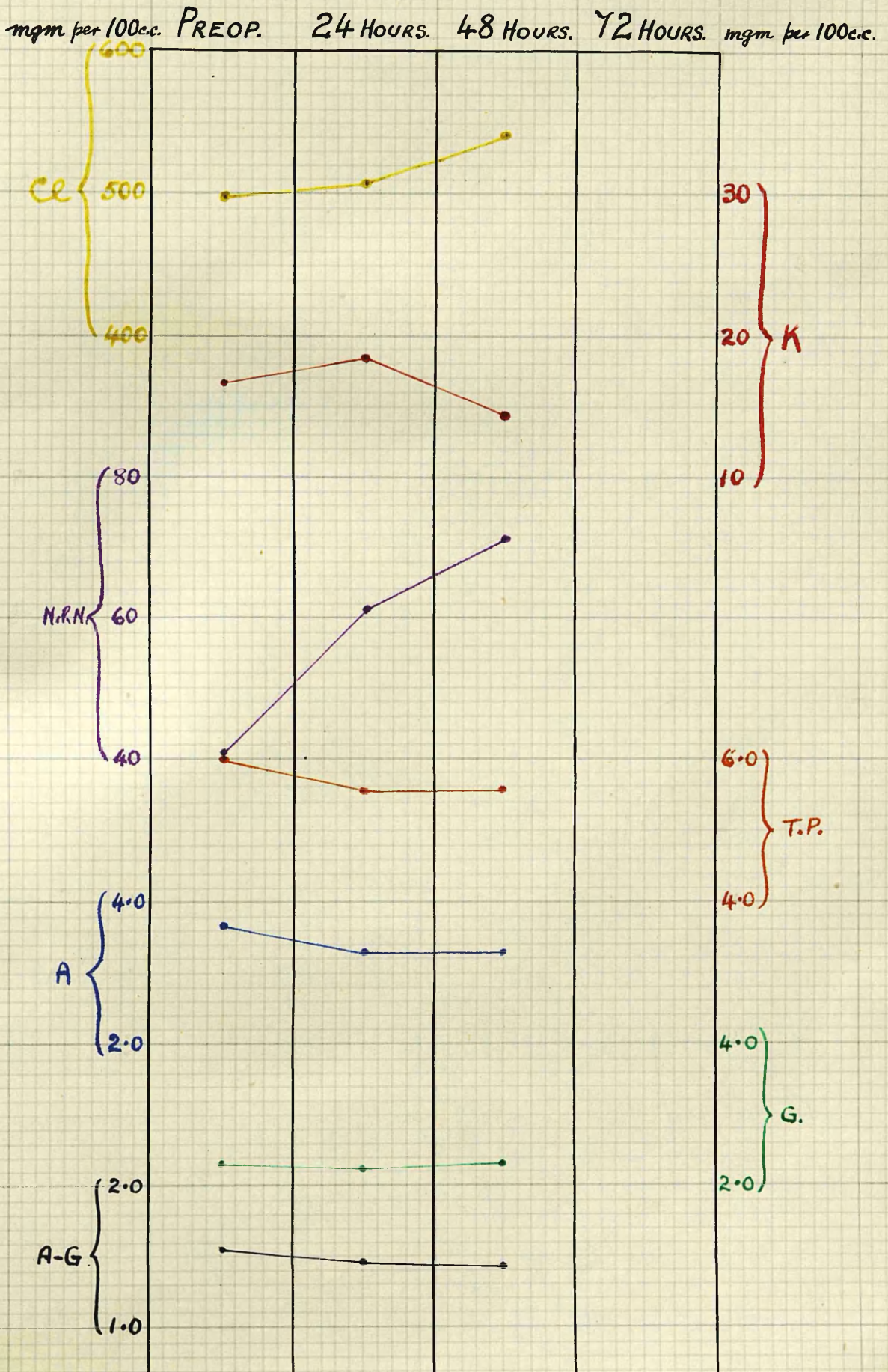
CASE 9.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Case 8.

Man, aged 58.

Constipated 4 days. Abdominal pain. Vomited once.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	562	583	551	572
Potass.	15.64	20.3	20.7	19.2
N.P.N.	25.2	30.8	32.2	29.4
T.P.	6.32	5.58	5.05	5.34
Albumen.	2.93	3.06	3.65	3.48
Globulin.	3.39	2.52	1.40	1.86
A-G Ratio	0.86	1.21	2.61	1.87

Case 9.

Man, aged 61.

Constipated 4 days. Abdominal pain. Vomiting marked.

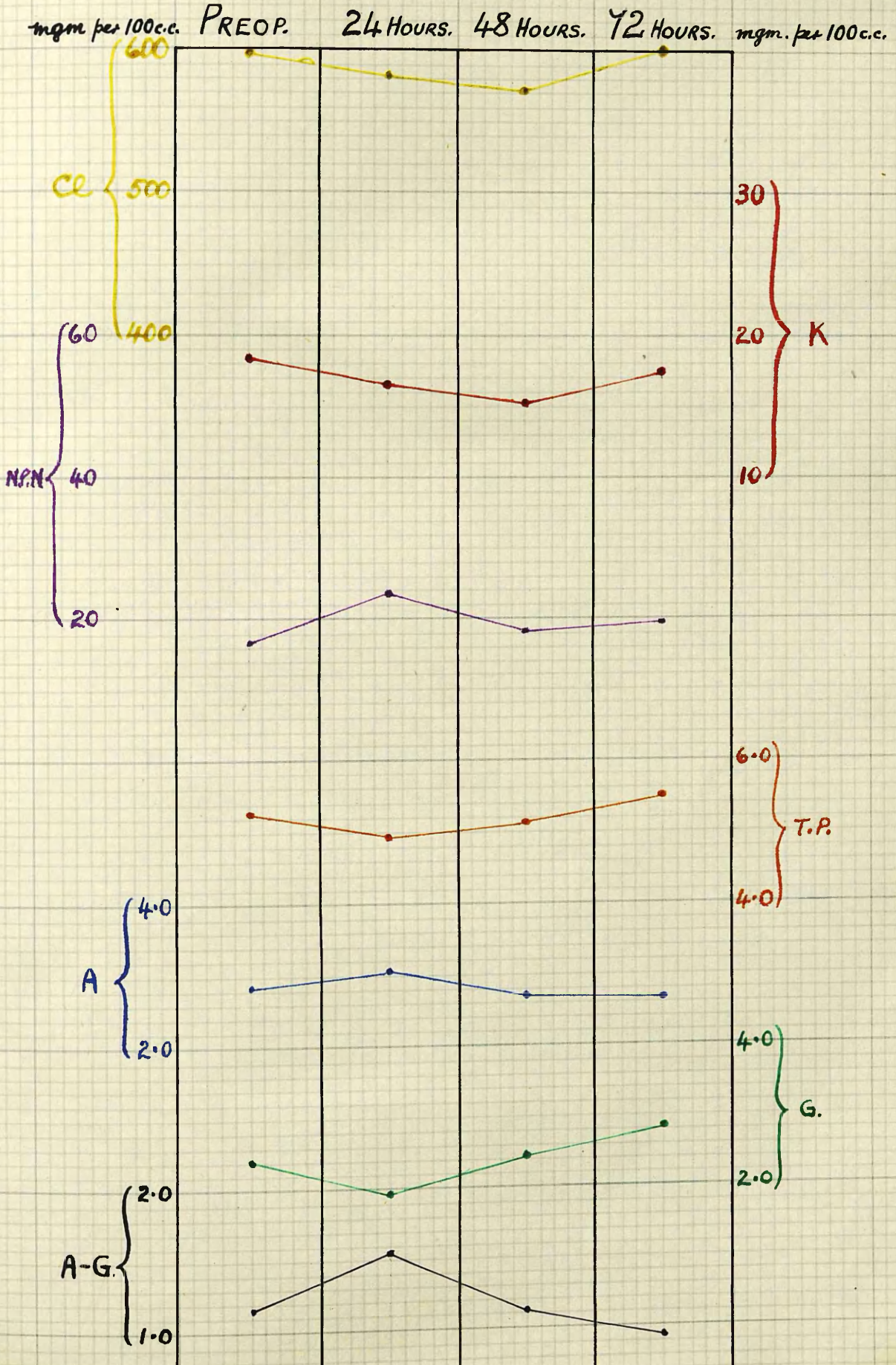
Carcinoma of splenic flexure.

Operation - caecostomy.

Death on 3rd day after operation.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	498	509	541	--
Potass.	16.8	18.4	14.5	--
N.P.N.	40.6	61.6	71.4	--
T.P.	6.04	5.56	5.59	--
Albumen.	3.67	3.31	3.29	--
Globulin.	2.37	2.25	2.30	--
A-G Ratio	1.55	1.47	1.43	--

CASE 10.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mgm. per 100 c.c.
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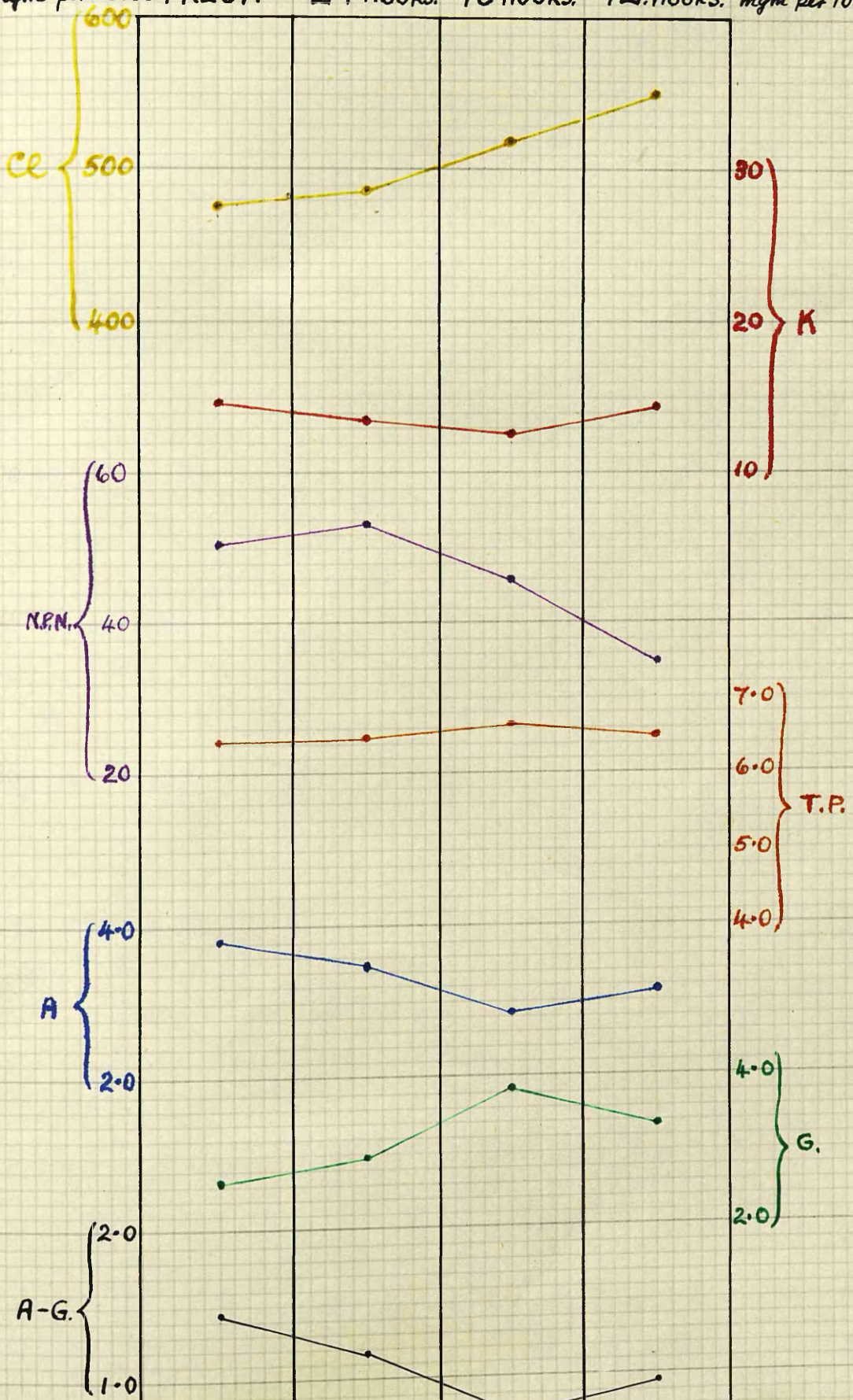
CASE 11.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mgm per 100c.c.
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Case 10.

Woman, aged 58.

Constipated 7 days. Abdominal pains. Vomited once.

Carcinoma of sigmoid.

Operation - descending Colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	598	583	572	604
Potass.	18.4	16.8	15.2	17.6
N.P.N.	16.8	23.8	18.2	19.6
T.P.	5.23	4.98	5.14	5.52
Albumen.	2.82	3.04	2.71	2.68
Globulin.	2.41	1.94	2.43	2.84
A-G Ratio	1.17	1.57	1.17	0.94

Case 11.

Man, aged 48.

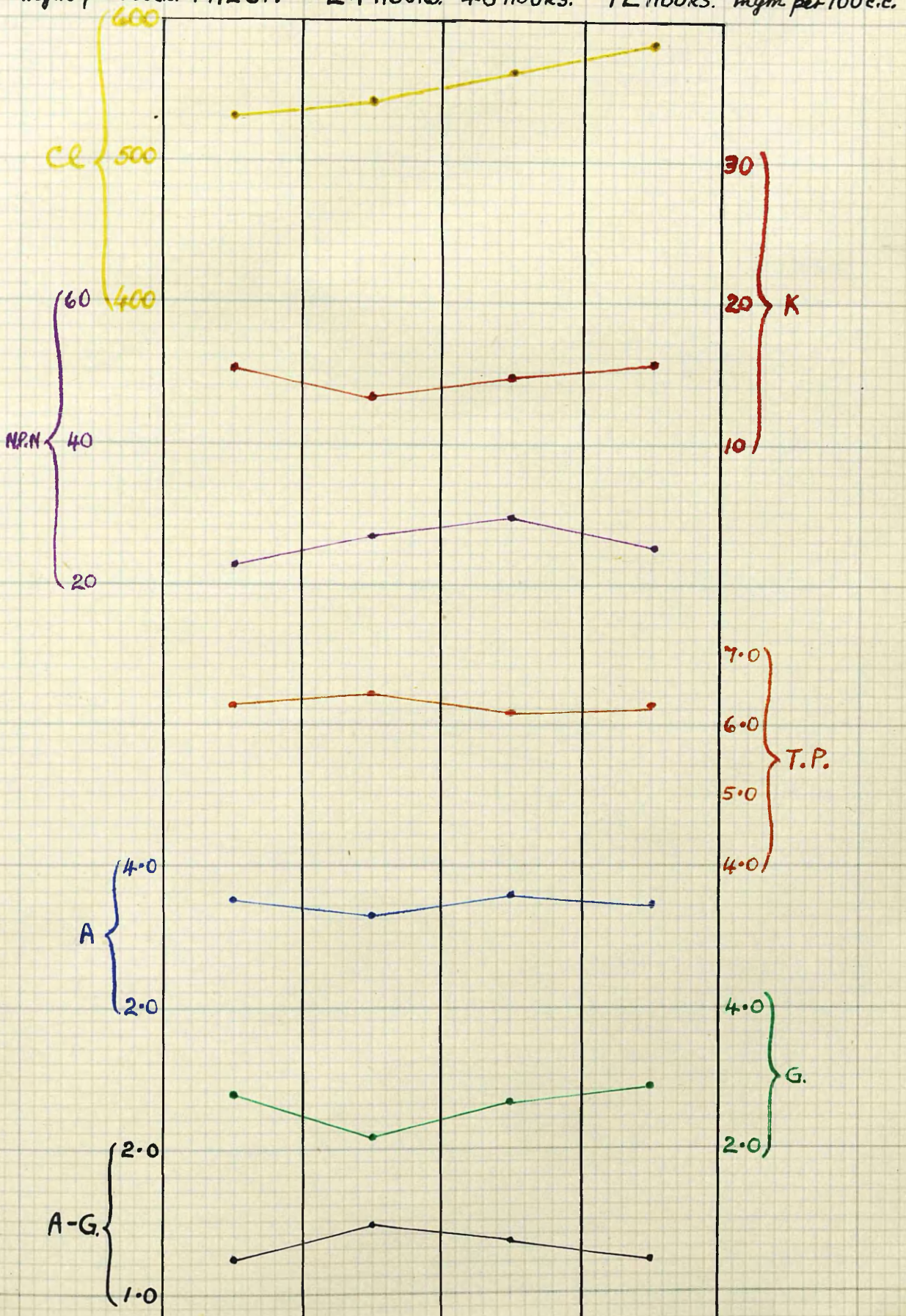
Constipated 4 days. Abdominal pains. Vomited frequently.

Carcinoma of descending colon. Much small bowel distension.

Operation - transverse colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	477	488	520	551
Potass.	14.8	13.7	12.9	14.5
N.P.N.	50.4	53.6	46.2	35.0
T.P.	6.42	6.49	6.62	6.51
Albumen.	3.81	3.52	2.81	3.17
Globulin.	2.61	2.97	3.81	3.34
A-G Ratio.	1.46	1.19	0.74	0.95

CASE 12.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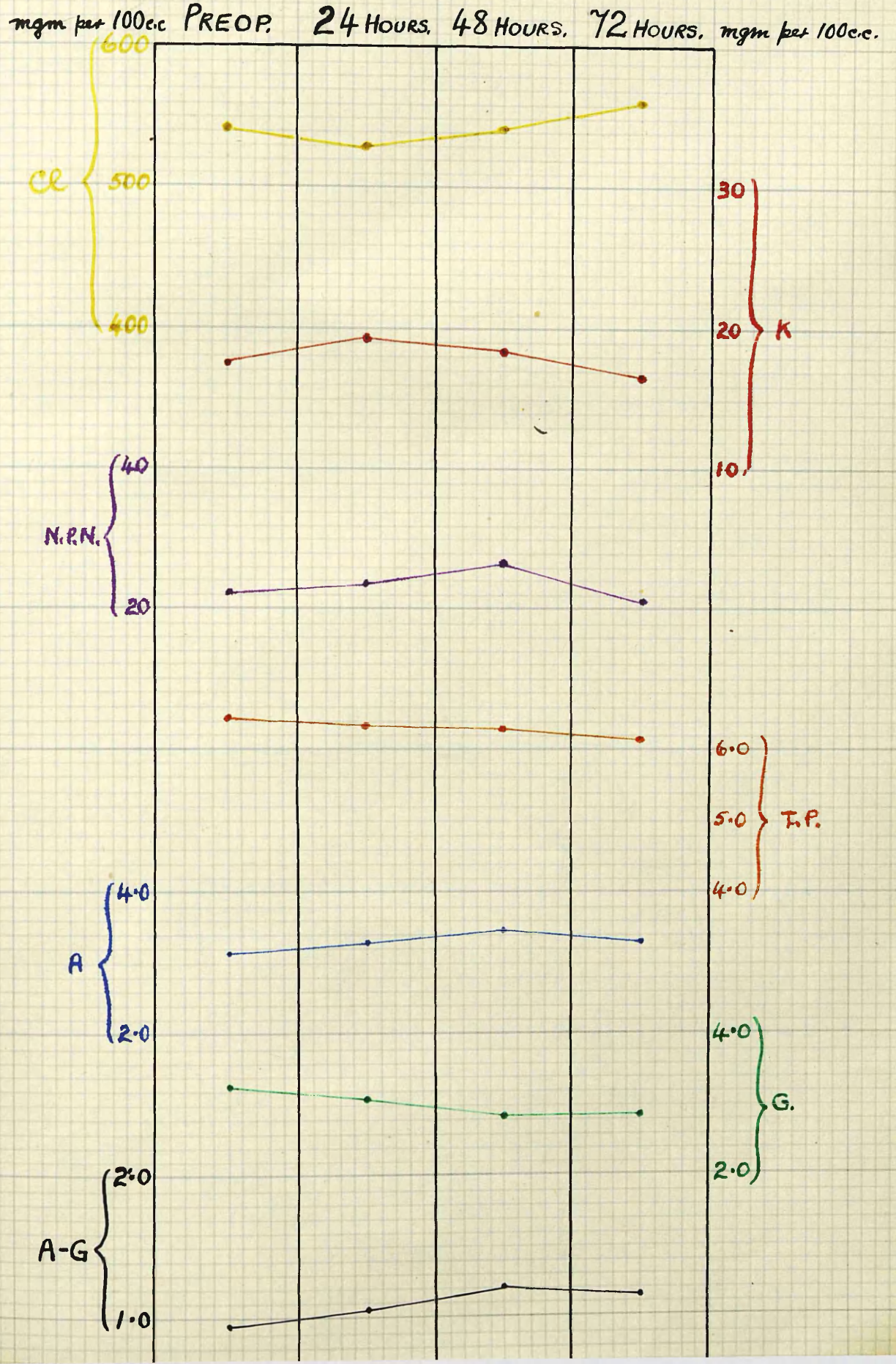
CASE 13.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mgm per 100c.c.
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Case 12.

Man, aged 59.

Constipated 4 days. Abdominal pains. Vomited slightly.

Carcinoma of transverse colon.

Operation - caecostomy. Recovery uninterrupted.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	530	541	562	583
Potass.	15.25	13.3	14.9	15.6
N.P.N.	22.4	26.6	29.4	25.2
T.P.	6.25	6.40	6.16	6.24
Albumen.	3.47	3.23	3.54	3.41
Globulin.	2.78	2.17	2.62	2.83
A-G Ratio	1.25	1.49	1.35	1.20

Case 13.

Man, aged 27.

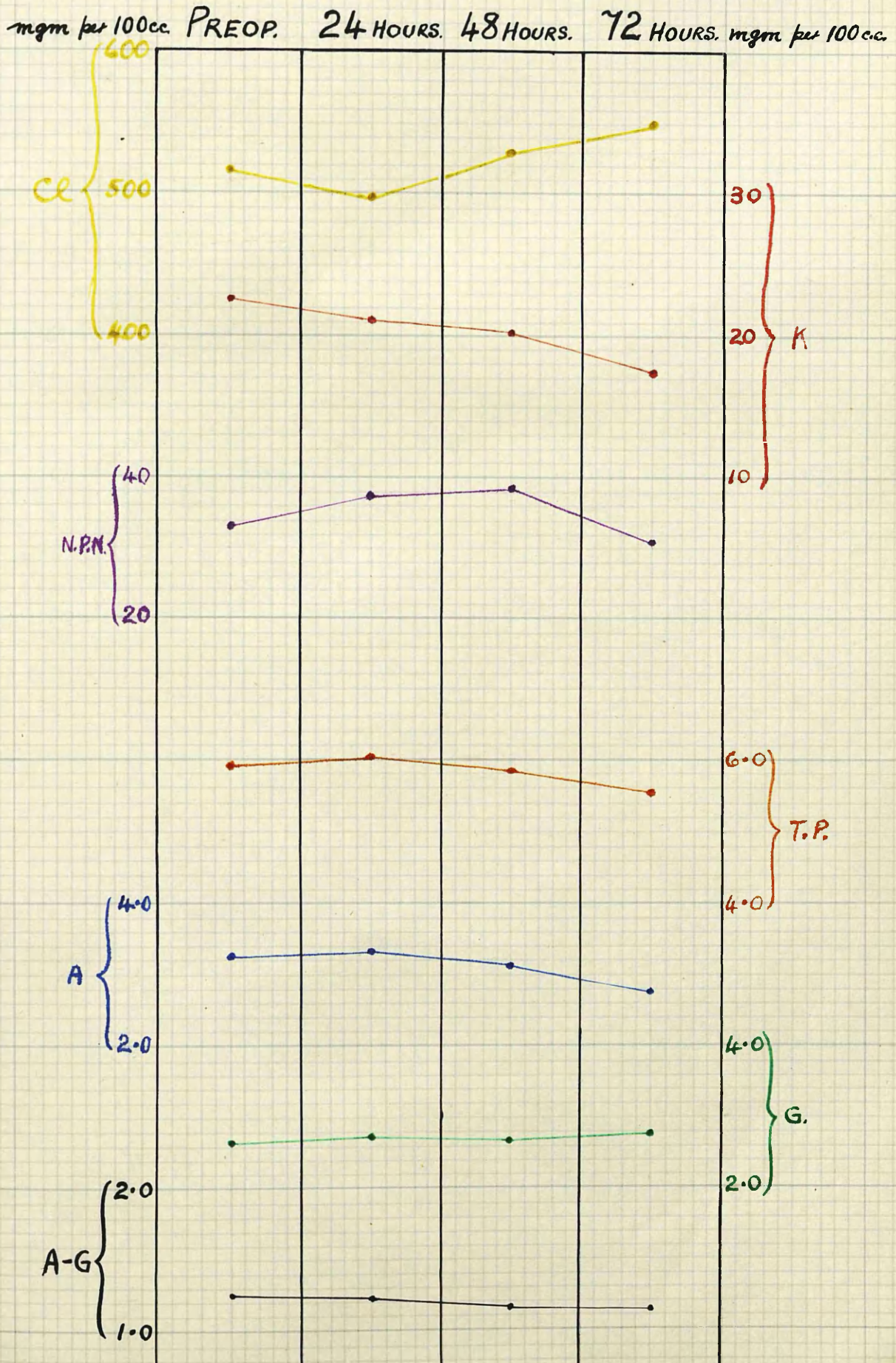
Constipated 4 days. Abdominal pains. Vomited slightly.

Carcinoma of rectum.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	530	541	562
Potass.	17.6	19.2	18.4	16.8
N.P.N.	22.4	23.8	26.6	21.0
T.P.	6.42	6.38	6.31	6.19
Albumen.	3.15	3.29	3.46	3.33
Globulin.	3.27	3.09	2.85	2.86
A-G Ratio	0.96	1.06	1.21	1.16

CASE 14.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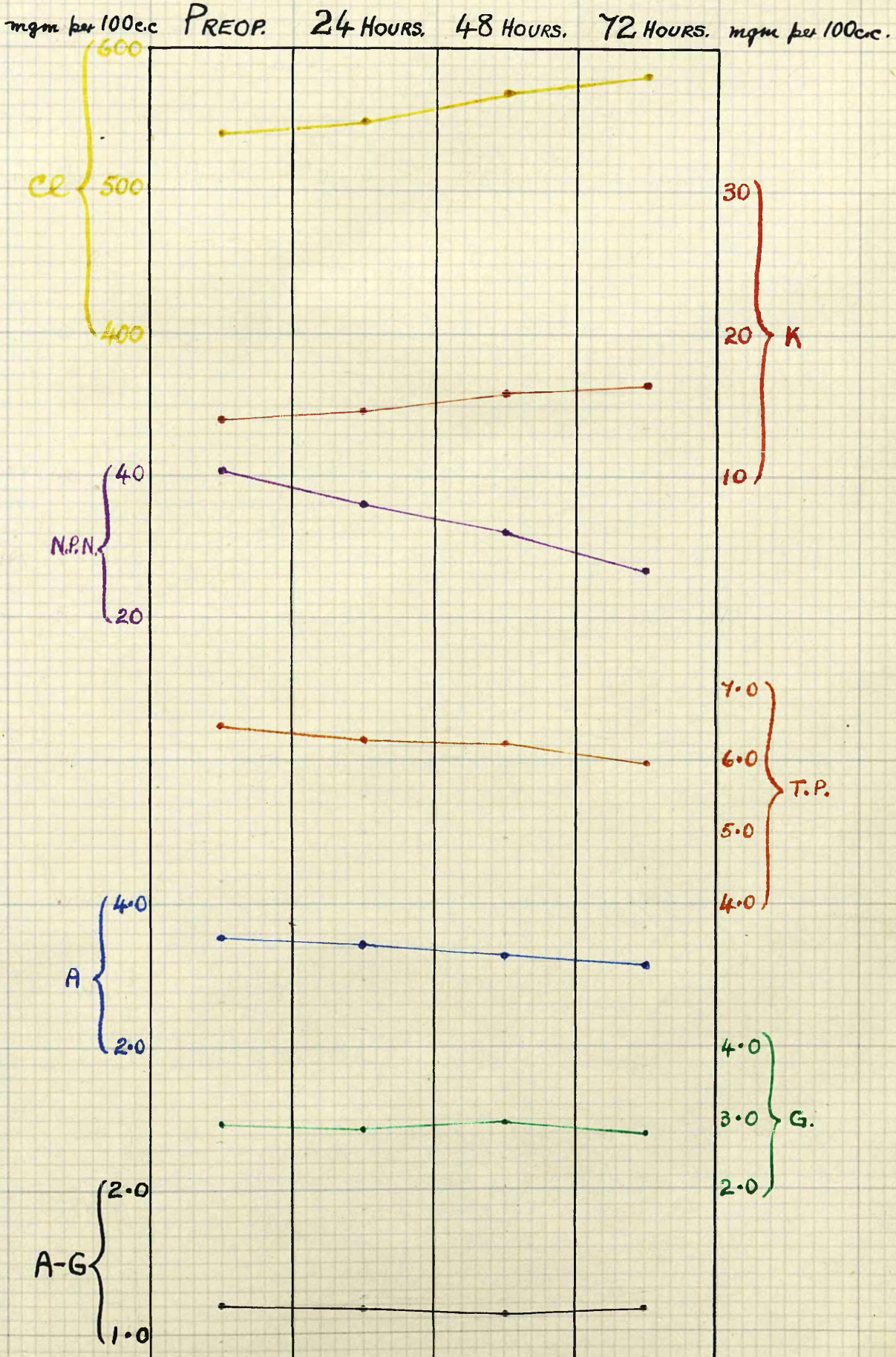
CASE 15.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Case 14.

Man, aged 62.

Constipated 5 days. Abdominal pains. Vomiting slight.

Carcinoma of descending colon.

Operation - caecostomy. Recovery uninterrupted.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	519	498	530	551
Potass.	22.29	21.11	20.3	17.6
N.P.N.	33.6	37.8	39.2	30.8
T.P.	5.90	6.05	5.84	5.58
Albumen.	3.26	3.31	3.14	2.79
Globulin.	2.64	2.74	2.70	2.79
A-G Ratio	1.24	1.21	1.16	1.00

Case 15.

Man, aged 74.

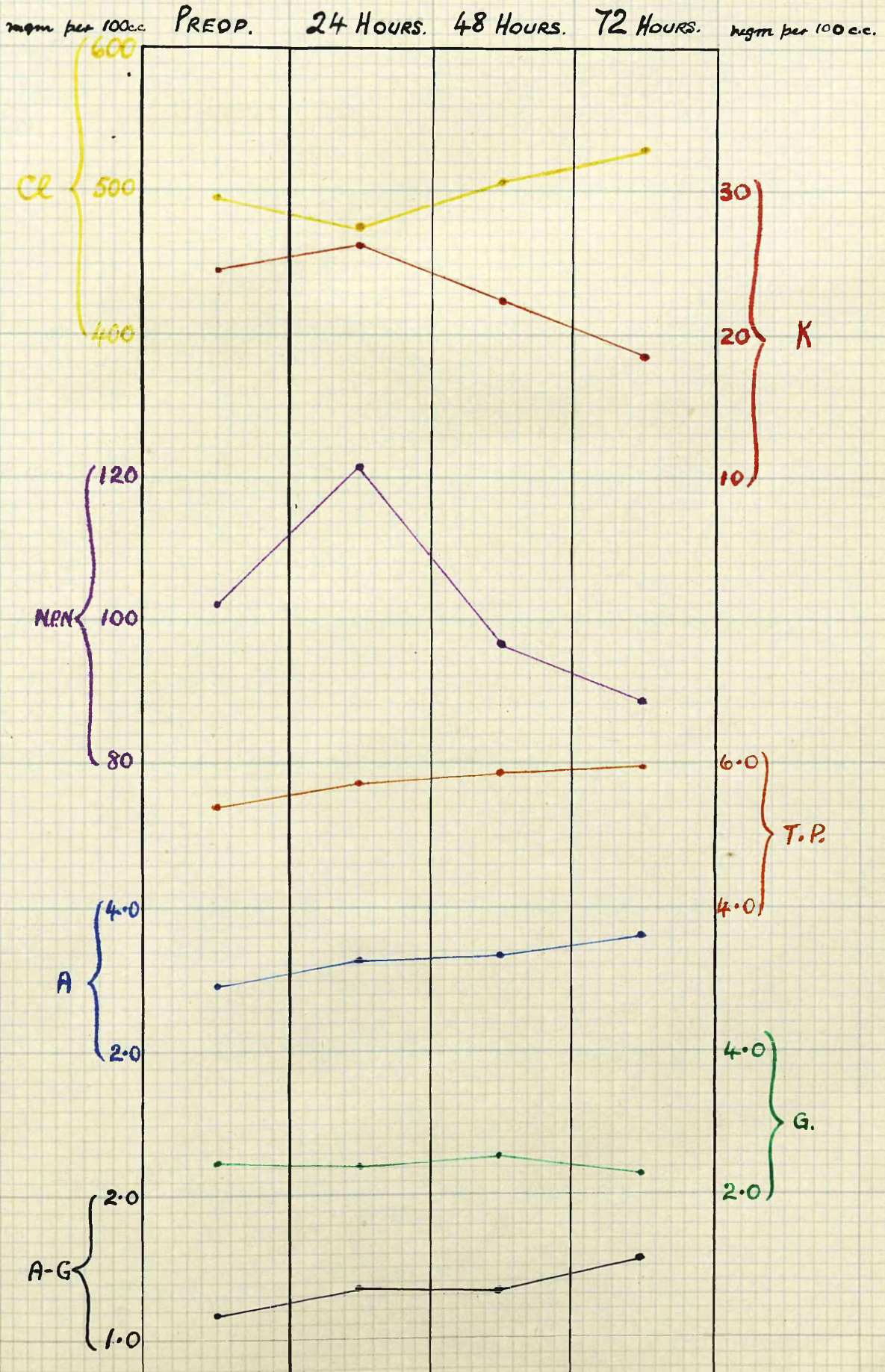
Constipated 5 days. Abdominal pain. Vomited once.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	551	572	583
Potass.	14.1	14.9	16.0	16.4
N.P.N.	40.6	36.4	32.2	26.6
T.P.	6.48	6.32	6.24	5.98
Albumen.	3.54	3.43	3.29	3.18
Globulin.	2.94	2.89	2.95	2.80
A-G Ratio	1.20	1.19	1.12	1.14

CASE 16.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mgm per 100 c.c.
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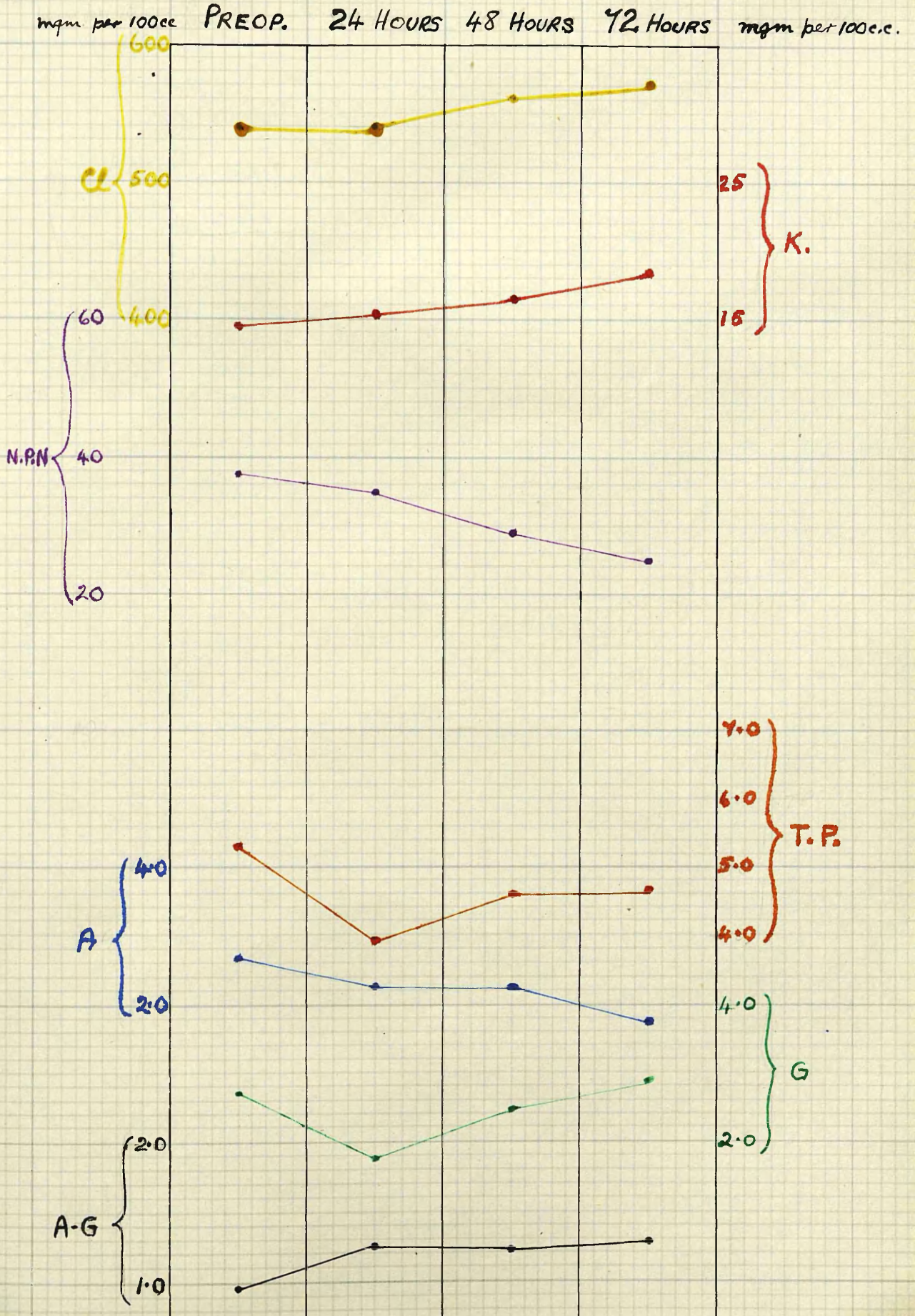
Case 17.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Case 16.

Man, aged 68.

Constipated 6 days. Abdominal pains. Vomiting marked.

Carcinoma of sigmoid. Much small bowel distension.

Operation - descending colostomy. Recovery slow.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	498	477	509	530
Potass.	24.6	26.2	22.3	18.4
N.P.N.	102.2	121.8	96.6	88.2
T.P.	5.40	5.70	5.87	5.92
Albumen.	2.94	3.28	3.34	3.61
Globulin.	2.46	2.42	2.53	2.31
A-G Ratio	1.19	1.36	1.32	1.56

Case 17.

Man, aged 60.

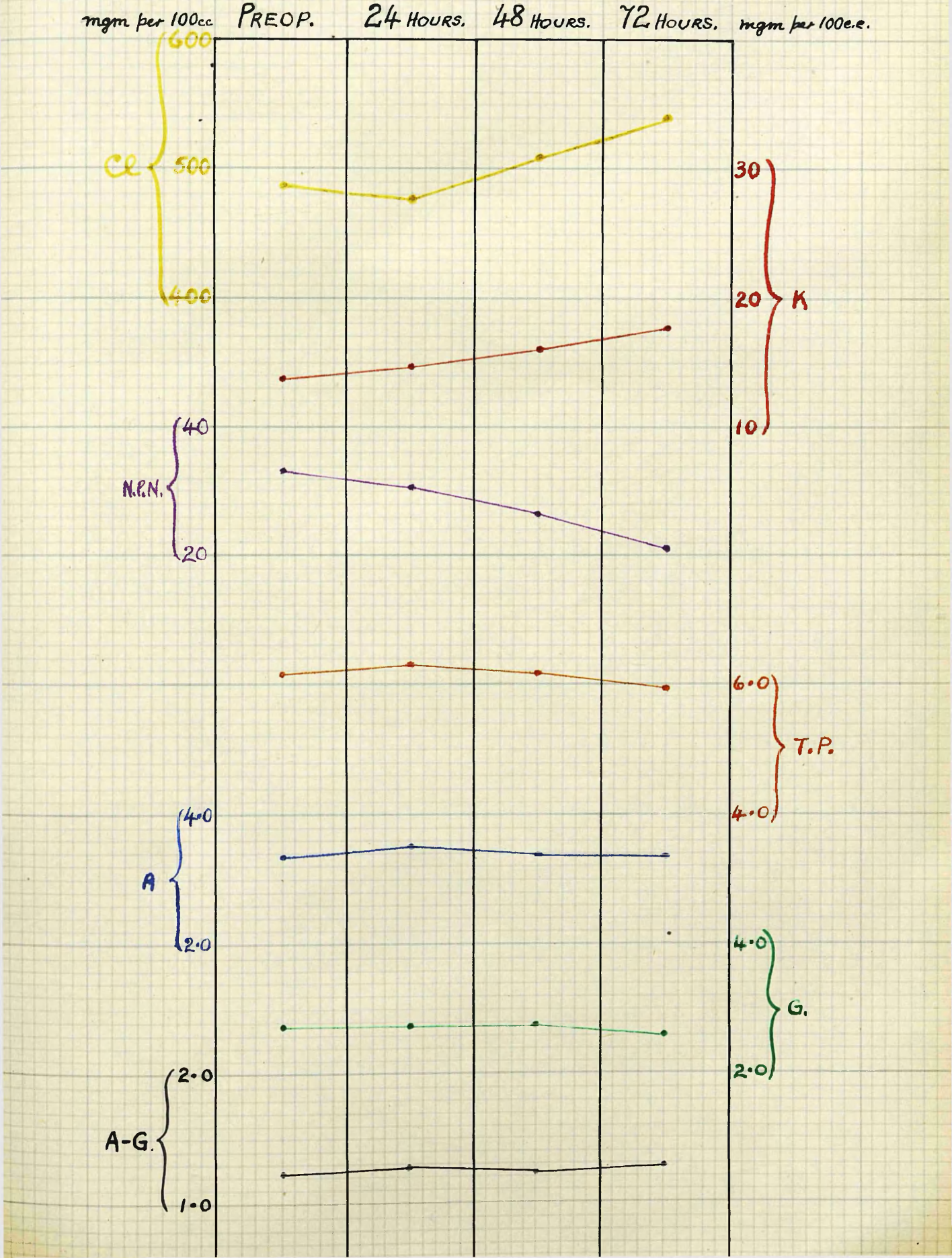
Constipated 5 days. Abdominal pains. Vomited once.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	541	562	572
Potass.	14.5	15.6	16.8	18.4
N.P.N.	37.8	35.0	29.4	25.2
T.P.	5.36	3.98	4.62	4.66
Albumen.	2.64	2.23	2.21	1.74
Globulin.	2.72	1.75	2.41	2.92
A-G Ratio	0.97	1.27	0.92	0.60

CASE 18.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PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100c.c.
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N.P.N.
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T.P.
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2·0
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G.
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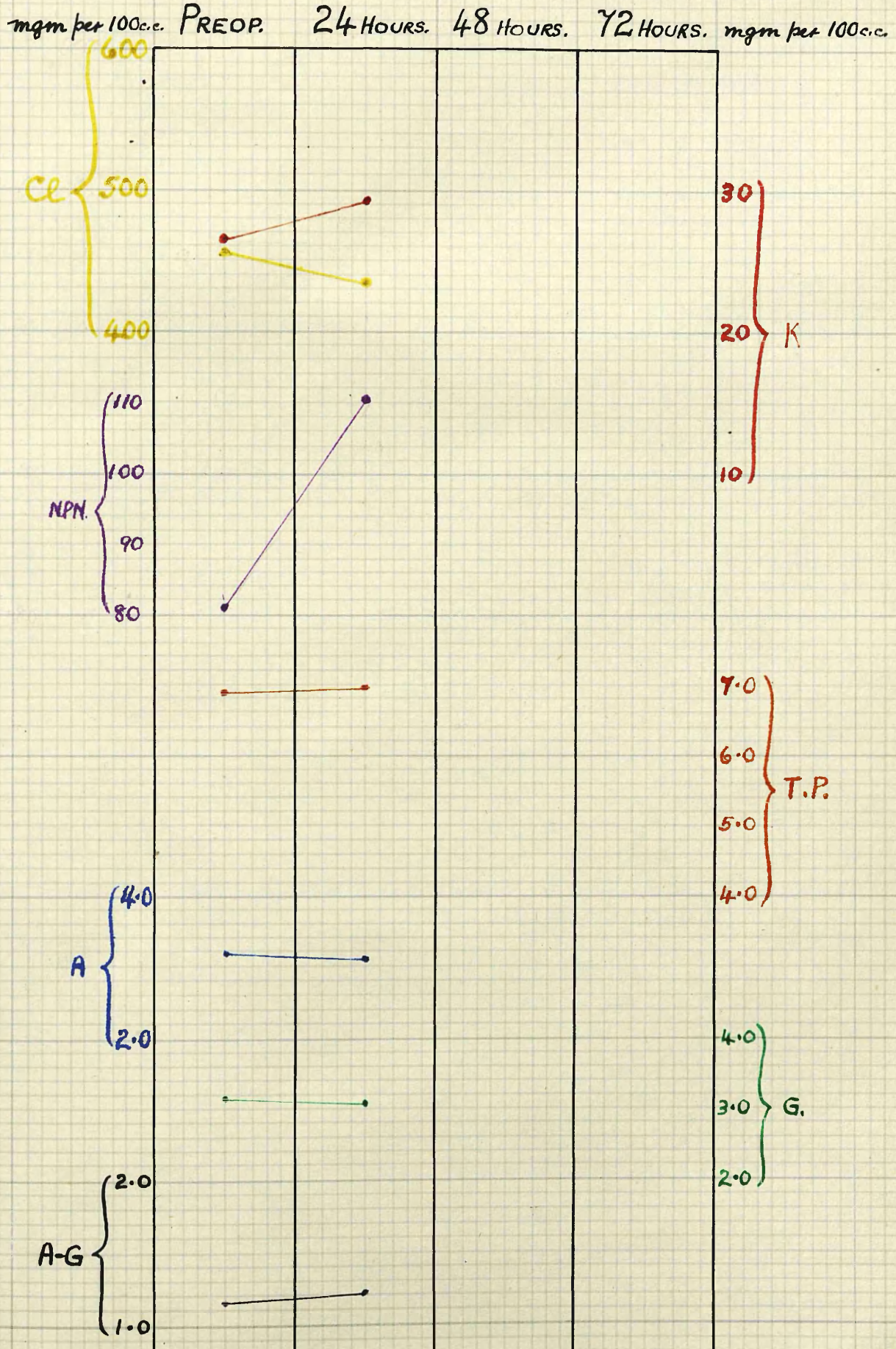
2·0
A-G.
1·0

CASE 19.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PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100c.c.
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NPN.
110
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K
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7·0
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T.P.
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4·0
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3·0
G.
2·0
A-G
2·0
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Case 18.

Woman, aged 63.

Constipated 6 days. Abdominal pain. Vomiting marked.

Carcinoma of hepatic flexure.

Operation - caecostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	488	477	510	541
Potass.	13.7	14.9	16.0	17.6
N.P.N.	33.6	30.8	26.6	21.0
T.P.	6.14	6.27	6.19	5.98
Albumen.	3.38	3.52	3.41	3.36
Globulin	2.76	2.75	2.78	2.62
A-G Ratio	1.23	1.27	1.23	1.28

Case 19.

Woman, aged 75.

Constipated 4 days. Abdominal pains. Vomiting profuse.

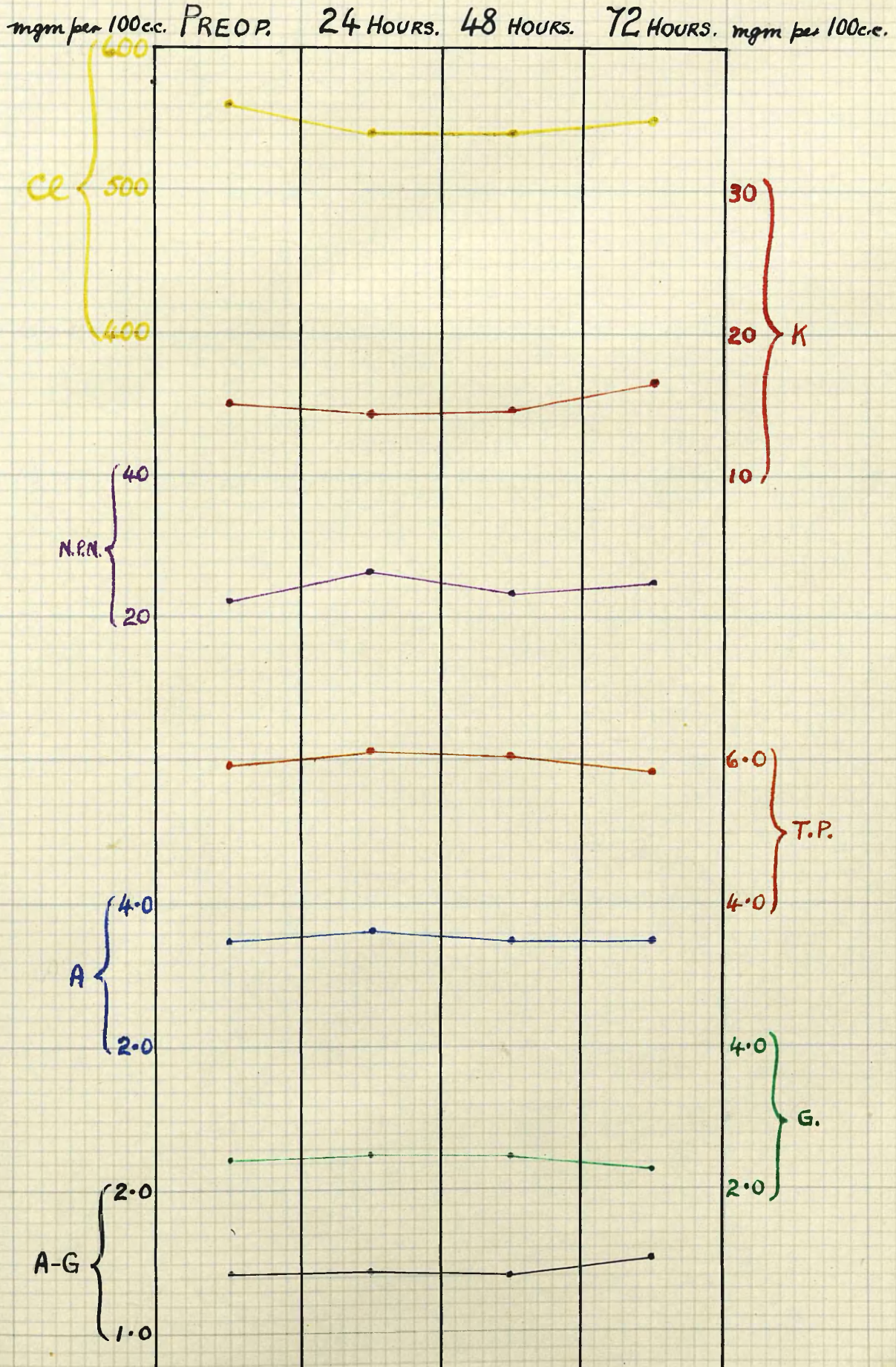
Carcinoma of splenic flexure.

Operation - transverse colostomy.

Death on 2nd day after operation.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	456	435	---	---
Potass.	26.2	29.3	---	---
N.P.N.	81.2	110.6	---	---
T.P.	6.87	6.94	---	---
Albumen.	3.68	3.82	---	---
Globulin.	3.19	3.12	---	---
A-G Ratio	1.15	1.22	---	---

CASE 20.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PREOP.
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48 HOURS.
72 HOURS.
mgm per 100 c.c.
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G.
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A-G
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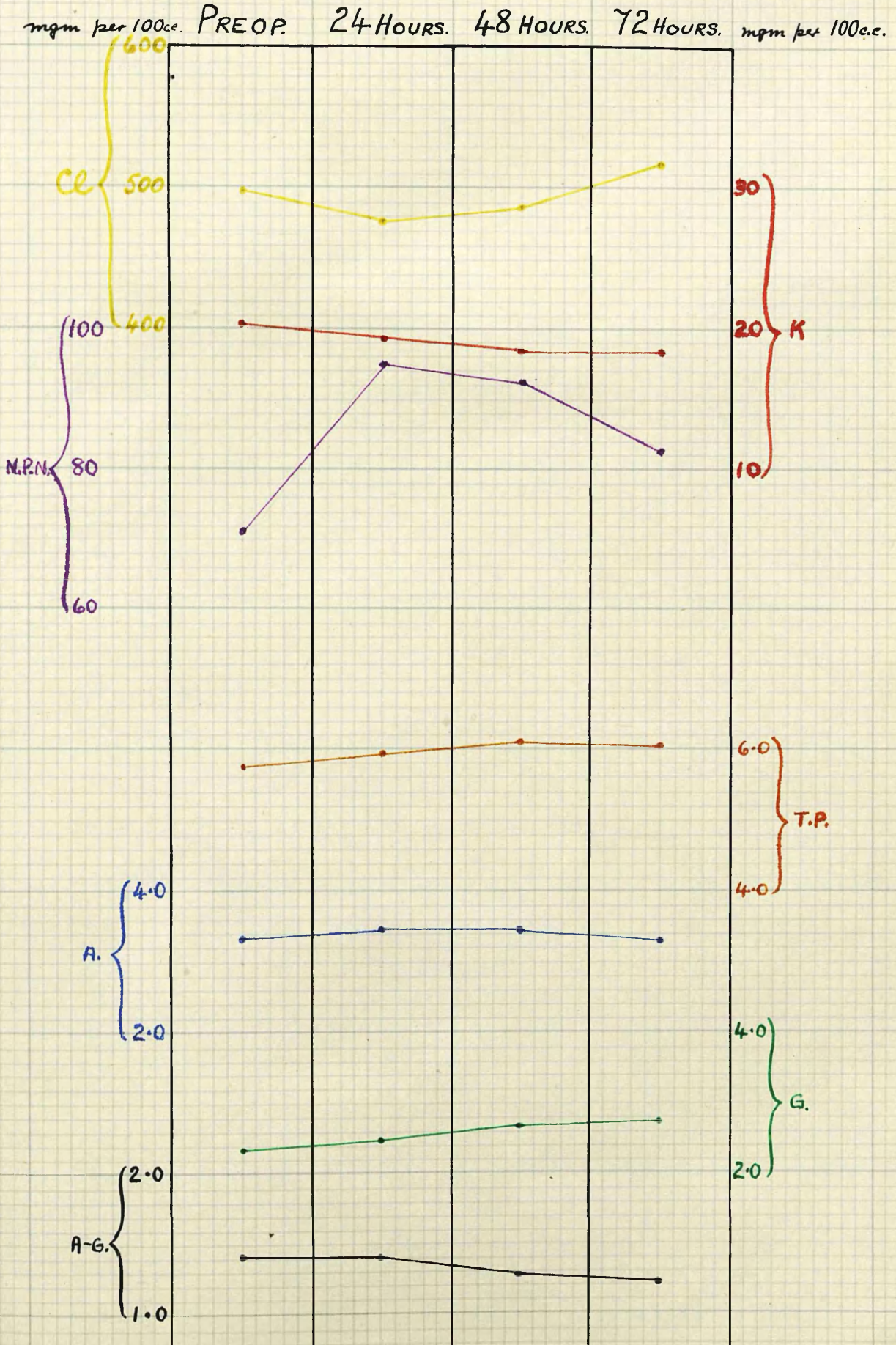
Case 21.


mgm per 100cc.
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24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100c.c.
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T.P.
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4·0
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2·0
2·0
A-G.
1·0

Case 20.

Man, aged 69.

Constipated 3 days. Abdominal pains. Vomited once.

Carcinoma of splenic flexure.

Operation - transverse colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	562	541	541	551
Potass.	15.2	14.5	14.9	16.8
N.P.N.	22.4	26.6	23.8	25.2
T.P.	5.92	6.14	6.06	5.82
Albumen.	3.47	3.61	3.54	3.51
Globulin.	2.45	2.53	2.52	2.31
A-G Ratio	1.42	1.43	1.40	1.52

Case 21.

Man, aged 56.

Constipated 6 days. Abdominal pains. Vomiting marked.

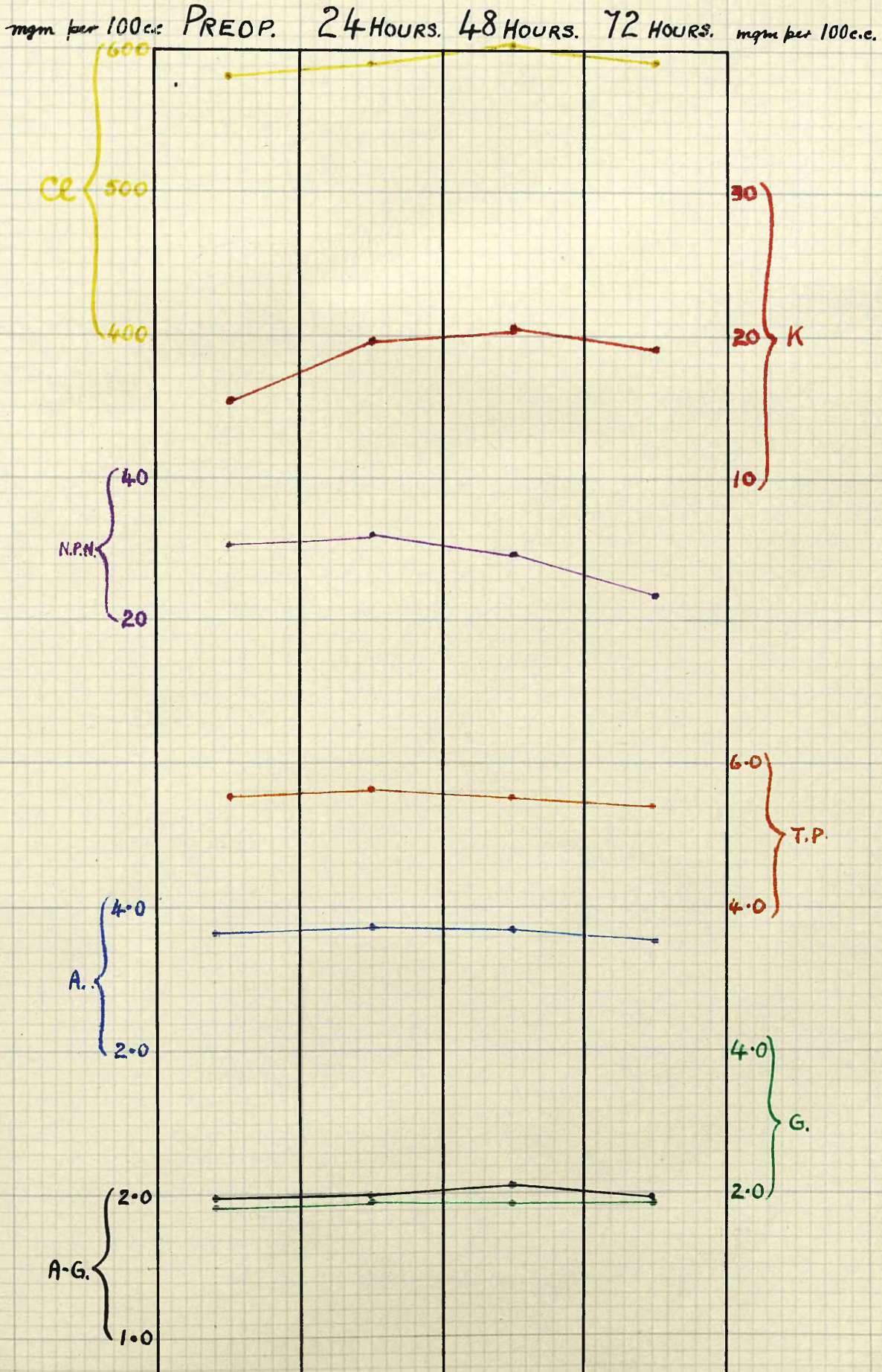
Carcinoma of transverse colon.

Operation - caecostomy. Much small bowel distension.

Recovery slow.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	498	477	488	519
Potass.	20.3	19.2	18.4	18.4
N.P.N.	71.4	95.2	92.4	82.6
T.P.	5.76	5.94	6.11	6.04
Albumen.	3.38	3.47	3.44	3.32
Globulin.	2.38	2.47	2.67	2.72
A-G Ratio	1.42	1.40	1.29	1.22

CASE 22.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PREOP.
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48 HOURS.
72 HOURS.
mgm per 100 c.c.
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N.P.N.
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A-G.
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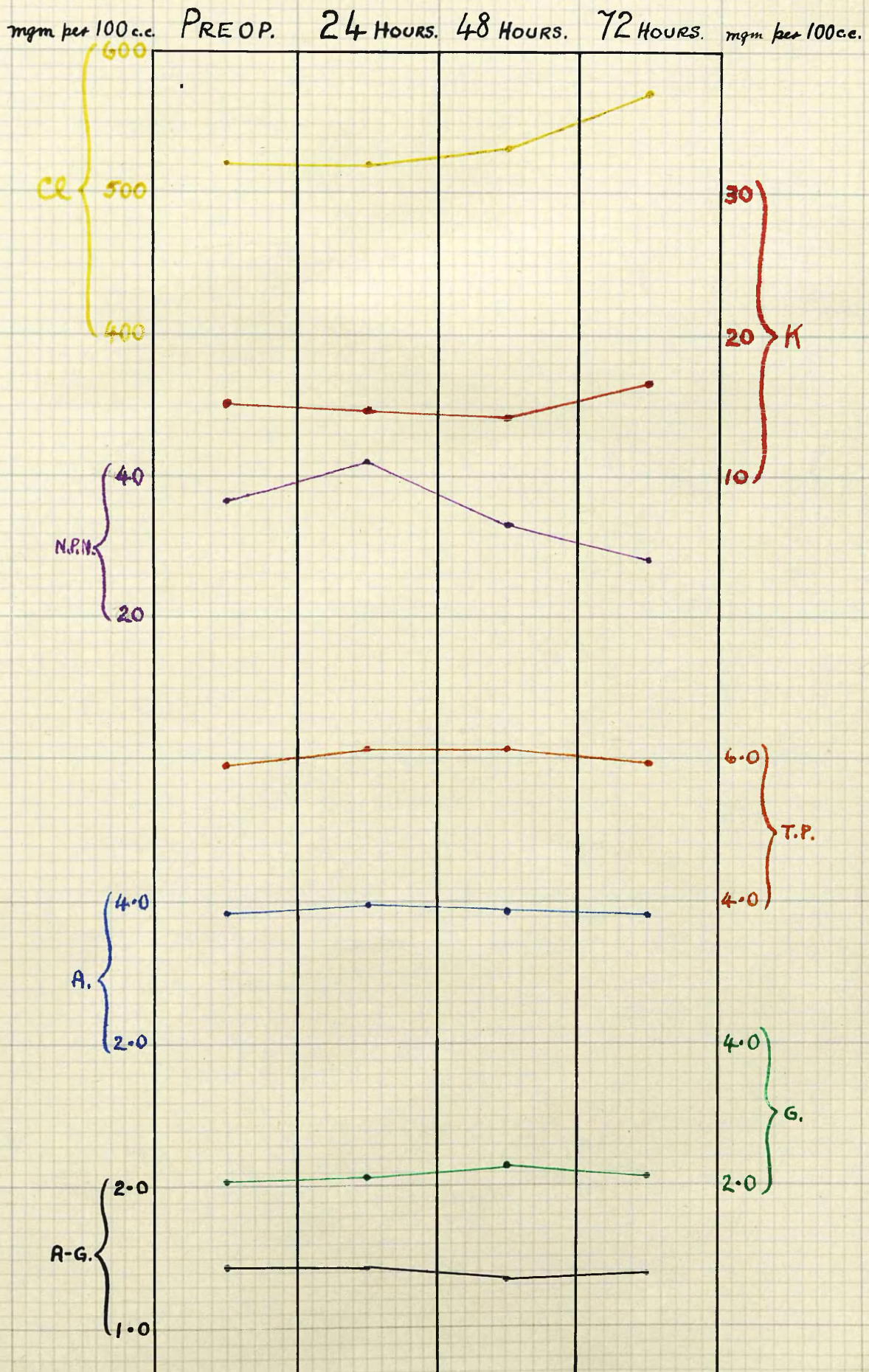
CASE 23.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72 HOURS.
mgm per 100 c.c.
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A-G.
1·0

Case 22.

Man, aged 49.

Constipated 5 days. Abdominal pains. Vomited once.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	583	593	604	593
Potass.	15.6	19.9	20.7	19.2
N.P.N.	30.8	32.2	29.4	23.8
T.P.	5.53	5.62	5.54	5.41
Albumen.	3.67	3.74	3.71	3.58
Globulin.	1.86	1.88	1.83	1.83
A-G Ratio	1.97	1.99	2.06	1.96

Case 23.

Man, aged 67.

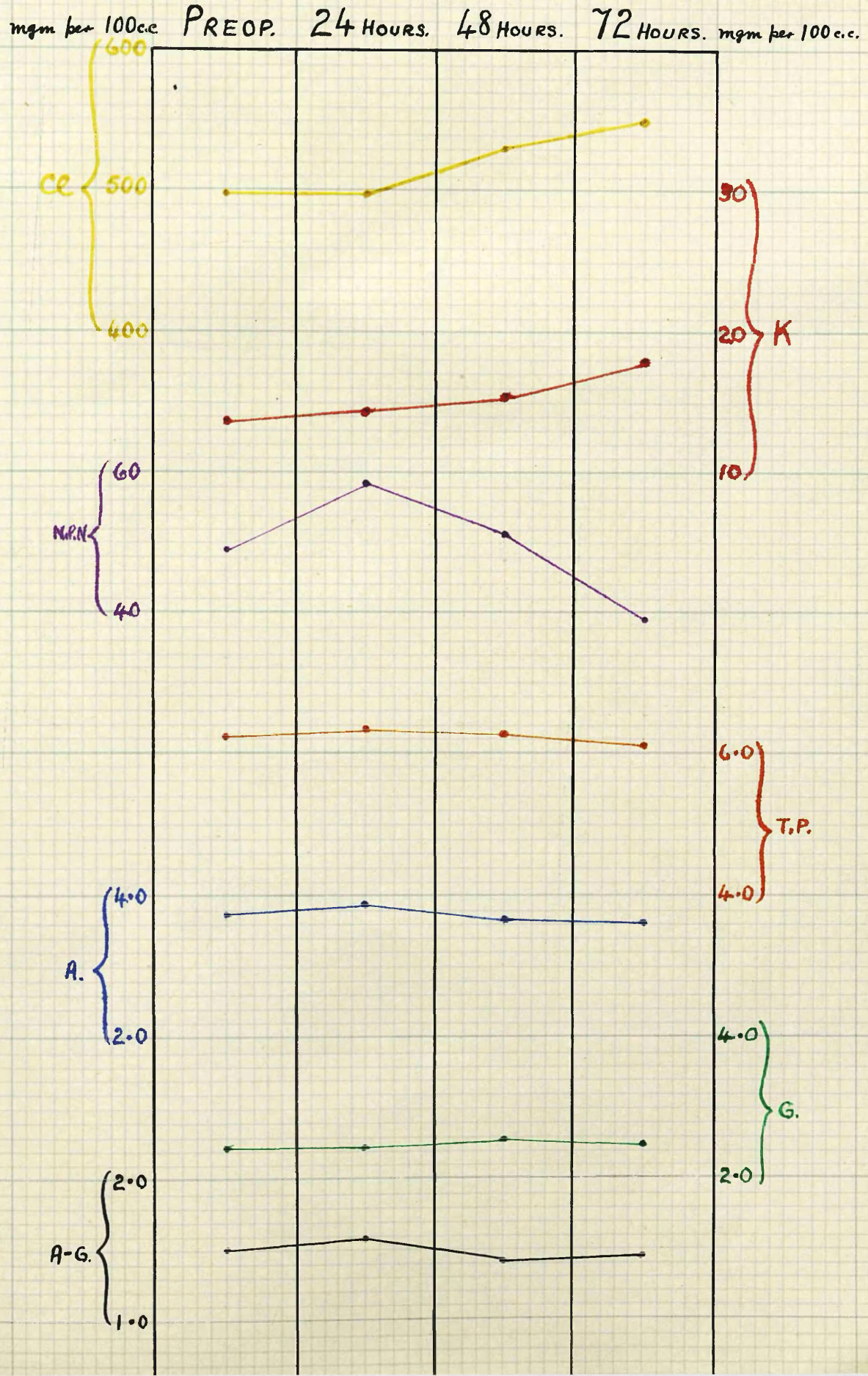
Constipated 5 days. Abdominal pains. Vomiting slight.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	541	551	572
Potass.	15.2	14.9	14.5	16.8
N.P.N.	36.4	42.0	33.6	28.0
T.P.	5.92	6.14	6.18	5.98
Albumen.	3.86	3.98	3.89	3.82
Globulin.	2.06	2.16	2.29	2.16
A-G Ratio	1.87	1.84	1.70	1.77

CASE 24.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72 HOURS.
mgm per 100 c.c.
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A-G.
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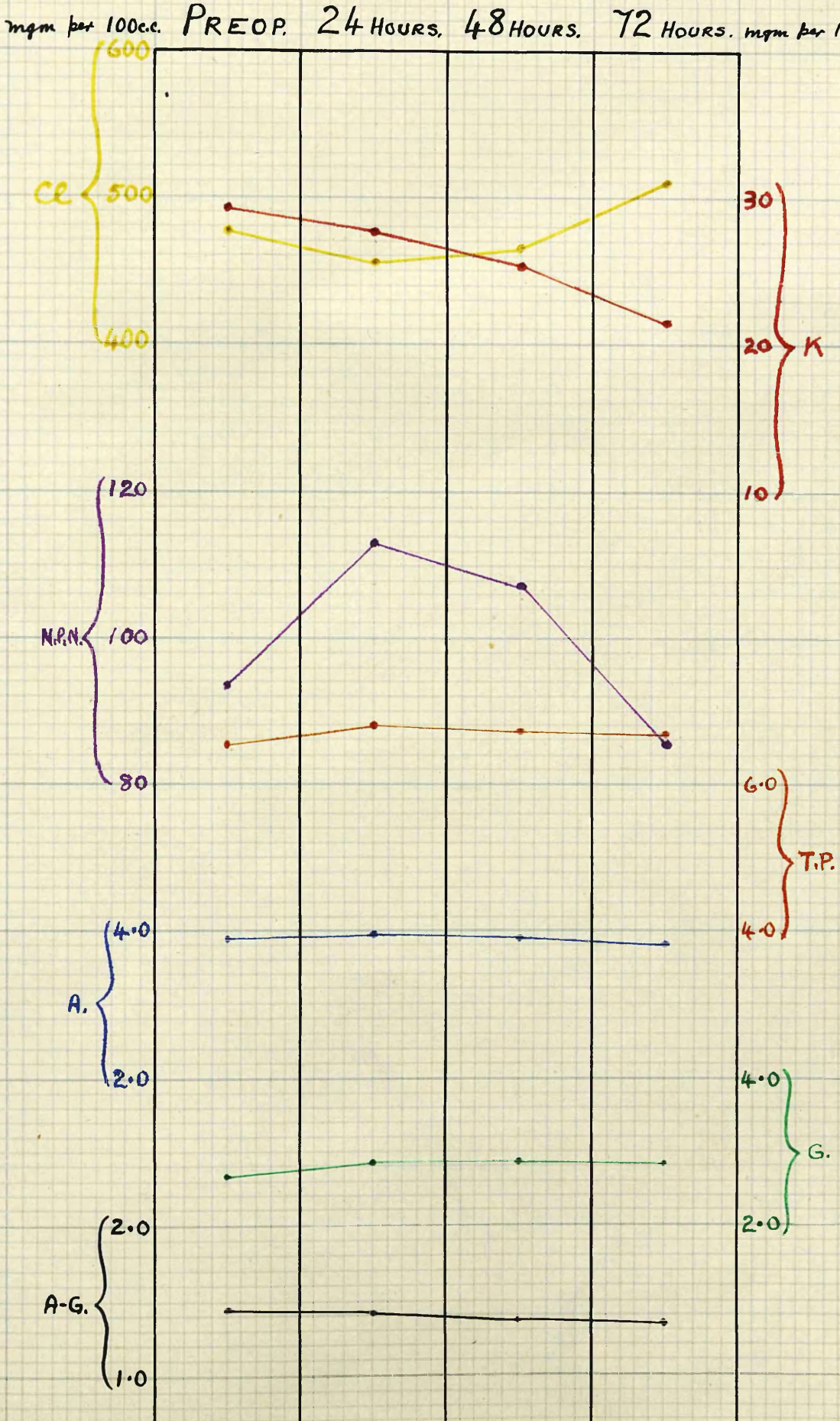
Case 25.


mgm per 100c.c.
PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100c.c.
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2·0
A-G.
1·0

Case 24.

Woman, aged 71.

Constipated 4 days. Abdominal pains. Vomiting marked.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	498	498	530	551
Potass.	13.7	14.5	15.6	18.0
N.P.N.	49.0	58.8	51.8	39.2
T.P.	6.21	6.34	6.27	6.14
Albumen	3.75	3.89	3.68	3.62
Globulin.	2.46	2.45	2.59	2.52
A-G Ratio	1.52	1.59	1.42	1.44

Case 25.

Man, aged 57.

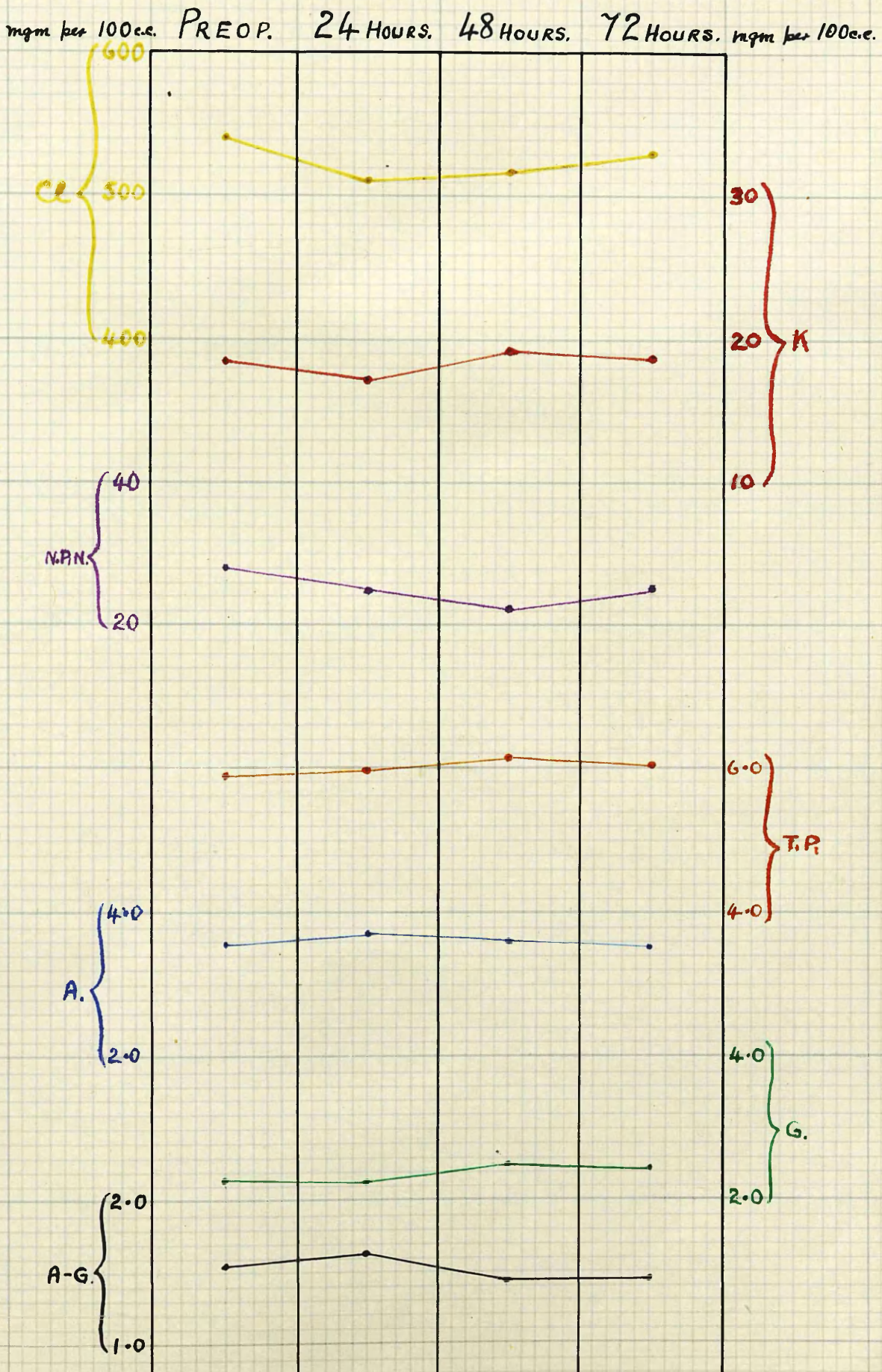
Constipated 6 days. Abdominal pains. Vomiting marked.

Carcinoma of sigmoid. Much small bowel distension.

Operation - descending colostomy. Recovery stormy.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	477	456	467	510
Potass.	29.3	27.8	25.4	21.5
N.P.N.	93.8	113.4	107.8	85.4
T.P.	6.58	6.81	6.76	6.64
Albumen.	3.91	3.98	3.92	3.81
Globulin.	2.67	2.83	2.84	2.83
A-G Ratio	1.46	1.41	1.38	1.35

CASE 26.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72 HOURS.
mgm per 100 c.c.
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N.P.N.
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A-G.
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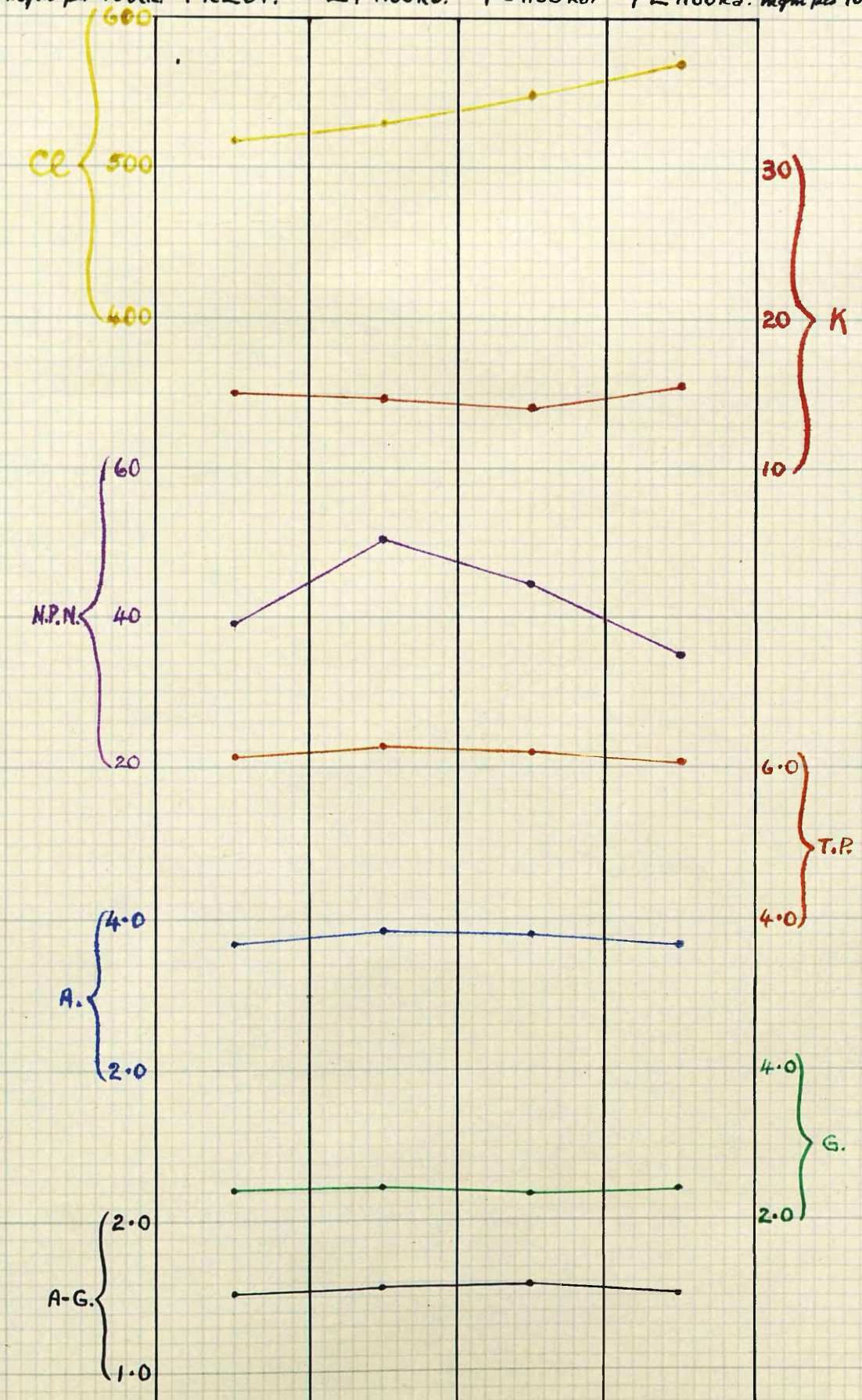
CASE 27.


mgm per 100c.c.
PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100c.c.
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A-G.
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Case 26.

Man, aged 60.

Constipated 4 days. Abdominal pains. Vomited once.

Carcinoma of splenic flexure.

Operation - transverse colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	510	519	530
Potass.	18.4	17.2	19.2	18.8
N.P.N.	28.0	25.2	22.4	25.2
T.P.	6.58	6.81	6.76	6.64
Albumen.	3.91	3.98	3.92	3.81
Globulin.	2.67	2.83	2.84	2.83
A-G Ratio	1.46	1.41	1.38	1.35

Case 27.

Man, aged 68.

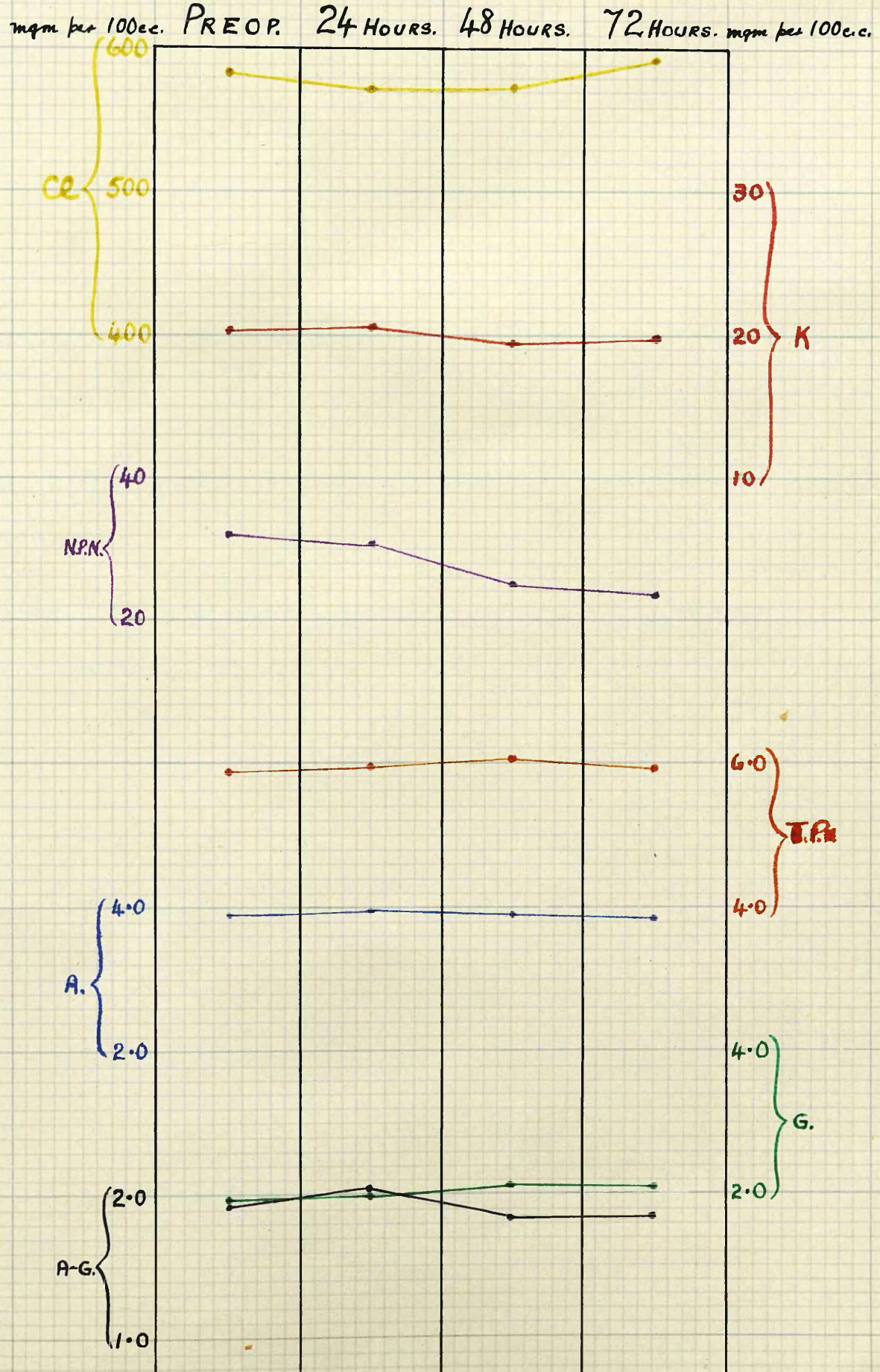
Constipated 4 days. Abdominal pains. Vomiting slight.

Carcinoma of hepatic flexure.

Operation - caecostomy. Recovery uninterrupted.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	519	530	551	572
Potass.	15.2	14.9	14.1	15.6
N.P.N.	39.2	50.4	44.8	35.0
T.P.	6.14	6.27	6.20	6.11
Albumen.	3.72	3.84	3.81	3.69
Globulin.	2.42	2.43	2.29	2.42
A-G Ratio	1.54	1.58	1.59	1.52

Case 28.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24 HOURS.
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mgm per 100c.c.
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A-G.
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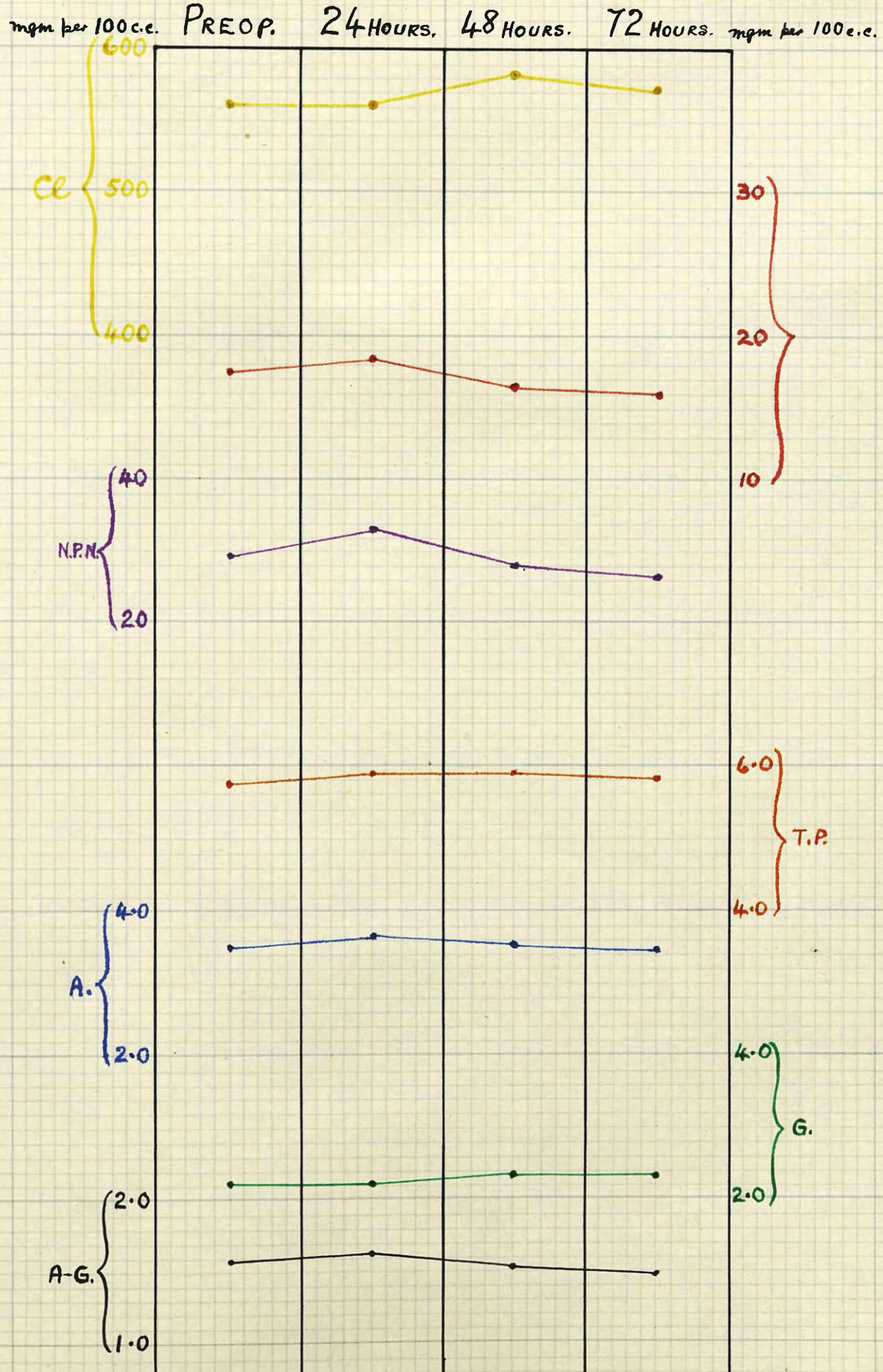
CASE 29.


mgm per 100 c.c.
PREOP.
24 HOURS.
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72 HOURS.
mgm per 100 c.c.
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N.P.N.
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A-G.
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Case 28.

Woman, aged 64.

Constipated 4 days. Abdominal pains. Vomited once.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	583	572	572	593
Potass.	20.3	20.7	19.5	19.9
N.P.N.	32.2	30.8	25.2	23.8
T.P.	5.84	5.96	6.05	5.94
Albumen.	3.88	3.97	3.92	3.86
Globulin.	1.96	1.99	2.13	2.08
A-G Ratio	1.98	2.00	1.84	1.85

Case 29.

Man, aged 72.

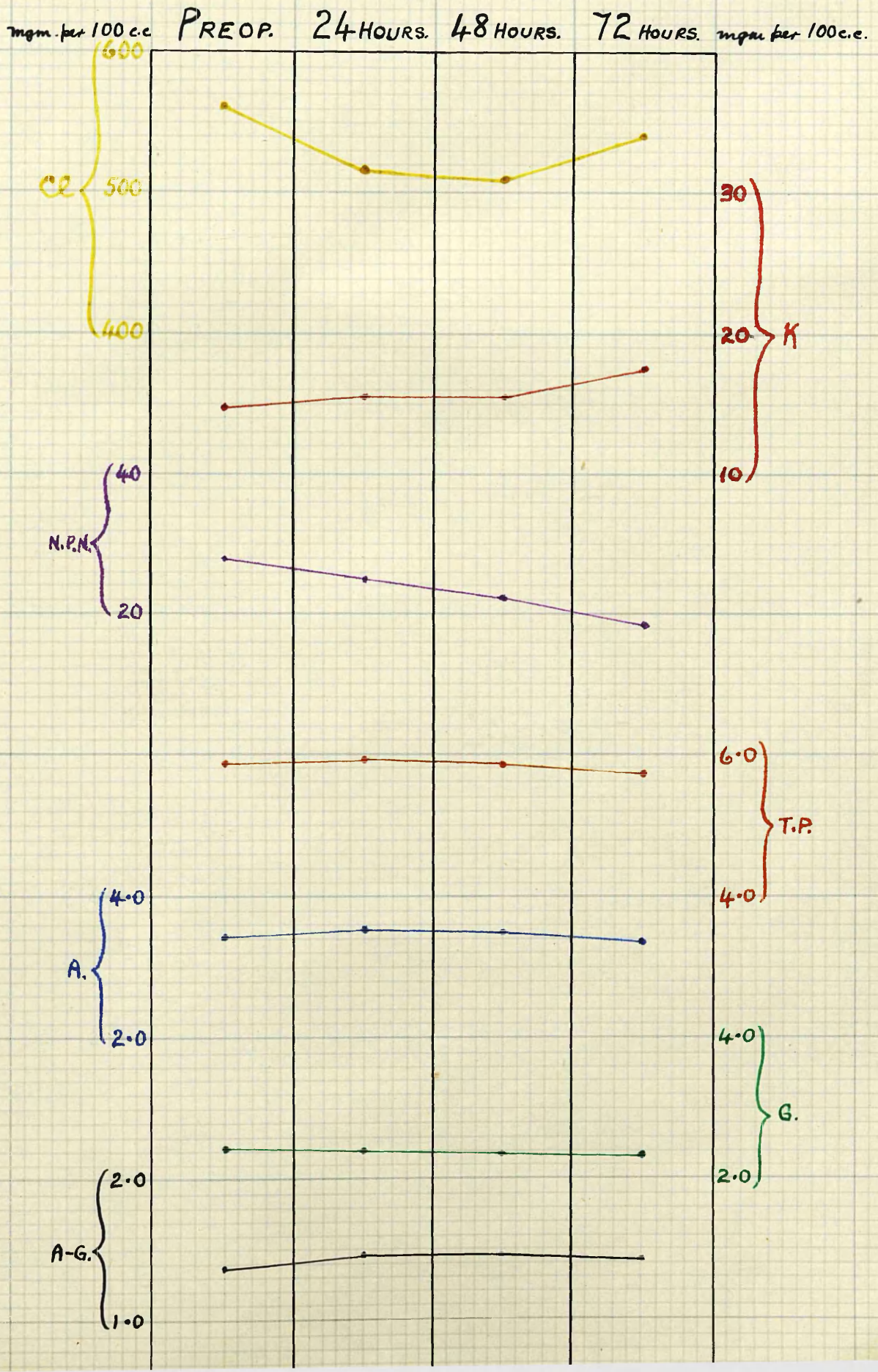
Constipated 3 days. Abdominal pains. Vomiting slight.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	562	562	583	572
Potass.	17.6	18.4	16.8	16.0
N.P.N.	29.4	33.6	28.0	26.6
T.P.	5.76	5.87	5.92	5.81
Albumen.	3.54	3.65	3.58	3.48
Globulin.	2.22	2.22	2.34	2.33
A-G Ratio	1.59	1.64	1.53	1.49

CASE 30.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PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100 c.c.
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N.P.N.
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T.P.
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G.
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2·0
A-G.
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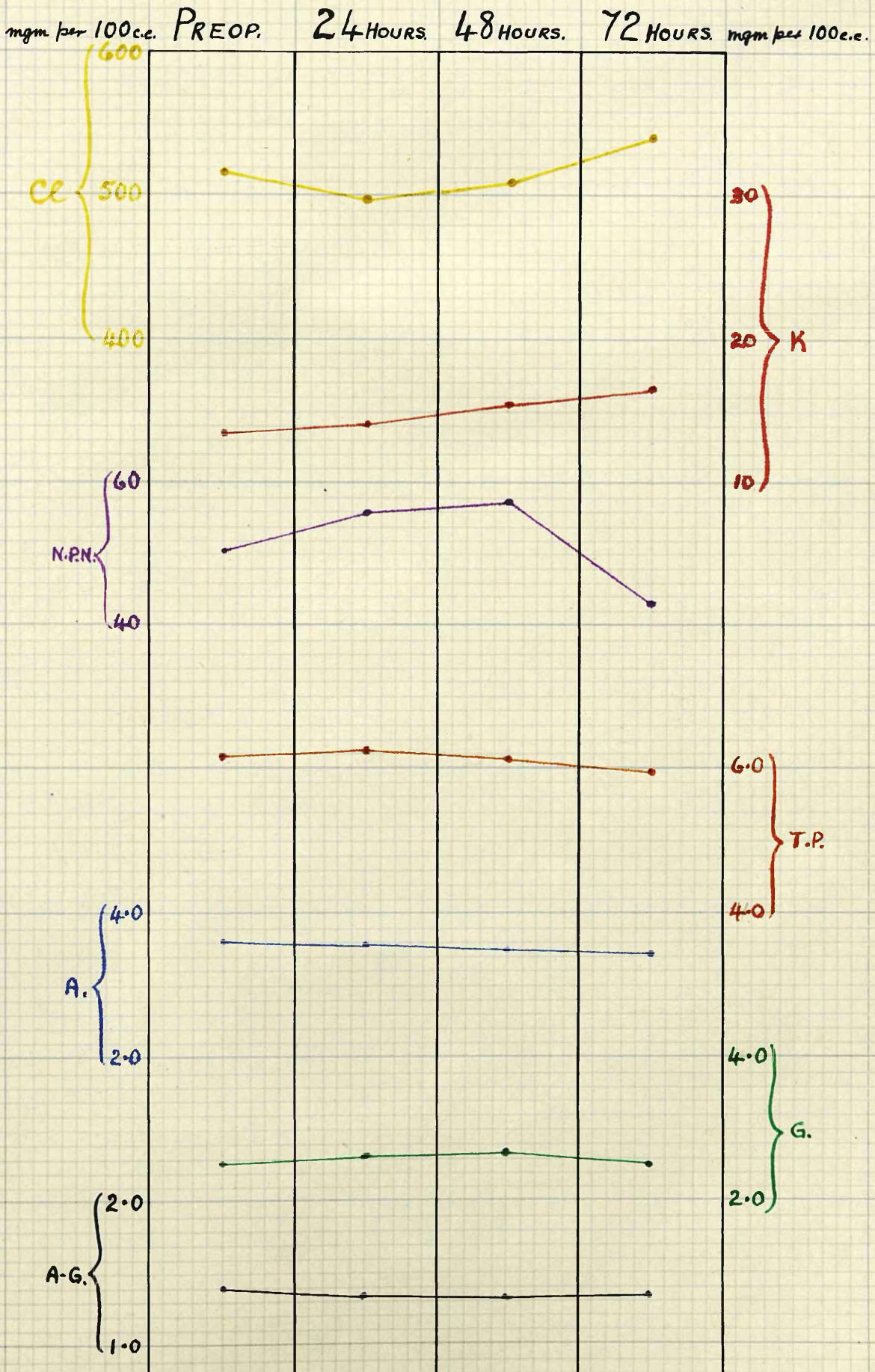
Case 31.


mgm per 100 c.c.
Preop.
24 Hours.
48 Hours.
72 Hours.
mgm per 100 c.c.
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K
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N.P.N.
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T.P.
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4.0
2.0
A-G.
2.0
1.0

Case 30.

Woman, aged 61.

Constipated 5 days. Abdominal pains. Vomited slightly.

Carcinoma of transverse colon.

Operation - colostomy. Recovery uninterrupted.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	562	519	510	541
Potass.	14.9	15.6	15.6	17.6
N.P.N.	28.0	25.2	22.4	18.2
T.P.	5.86	5.93	5.85	5.76
Albumen.	3.41	3.53	3.47	3.39
Globulin.	2.45	2.40	2.38	2.37
A-G Ratio	1.39	1.47	1.46	1.43

Case 31.

Man, aged 59.

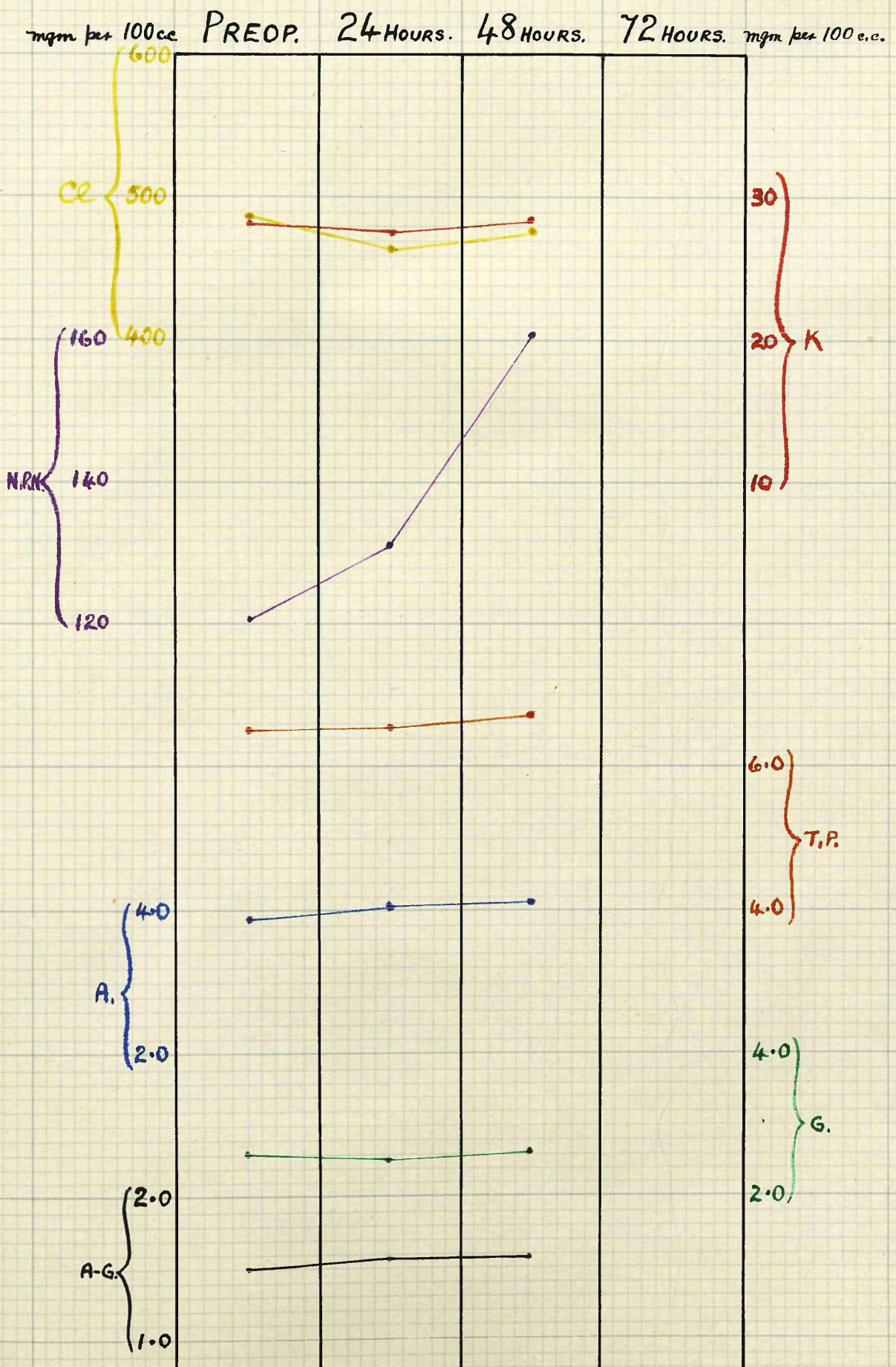
Constipated 5 days. Abdominal pains. Vomiting slight.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	519	498	510	541
Potass.	13.7	14.1	15.6	16.8
N.P.N.	50.4	56.0	57.4	43.4
T.P.	6.18	6.21	6.16	5.98
Albumen.	3.61	3.58	3.49	3.41
Globulin.	2.57	2.63	2.67	2.57
A-G Ratio	1.40	1.36	1.31	1.33

CASE 32.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48 HOURS.
72 HOURS.
mgm per 100 c.c.
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Cl
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N.P.N.
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6.0
T.P.
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4.0
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A-G.
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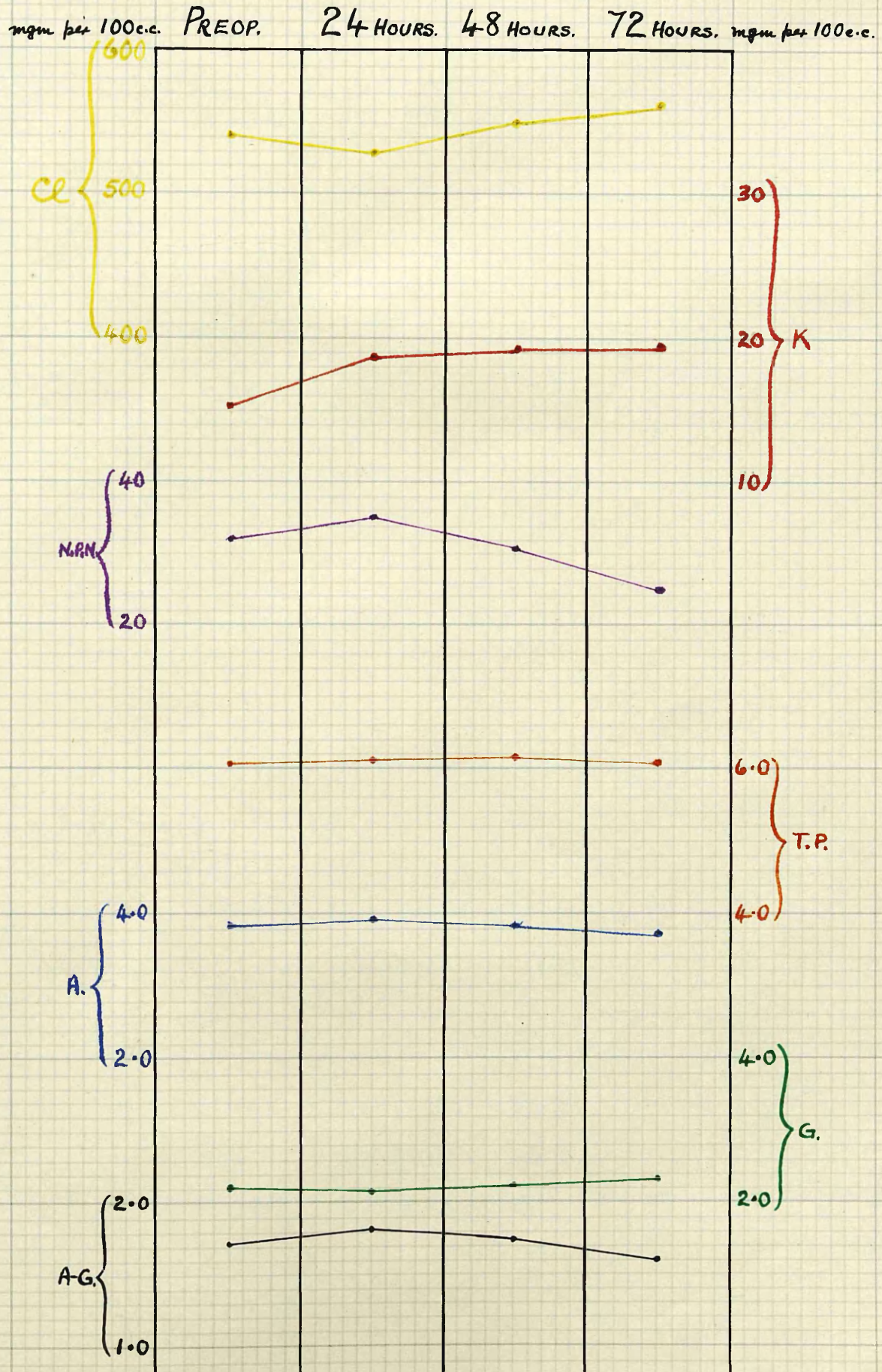
CASE 33.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PREOP.
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48 HOURS.
72 HOURS.
mgm per 100 c.c.
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T.P.
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G.
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2·0
A-G.
1·0

Case 32.

Man, aged 66.

Constipated 6 days. Abdominal pains. Vomiting marked.

Carcinoma of descending colon.

Operation - caecostomy. Much small bowel distension.

Death on 3rd day after operation.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	488	466	477	---
Potass	28.5	27.8	28.5	---
N.P.N.	120.4	131.6	161.0	---
T.P.	6.52	6.59	6.71	---
Albumen.	3.92	4.03	4.10	---
Globulin.	2.60	2.56	2.61	---
A-G Ratio	1.51	1.57	1.57	---

Case 33.

Man, aged 70.

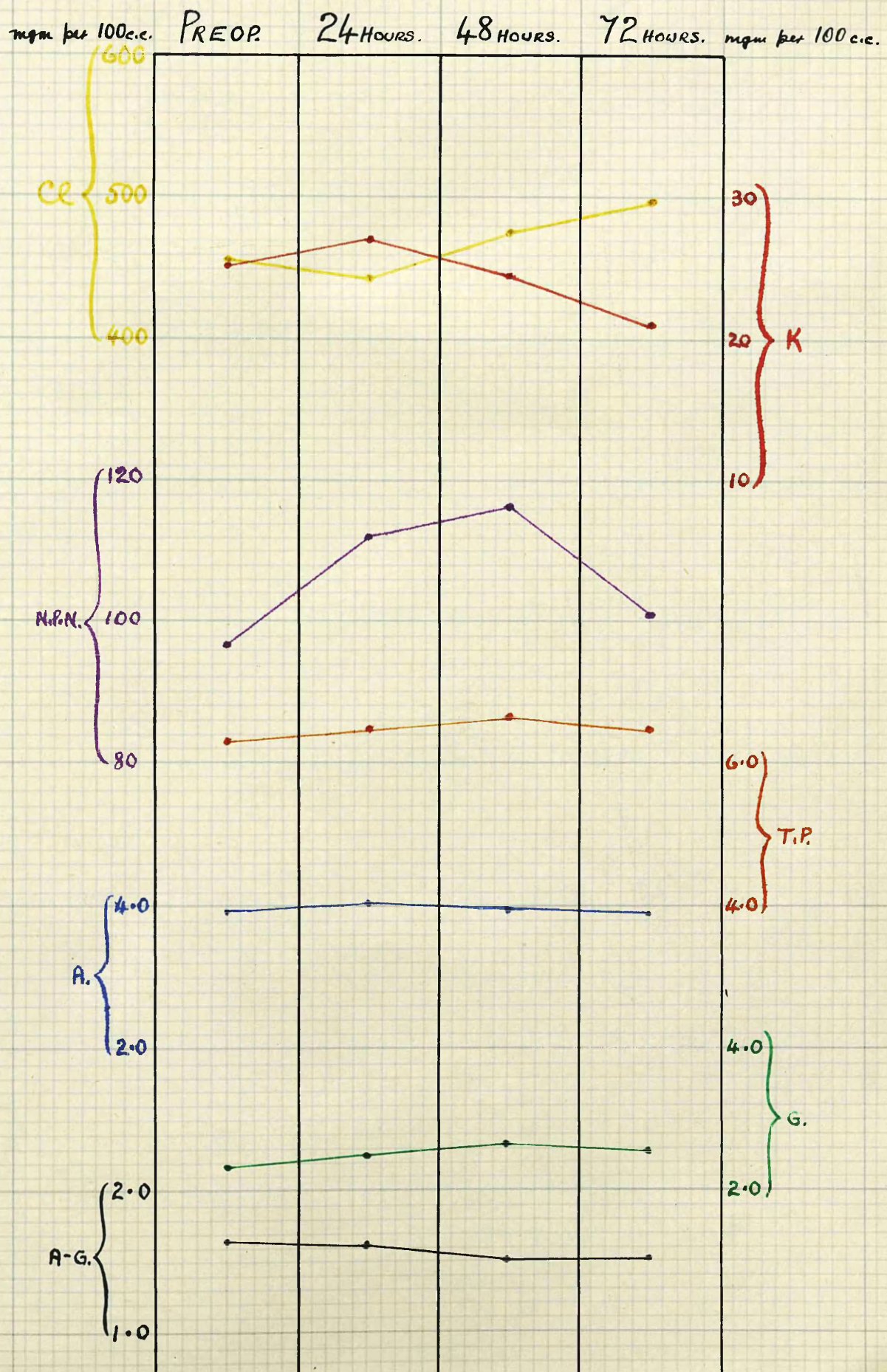
Constipated 4 days. Abdominal pains. Vomited once.

Carcinoma of descending colon.

Operation - transverse colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	530	551	562
Potass.	15.6	18.8	19.2	19.5
N.P.N.	32.2	35.0	30.8	25.2
T.P.	6.04	6.12	6.18	6.09
Albumen.	3.83	3.94	3.86	3.75
Globulin.	2.21	2.18	2.22	2.34
A-G Ratio.	1.73	1.81	1.74	1.60

CASE 34.


mgm per 100 c.c.
PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100 c.c.
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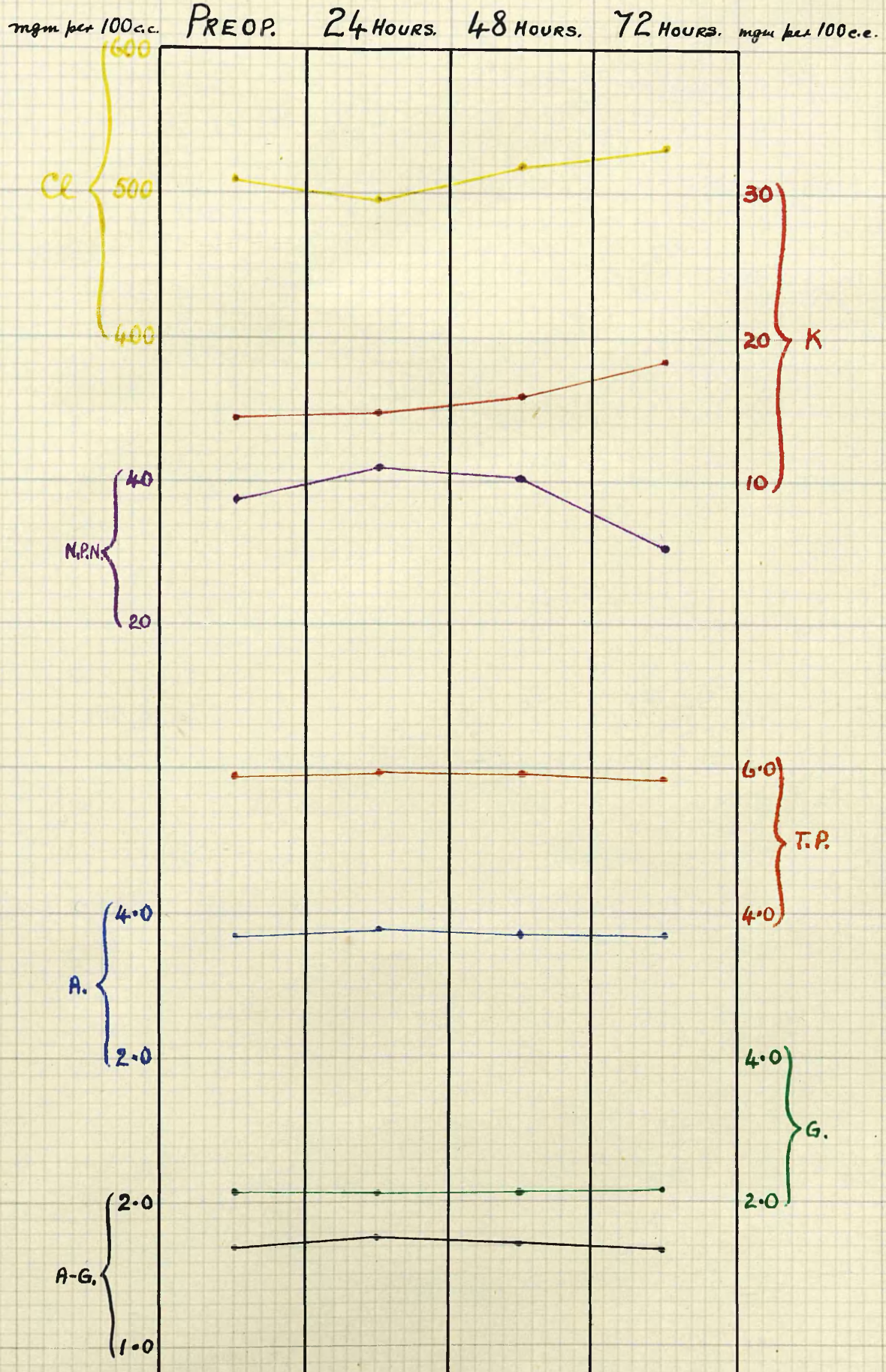
Cl
500
400
30
20
K
10
120
N.P.N.
100
80
6·0
T.P.
4·0
4·0
A.
2·0
4·0
G.
2·0
2·0
A-G.
1·0

Case 35.


mgm per 100 c.c.
Preop.
24 Hours.
48 Hours.
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mgm per 100 c.c.
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N.P.N.
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A-G.
1·0

Case 34.

Man aged 70.

Constipated 4 days. Abdominal pains. Vomiting profuse.

Carcinoma of splenic flexure. Much ileal distension.

Operation - transverse colostomy. Convalescence stormy.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	456	446	477	498
Potass.	25.4	27.0	24.6	21.1
N.P.N.	96.6	112.0	116.2	100.8
T.P.	6.31	6.49	6.62	6.51
Albumen.	3.94	4.01	3.98	3.92
Globulin.	2.37	2.48	2.64	2.59
A-G Ratio	1.66	1.62	1.51	1.51

Case 35.

Man, aged 62.

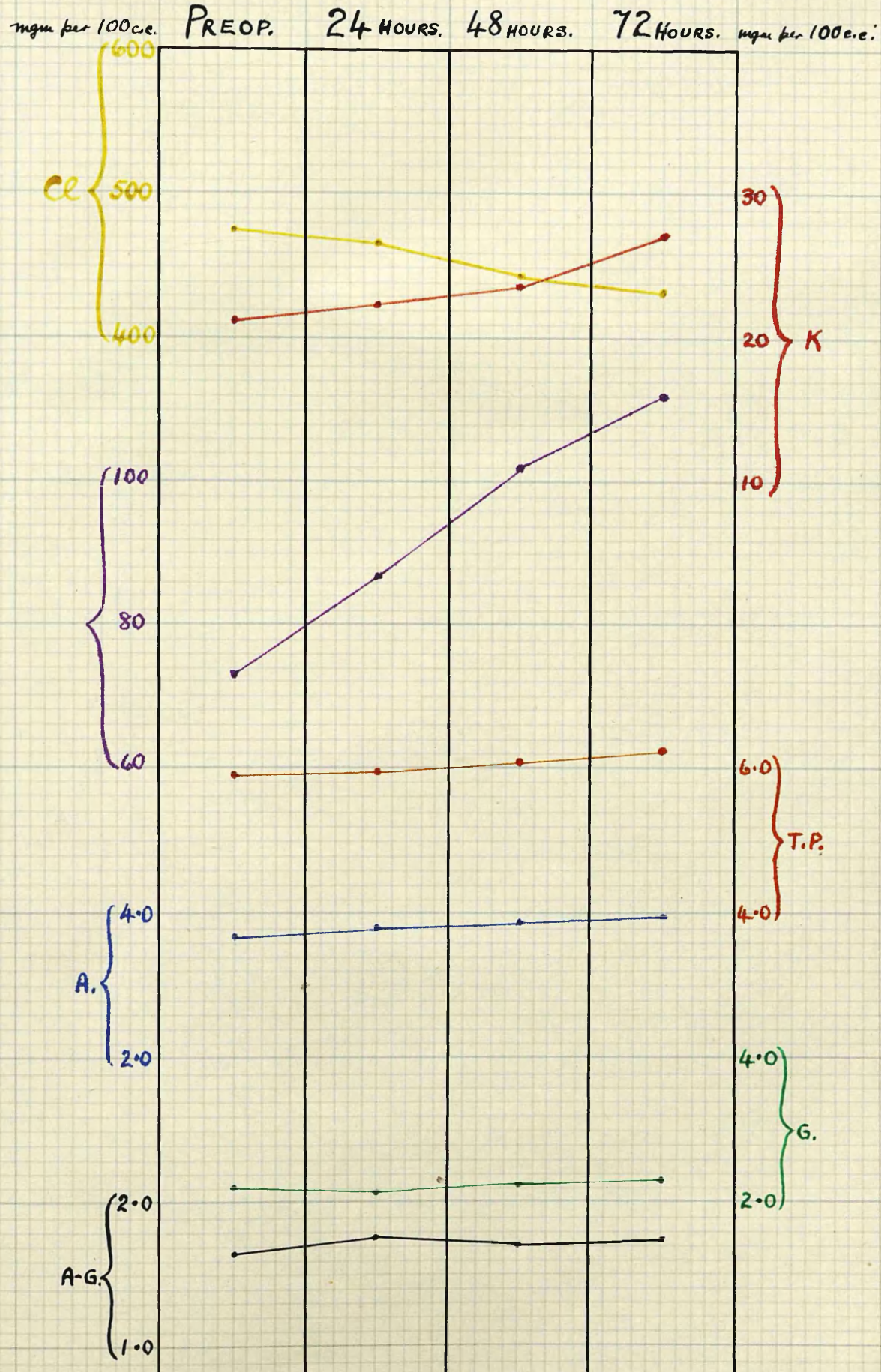
Constipated 3 days. Abdominal pains. Vomiting marked.

Carcinoma of ascending colon.

Operation - caecostomy. Convalescence uninterrupted.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	510	498	519	530
Potass.	14.5	14.9	16.0	18.4
N.P.N.	37.8	42.0	40.6	30.8
T.P.	5.89	5.95	5.91	5.84
Albumen.	3.71	3.80	3.74	3.67
Globulin.	2.18	2.15	2.17	2.17
A-G Ratio	1.70	1.77	1.72	1.69

CASE 36.


mgm per 100 c.c.
PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100 c.c.
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Cl
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400
30
20
K
10
100
80
60
6·0
T.P.
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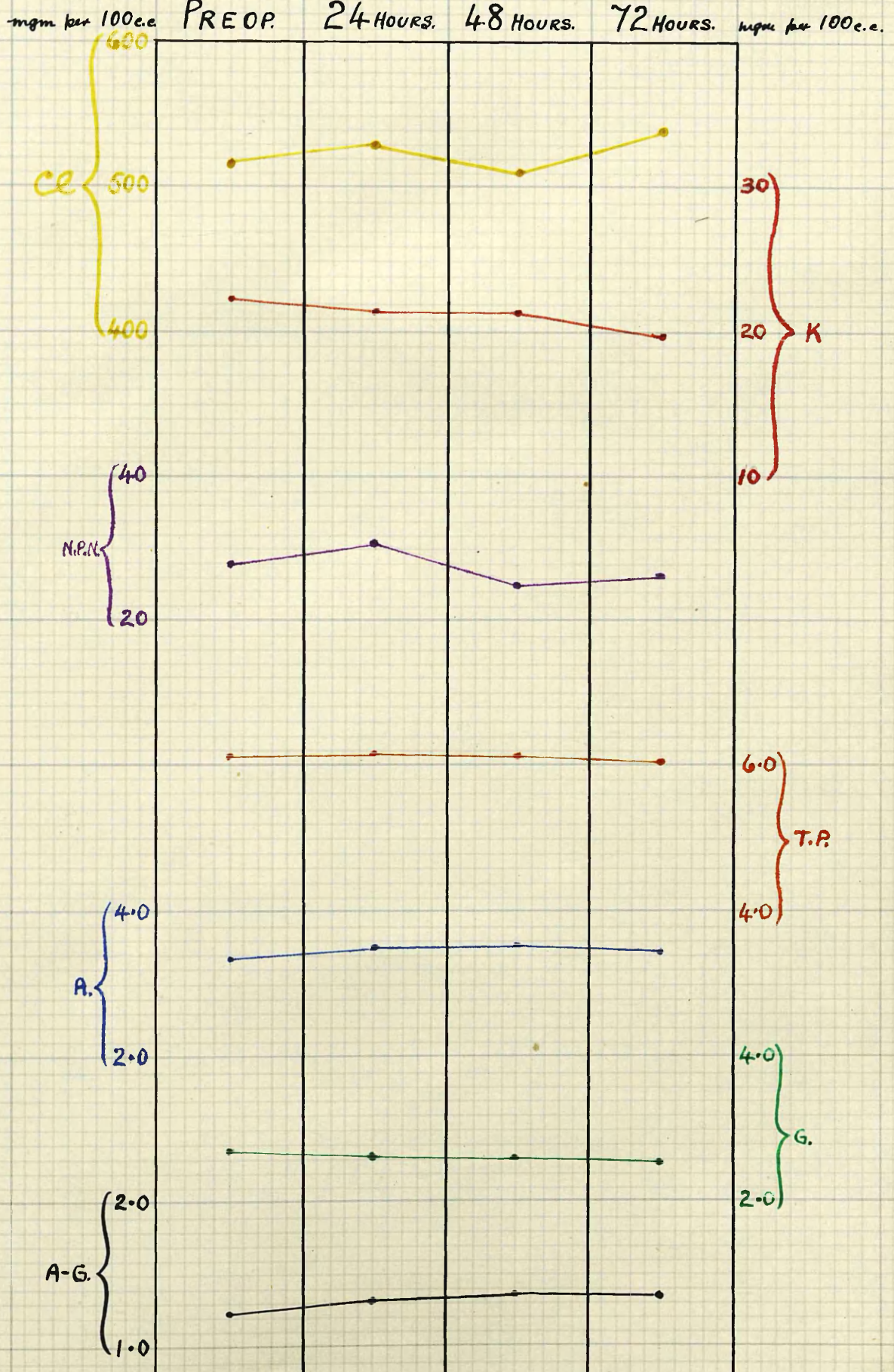
4·0
A.
2·0
4·0
G.
2·0
2·0
A-G.
1·0

CASE 37.


mgm per 100 c.c.
PREOP.
24 HOURS.
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mgm per 100 c.c.
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N.P.N.
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T.P.
4·0
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G.
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A-G.

Case 36.

Man, aged 55.

Constipated 4 days. Abdominal pains. Vomiting profuse.

Carcinoma of splenic flexure. Much ileal distension.

Operation - transverse colostomy.

Death 4 days after operation.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	477	467	446	435
Potass.	21.1	22.3	23.9	27.0
N.P.N.	72.8	86.8	102.2	112.0
T.P.	5.91	5.96	6.10	6.21
Albumen.	3.69	3.81	3.86	3.94
Globulin.	2.22	2.15	2.24	2.27
A-G Ratio	1.66	1.77	1.72	1.74

Case 37.

Man, aged 73.

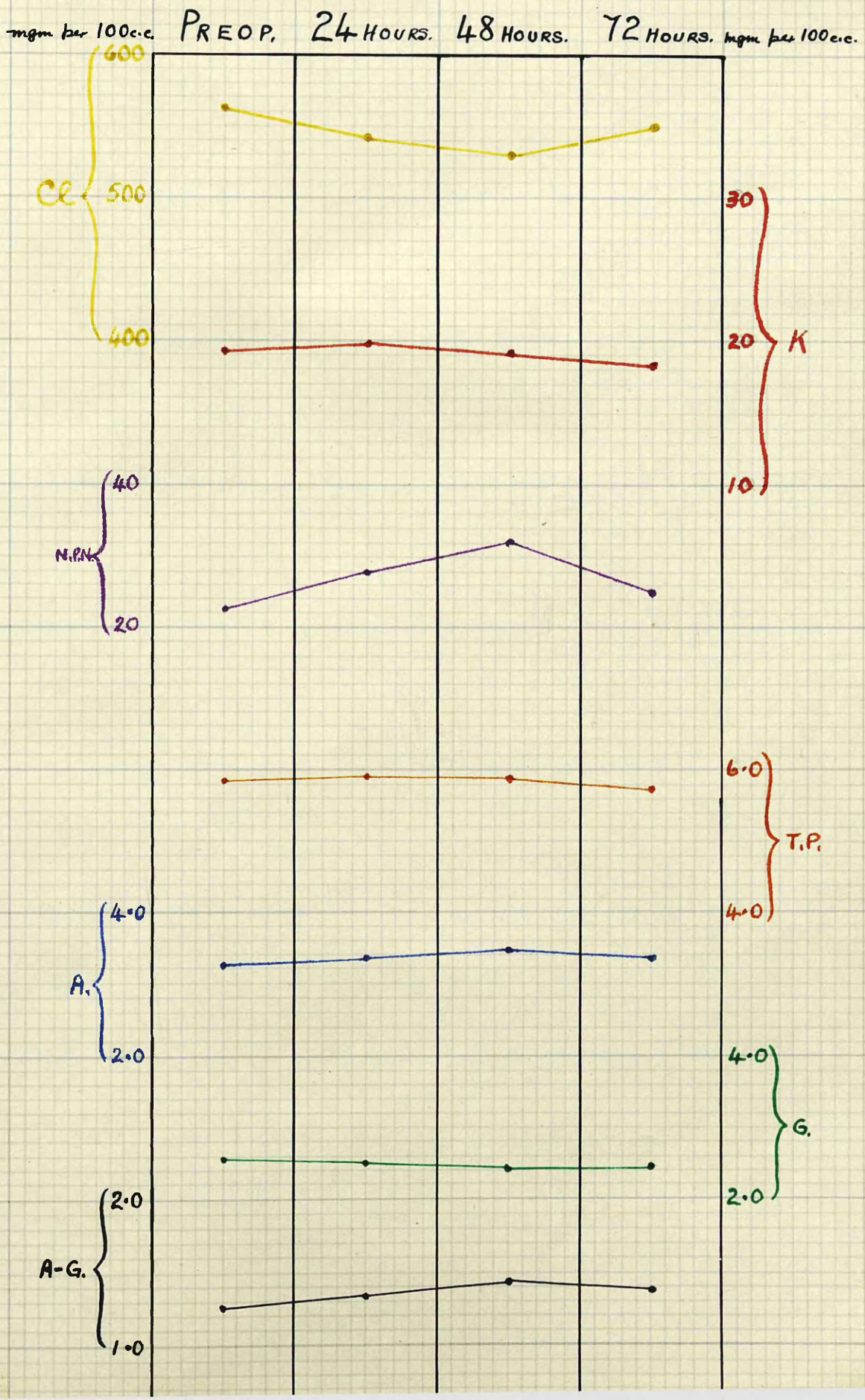
Constipated 4 days. Abdominal pains. Vomiting marked.

Carcinoma of hepatic flexure.

Operation - caecostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	519	530	510	541
Potass.	22.3	21.5	21.5	19.9
N.P.N.	28.0	30.8	25.2	26.6
T.P.	6.12	6.18	6.16	6.05
Albumen.	3.37	3.52	3.54	3.46
Globulin.	2.75	2.66	2.62	2.59
A-G Ratio	1.23	1.32	1.35	1.34

Case 38.


mgm per 100 c.c.
Preop.
24 Hours.
48 Hours.
72 Hours.
mgm per 100 c.c.
600
Cl
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10
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N.P.N.
20
6·0
T.P.
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A.
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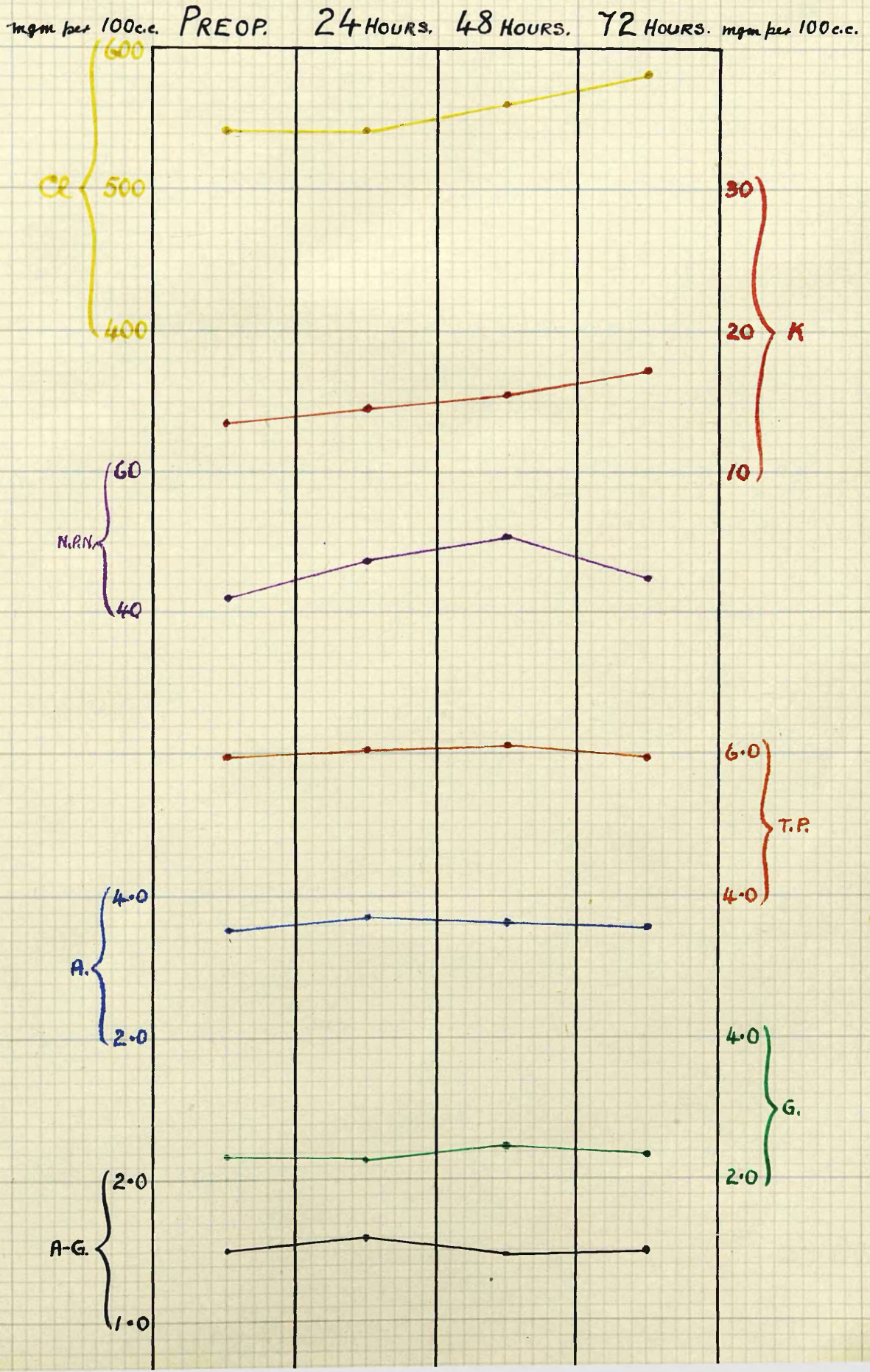
G.
2·0
2·0
A-G.
1·0

Case 39.


mgm per 100c.c.
PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100c.c.
600
Cl
500
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20
K
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N.P.N.
40
6·0
T.P.
4·0
4·0
A.
2·0
4·0
G.
2·0
2·0
A-G.
1·0

Case 38.

Man, aged 50.

Constipated 5 days. Abdominal pains. Vomited once.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	562	541	530	551
Potass.	19.2	19.9	19.2	18.4
N.P.N.	22.4	28.0	32.2	25.2
T.P.	5.82	5.91	5.87	5.78
Albumen.	3.24	3.34	3.46	3.35
Globulin.	2.58	2.53	2.41	2.43
A-G Ratio	1.26	1.34	1.43	1.38

Case 39.

Woman, aged 68.

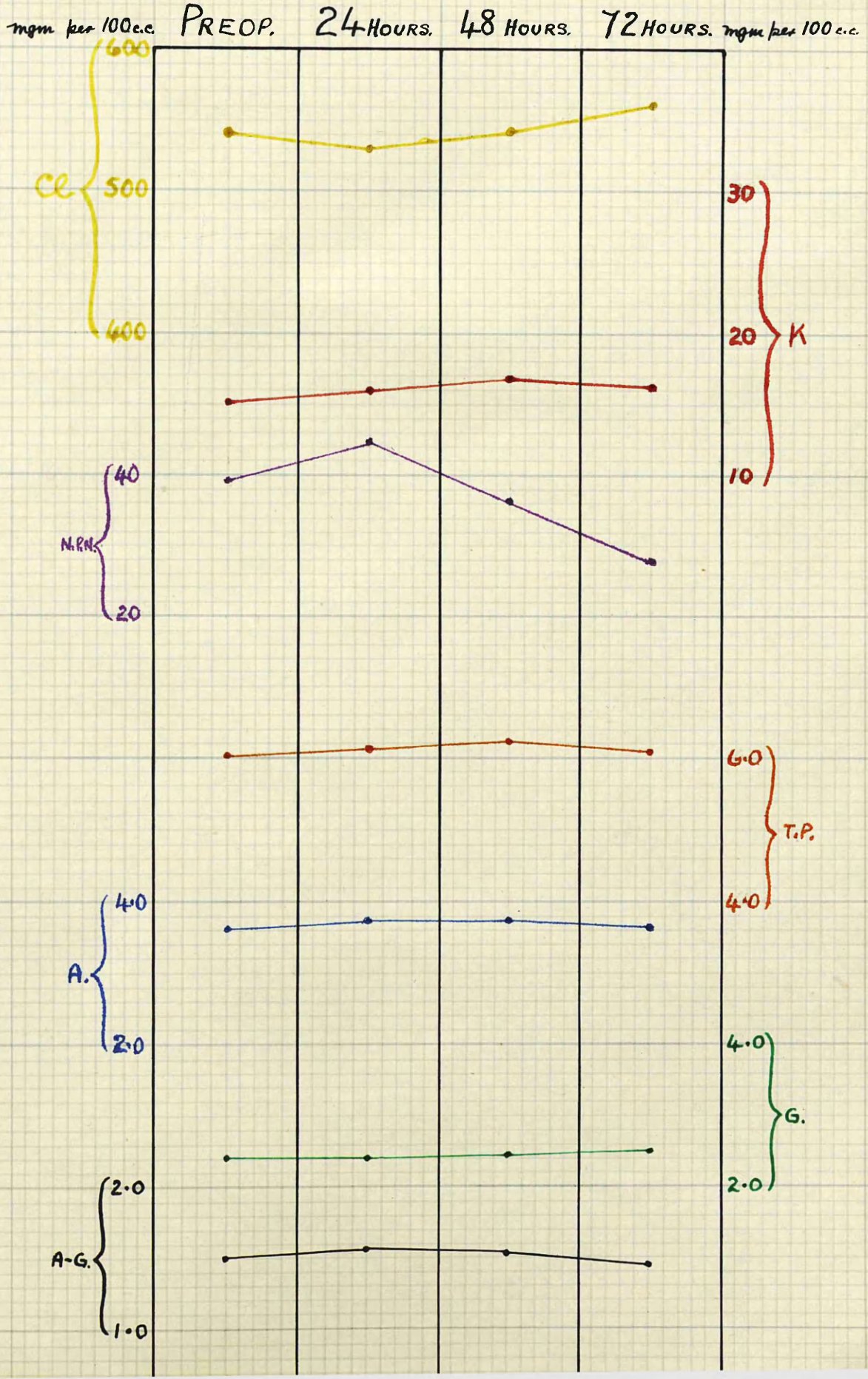
Constipated 4 days. Abdominal pains. Vomiting slight.

Carcinoma of sigmoid.

Operation - descending colostomy. Recovery uneventful.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	541	562	583
Potass.	13.7	14.5	15.6	17.2
N.P.N.	42.0	47.6	50.4	44.8
T.P.	5.95	6.03	6.12	5.98
Albumen.	3.57	3.71	3.65	3.59
Globulin.	2.38	2.32	2.47	2.39
A-G Ratio	1.50	1.60	1.48	1.50

CASE 40.


mgm per 100 c.c.
PREOP.
24 HOURS.
48 HOURS.
72 HOURS.
mgm per 100 c.c.
600
Cl
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20
K
10
40
N.P.N.
20
6·0
T.P.
4·0
4·0
A.
2·0
4·0
G.
2·0
2·0
A-G.
1·0

Case 40.

Man, aged 59.

Constipated 4 days. Abdominal pains. Vomiting slight.

Carcinoma of splenic flexure.

Operation - transverse colostomy.

Convalescence uninterrupted.

	Preop.	24 Hours	48 Hours	72 Hours
Chlor.	541	530	541	562
Potass.	15.2	16.0	16.8	16.4
N.P.N.	39.2	44.8	36.4	28.0
T.P.	6.02	6.14	6.21	6.13
Albumen.	3.61	3.74	3.76	3.63
Globulin.	2.41	2.40	2.45	2.50
A-G Ratio	1.50	1.56	1.53	1.45

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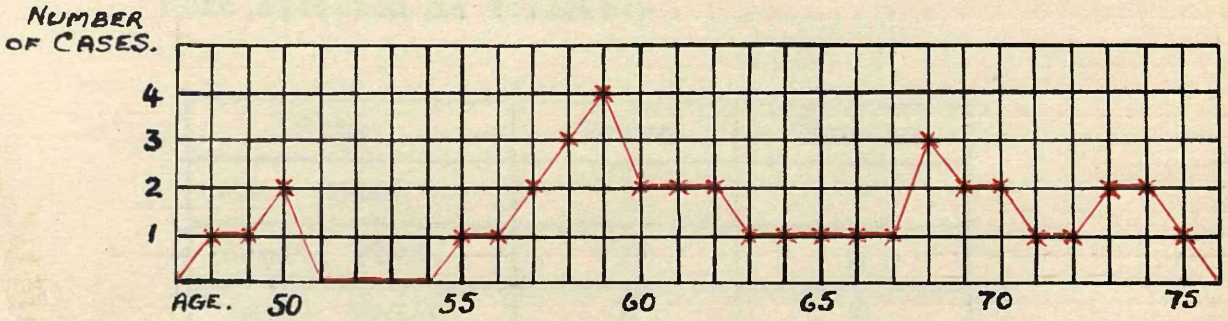
CHAPTER 6.

SUMMARY OF RESULTS.

1. Age. With the exception of one case whose age was 27, all the patients in this series were between the ages of 48 and 75.

Average. The average age was 62.35 years. The number of cases increased up to the age of 59, fell to a low level between the ages of 63 and 67, and a second, but lower, rise at the age of 68 fell gradually.



Males. The male cases included the man of 27 and the range covered the ages of 48 to 74. Their average age was 61.61 years.

Females. The female cases varied from 58 to 75, and their average age was 64.89 years.

Site of growth. The average age at the different sites of growth were as follows:-

Site	No. of Cases	Average Age
Asc. Colon	1	62.0
Hepat. Flex.	4	65.75
Trans. Colon	3	58.67
Splenic Flex.	8	65.25
Desc. Colon	5	63.0
Sigmoid	17	63.0
Rectum	2	42.5

2. Sex. There were 31 males and 9 females in the series. In regard to the site of the obstruction the sexes were affected as follows:-

Site	Males	Females
Asc. Colon	1	-
Hepat. Flex.	2	2
Trans. Colon	2	1
Splenic Flex.	7	1
Desc. Colon	5	-
Sigmoid	12	5
Rectum	2	-

3. Duration of obstruction.

No case had been ill less than 3 days and none more than 7 days.

The average time was 4.62 days.

The average duration of illness at the various sites was as follows:-

Site	Duration
Asc. Colon	3.0
Hepatic Flex.	4.75
Trans. Colon	5.0
Splen. Flex.	4.0
Desc. Colon	4.6
Sigmoid	5.0
Rectum	4.0

4. Sites of obstruction.

The commonest site of obstruction was the sigmoid colon - 17 cases, forming 42.5% of all the cases.

Site	No. of Cases	% of Total
Asc. Colon	1	2.5
Hepat. Flex.	4	10.0
Trans. Colon	3	7.5
Splen. Flex.	8	20.0
Desc. Colon	5	12.5
Sigmoid	17	42.5
Rectum	2	5.0

Obstruction of the left or distal half of the colon, from splenic flexure to rectum, accounted for no less than 32 cases, a percentage of 80, while obstruction of the proximal part alone accounted for only 8 cases, a percentage of 20.

5. Death Rate.

There were 5 deaths during the period of observation. This is 12½% of the total cases.

Death	Age	Sex	Duration	Site	Day after Op.
Case 1	73	M.	5	Splenic	4
Case 9	61	M.	4	Splenic	3
Case 19	75	F.	4	Splenic	2
Case 32	66	M.	6	Descend.	3
Case 36	55	M.	4	Splenic	4

The average age of the deaths is 66 years, which is only a little higher than the average age of the whole series.

There is no peculiarity in the proportion of the deaths in the sexes.

The average duration of the illness prior to operation is 4.6 days, which is almost the same average duration for the whole series. Four of the five deaths occurred in those suffering from carcinoma of the splenic flexure. This is no less than 50% of the cases occurring at this site.

The fact that the greatest number of deaths occurred on the 3rd and 4th days tends to suggest that they died from some biochemical change or from exhaustion rather than from anaesthetic causes or paralytic ileus.

6. Biochemical Results.

Chlorides.

Thirty cases showed a low preoperative level. 6 of the remaining 10 showed a low normal level. The average result was 526 mgm. per 100 c.c. serum. 24 hours after operation, 27 cases showed a further fall in the serum chlorides, in 5 others it was the same as before operation, and in 8 it had commenced to return to normal.

The average result was 523 mgm.

On the 2nd day after operation, 1 patient died, 2 patients did not show a rise in the level (Cases 20 and 28), but in one of them (Case 28) the level was already normal, and in 5 others (Cases 8, 10, 30, 36 and 38) there was a still further fall. Therefore, 32 patients had started to make good their serum chlorides.

The average result on this day was 531 mgm.

On the 3rd day after operation, other 2 patients died and, of the remaining 37, only 1 (Case 36) failed to maintain a normal figure or to improve his chloride balance. This patient's serum chloride continued to fall and he died on the following day.

The average result for the 3rd day after operation was 552 mgm.

	Highest	Lowest	Average
Preop.	598	456	526
24 Hours	593	435	523
48 Hours	604	446	531
72 Hours	604	435	552

Of the 5 deaths in the series, all had lowered chloride levels before death, but only in 3 (Cases 19, 32 and 36) was it abnormally low, being 477 mgm. in Case 32 and 435 in Cases 19 and 36.

Potassium.

24 cases showed a lowered potassium level before operation, 6 were within normal limits and 10 were above the normal. Normal was considered to be 18 - 21 mgm. of potassium per 100 c.c. of serum (83).

On the day following operation, 19 of the low cases had commenced to rise, while the other 5 went lower still. 3 of the 6 normal levels became sub-normal, while 5 of those showing raised levels went higher, but the other 5 became lower.

On the 2nd day after operation, 6 of the 10 raised levels had commenced to fall, but the other 3 were still rising. The 10th case had died. 17 of the lowered cases were still rising. Thus the

bulk of the cases were returning to normal.

72 hours after operation the position was as follows. There was another death among the cases with a raised preoperative level and of the remaining 8, 7 had definitely fallen towards normal, but the 8th, whose potassium was still rising, died on the following day. One of the normal cases (Case 1) showed rather a low serum potassium and this patient died next day. 2 of the subnormal cases still showed a lowered result which was continuing to fall. These cases, however, eventually recovered.

The question arose as to why 24 cases should have a lowered serum potassium and 10 should be raised. Some of the main facts about the latter group are in the following table:-

Case	Age	Durat.	Site	S.I. Distens	Potassium				Death
2	74	4	Sig.	-	27.1	25.0	27.4	23.1	--
3	58	6	Sig.	+ +	27.4	28.5	24.6	22.3	--
14	62	5	Desc.	-	22.9	21.1	20.3	17.6	--
16	68	6	Sig.	+ +	24.6	26.2	22.3	18.4	--
19	75	4	Splen.	+ +	26.2	29.3	--	--	2nd Day
25	57	6	Sig.	+ +	29.3	27.8	25.4	21.5	--
32	66	6	Desc.	+ +	28.5	27.8	28.5	--	3rd Day
34	70	4	Desc.	+ +	25.4	27.0	24.6	21.1	--
36	55	4	Splen.	+ +	21.1	22.3	23.9	27.0	4th Day
37	73	4	Hepat.	-	22.3	21.5	21.5	19.9	--

Age. The average age of these patients is 65.8 years, which is little different from the average age of the whole series.

Duration of illness. The average duration of these cases, 4.9 days, is also very little removed from that of the whole series.

Sites. All sites appear to be proportionately involved.

Amount of small intestinal distension. This factor is more striking in its relationship to the high level of potassium. Only 3 cases out of the 10 did not show marked small intestinal distension and in 2 of them (Cases 14 and 37) the serum potassium was not very much over normal.

Deaths. Deaths in this group occurred only in those with marked small bowel distension and occurred on different days of the illness.

N.P.N.

The bulk of the cases had raised serum N.P.N. before operation. In 13 cases it was within normal limits.

On the day after operation, the level rose in 32 cases but dropped in 8 towards a normal level.

On the 2nd day after operation, there was a further rise in 13 cases, and in 1 it remained the same, but all the others showed a fall. Of the 13, 2 died on the same day and 10 of the remaining 11 showed a distinct return to normal on the 3rd day. The 11th died on the 4th day.

Thus all the cases were showing a distinct improvement in their N.P.N. figures by the 3rd day after operation except those who were moribund.

The average N.P.N. results for the 4 days of observation were as follows:-

Preop.	1st Day	2nd Day	3rd Day
47.8	55.3	51.1	39.9

Total Proteins.

The remarkable thing about these results was that none was particularly high. The normal was considered to be from 5.6 - 8.5 grms. with an average of 7.0 grms. (83). The highest was in Case 19, where the total concentration was 6.87 grms. This patient was the one who died on the 2nd day after operation.

The lowest figure obtained was in Case 10, the amount of the serum proteins being 5.23 grms.

On the 1st day after operation, nearly all the cases showed an increasing concentration of proteins.

On the 2nd day after operation, only 13 were still showing a rise in their serum protein figures. This included all those with marked small bowel distension except Cases 3 and 25, whose figures had become lower.

Only 2 patients (Cases 16 and 36) were still rising above their preoperative levels on the 3rd postoperative day. Both of these cases had marked

small bowel distension, and Case 36 was one of the deaths. All the other cases showed a slight drop.

The average results of these cases are as follows:-

Preop.	1st Day	2nd Day	3rd Day
6.06	6.00	6.02	5.98

Albumen.

None of the serum albumen results was abnormally high, if the normal limits be taken as 3.8 - 5.2 grms. with an average of 4.0 grms. (83). The highest concentration was 3.94 grms. in Case 34. This was one of the patients with marked small bowel obstruction. The lowest was 2.64% in Case 17.

Before operation there were only 9 cases whose albumen concentration was not below the low normal of 3.8%. All the other 31 had subnormal results.

On the day after operation 30 patients showed an increase in the amount of albumen present, but in 10 there was a slight fall. With the exception of those who were moribund and in whom there was an increasing concentration until death, there was a fall to a lower level in every case by the 3rd day after operation.

It has been impossible to account for a fall in the serum albumen on the day after operation in

the 10 cases previously mentioned. They include some cases with marked small intestinal distension, but the proportion is not near sufficient to draw an exact conclusion.

The average results for the whole series was as follows:-

Preop.	1st Day	2nd Day	3rd Day
3.52	3.58	3.57	3.50

Globulins.

In contrast to the albumens, no case had a globulin concentration below the accepted normal of 1.6 - 3.4 grms. with an average of 2.0 grms. (83) before operation, and in no case was it above normal. The highest figure obtained was 3.39 grms. in Case 8 and the lowest 1.86 grms. in Case 22. Neither of these cases was abnormal in any respect.

On the first day after operation there was a drop in the globulin percentage in 26 cases. One other remained at the same level and the rest rose slightly.

On the 2nd day after operation, 26 cases showed a rise in concentration of globulin, the remainder showing a fall.

On the 3rd day, 17 cases were still showing a rise, while 18 were falling and 2 remained at the same level. It must be pointed out in connection with these calculations that many of the falls and

rises were of the order of 0.01% or 0.02% and that the globulin results were obtained from the total protein results by subtracting the albumen figures and that any small alteration in the latter would cause a reflection in the globulin figures.

The average result of the whole series of cases was as follows:-

Preop.	1st Day	2nd Day	3rd Day
2.48	2.45	2.45	2.47

Albumen-Globulin Ratio.

9 cases were abnormally low, but no case was too high before operation. 2 of the former had small bowel distension and one of them (Case 19) died on the 2nd day after operation. The others did not appear to present any abnormal feature.

28 cases showed an increase in the ratio on the day after operation.

On the 2nd day after operation, 26 cases showed a fall in the ratio and on the 3rd post-operative day 21 of the 37 patients who were still alive showed a further drop.

The average figures for the series were as follows:-

Preop.	1st Day	2nd Day	3rd Day
1.43	1.52	1.53	1.48

7. Small Bowel Distension.

The relationship of gross small bowel distension to marked changes in the blood chemistry have been pointed out previously. It is interesting to tabulate these cases and to compare the results obtained.

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Case	Durat.	Cl.	K.	N.P.N.	T.P.	A.	G.	A/G.	Death
3	6	496	27.37	174	5.85	3.93	1.92	2.05	--
11	4	477	14.8	50	6.42	3.81	2.61	1.46	--
16	6	498	24.6	102	5.40	2.94	2.46	1.19	--
19	4	456	26.2	81	6.87	3.68	3.19	1.15	2
21	6	498	20.3	71	5.76	3.38	2.38	1.42	--
25	6	477	29.3	94	6.58	3.91	2.67	1.46	--
32	6	488	28.5	120	6.52	3.92	2.60	1.51	3
34	4	456	25.4	97	6.31	3.94	2.37	1.66	--
36	4	477	21.1	73	5.91	3.69	2.22	1.66	4

It is even more interesting to compare the average results of the above 9 cases with the average results for the whole series and with the remaining 31 cases who did not show marked abdominal distension.

	Cl.	K.	N.P.N.	T.P.	A.	G.	A/G.
Whole Series	526	18.4	48	6.06	3.52	2.48	1.43
S.I. Distended	480	24.2	96	6.18	3.69	2.48	1.50
S.I. Not Distended	539	16.7	34	6.02	3.48	2.48	1.40

Thus the following facts are brought out:-

(1) Chlorides are very low in every case and the average for the 9 cases is well below the average of the whole series.

(2) Potassium results are high in these cases compared with the average figure for the whole series. This is more noticeable when compared with the average results obtained in cases which had no marked abdominal distension, and which had subnormal potassium concentrations. Taken individually, 3 cases did show normal or subnormal results.

(3) N.P.N. Every case showed a result above the average for the whole series. The average result is striking compared with that for the whole series and for those cases not showing gross small bowel distension.

(4) Total proteins. There was an increased concentration of proteins compared with those

not distended, although 4 cases were below all the average figures.

(5) Albumen. The albumen concentration was also raised in this series. Only 2 cases were below the average for the whole series.

(6) Globulin. There was no alteration in the amount of globulins present in any of the averages. 5 cases were below the average and 4 were above it.

(7) Albumen-Globulin ratio. There was a slight increase in the ratio not only compared with those who were not distended but also with the whole series. Only 3 cases had ratios below the average of the whole series.

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CHAPTER 7.

CONCLUSION.

The bloods obtained from a series of 40 consecutive cases of obstruction of the colon admitted to the Royal and Victoria Infirmaries, Glasgow, were examined prior to operation and for 3 days after operation, provided that the patients were still alive.

The estimations made were:-

(1) Serum chlorides.

(2) Serum potassium.

(3) Serum non-protein nitrogen.

(4) Total proteins of the serum.

(5) Serum albumen.

(6) Serum globulin.

(7) Albumen - Globulin ratio.

The results show that:-

(1) The serum chlorides are low in the majority of cases and tend to become lower after operation before regaining normality.

(2) Some cases have a raised serum potassium, but the majority have a lowered potassium value. The former are associated with small bowel distension, profuse vomiting and other marked changes in the blood chemistry. They are the patients who are liable to die.

(3) The non-protein nitrogen is raised in nearly every case and tends to become higher still after operation before returning to normal. All cases showed a return towards normal by the 3rd day after operation except those who were moribund. 4 of the 5 deaths showed a rising N.P.N.

(4) The total serum proteins were within normal limits even in those cases with profuse vomiting. The majority of the results were on the low side.

(5) The serum albumen results were all low and the average figure for the whole series was distinctly low and sub-normal.

(6) The serum globulin concentrations were a high normal.

(7) The albumen-globulin ratio was distinctly low.

(8) The lowered chloride value and the raised non-protein nitrogen cannot be due to vomiting and dehydration alone because their figures do not correspond.

(9) Potassium cannot be the lethal factor alone because it was not raised in 30 patients - 75% of the total. It was raised in 3 patients who died, but there were other factors of a more severe degree present in these cases.

(10) The lowered total protein content seems to be dependent on the lowered albumen concentration.

(11) Either the albumens were lowered or the globulins were increased, judging by the albumen-globulin ratio. As it has been shown that the total proteins and the albumen figures are lowered, it would seem that there is an actual loss of albumen in the blood. This compares favourably with Cuthbertson's and Tompsett's (35) findings in traumatic shock.

(12) It is suggested from the total protein figures that, even allowing for the loss of albumen, there is no marked degree of dehydration present in these patients.

(13) These findings tend to support the observations of Van Beuren (154) and Warren (161) that no specific poison of intestinal obstruction exists.

(14) There is no true biochemical guide to the assessment of the severity of the illness but, from the results and from the above conclusions, it is recommended that, in clinical practice, attention should be paid to

(a) The high N.P.N., and its daily estimation seems to be the best guide as to the progress of the illness;

(b) The lowered chloride level;

(c) Cases with much small intestinal distension. Not only does it make operation more difficult, but it is a sign of severe detrimental biochemical changes. The importance of distension has been stressed by Gendel and Fine (59) also, and they say that it leads to a more rapid death.

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CHAPTER 8.

BIBLIOGRAPHY.

1. Abbott, W. O. Johnston, G. G. — Surg. Gyn. Obs. 1938, lxvi, 4.
2. Aird, I. — Proc. Soc. Exper. Biol. & Med. 1935, xxxiii, 199.
3. Aird, I. — Edinburgh Med. Jour. 1936, xliii, 375.
4. Aird, I. — Edinburgh Med. Jour. 1937, xliv, 28.
5. Aird, I. — Brit. J. Surg. 1938, xxvi, 418.
6. Aird, I. Henderson, W. K. — Lancet. 1936, vii, 373.
7. Amberg, S. Helmholtz, H. F. — J. Pharmac. & Exper. Therap. 1918, xii, 19.
8. Andrus, W. D. Guest, G. M. — J. Clin. Invest. 1932, xi, 475.
9. Austin, R. C. — Ohio State Med. Jour. 1936, xxxii, 514.
10. Bacon, D. K. Anslow, R. E. — Arch. Surg. 1921, iii, 641.
11. Bailey, H. Carnow, J. — Brit. Med. J. 1934, i, 11.
12. Barnes, C. G. Lowe, T. E. — Post-Grad. Med. J. 1936, xii, 233.
13. Barrett, N. R. Battle, R. — St. Thomas's Hosp. Rep. 1936, i, 78.
14. Bartlett, W. — Arch. Surg. 1934, xxiii, 484.
15. Bingham, D. L. C. — Brit. Med. J. 1938, i, 67.

16. Bisgard, J. D. — Surgery. 1938, iv, 528.

17. Bonoff, K. M. — Calif. & West. Med. 1939, li, 154.

18. Brown, G. E. Eusterman, G. B. — Arch. Int. Med. 1923, xxxii, 425.

19. Butler, A. M. McKhann, C. F. — J. Pediat. 1933, iii, 84.

20. Carlson, A. J. — Amer. J. Physiol. 1906, xvi, 378.

21. Chapman, J. — Calif. & West. Med. 1935, xliii, 419.

22. Church, R. E. — Amer. J. Surg. 1935, xxix, 64.

23. Cohen, H. Miller, P. R. — J. Pediat. 1933, iii, 299.

24. Coller, F. A. Maddock, W. — J. Amer. Med. Assoc. 1932, xcix, 875.

25. Coller, F. A. Maddock, W. — International Clinics. 1934, iii, 190.

26. Coller, F. A. Dick, V. S. — J. Amer. Med. Assoc. 1936, cvii, 1522.

27. Cooke, A. M. — Quart. J. Med. 1932, nsi, 527.

28. Cooke, J. V. Rodenbaugh, F. H. — J. Exper. Med. 1916, xxiii, 717.

29. Cooke, J. V. Whipple, G. H. — J. Exper. Med. 1918, xxviii, 223.

30. Cooper, H. S. F. — Arch. Surg. 1928, xvii, 918

31. Cope, V. Z. — Brit. Med. J. 1932, ii, 544.

32. Cullen, G. E. — Ohio State Med. J. 1936, xxxii, 509

33. Curry, F. S. Bargen, J. A. — Surg. Gyn. Obs. 1935, lx, 667.

34. Cuthbertson, D. P. — Quart. J. Med. 1932, nsi, 233.

35. Cuthbertson, D. P. Tompsett, S. L. — Brit. J. Exper. Path. 1935, xvi, 471.

36. Darrow, D. C. Yannet, H. — J. Clin. Invest. 1935, xiv, 266.

37. Davis, H. A. Dragstedt, L. R. — Amer. J. Physiol. 1935, cxiii, 193.

38. De Beer, E. J. Johnston, C. G. — J. Biol. Chem. 1935, cxiii, 113.

39. Derow, H. A. — New England J. Med. 1935, ccxii, 509.

40. Dieckmann, W. J. Crossen, R. J. — Amer. J. Obst. & Gyn. 1927, xiv, 3.

41. Dragstedt, L. R. — Proc. Soc. Exper. Biol. & Med. 1928, xxv, 239.

42. Dragstedt, L. R. Ellis, J. C. — Amer. J. Physiol. 1930, xciii, 407.

43. Dragstedt, L. R. Moorhead, J. J. — J. Exper. Med. 1917, xxv, 421.

44. Eichelberger, L. Hastings, A. B. — J. Biol. Chem. 1937, cxviii, 205.

45. Ellis, L. B. Weiss, S. — J. Amer. Med. Assoc. 1933,c, 875.

46. Elman, R. Hartmann, A. F. — Arch. Surg. 1930, xx, 333.

47. Falconer, M. A. Lyall, A. — Brit. Med. J. 1937, ii, 1116.

48. Falconer, M. A. Lyall, A. — Lancet. 1937, ii, 1472.

49. Falconer, M. A. Osterberg, A. E. — Arch. Surg. 1939, xxxviii, 869.

50. Felty, A. R. Murray, H. A. — J. Biol. Chem. 1923, lvii, 573.

51. Fey, A. Cubbins, W. R. — Surg. Gyn. Obs. 1935, lx, 738.

52. Foster, W. C.
Hausler, R. W.
Arch. Int. Med.
1925, xxxvi, 31.

53. Gamble, J. L.
McIver, M. A.
J. Exper. Med.
1928, xlviii, 837.

54. Gamble, J. L.
McIver, M. A.
J. Exper. Med.
1928, Xlviii, 849.

55. Gamble, J. L.
Ross, G. S.
J. Biol. Chem.
1923, lvii, 633.

56. Gamble, J. L.
Ross, G. S.
J. Clin. Invest.
1924, i, 403.

57. Gatch, W. D.
Surgery.
1937, vi, 896.

58. Gatch, W. D.
Trusler, H. M.
Amer. J. Med. Sci.
1927, clxxiii, 640.

59. Gendel, S.
Fine, J.
Ann. Surg.
1939, cx, 25.

60. Gibson, J. G.
Branch, C. D.
Surg. Gyn. Obs.
1937, lxv, 741.

61. Gilman, A.
Amer. J. Physiol.
1934, cviii, 662.

62. Greene, E. I.
Greene, J.
Illinois Med. J.
1935, lxvii, 51.

63. Guest, G. M.
Andrus, W. D.
J. Clin. Invest.
1932, xi, 455.

64. Haden, R. L.
Guffey, D. C.
Amer. J. Obs. Gyn.
1924, viii, 486.

65. Haden, R. L.
Orr, T. G.
Surg. Gyn. Obs.
1923, xxxvii, 465.

66. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1923, xxxvii, 365.

67. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1923, xxxviii, 55.

68. Haden, R. L.
Orr, T. G.
Bull. John Hopk. Hosp.
1923, xxxiv, 26.

69. Haden, R. L.
Orr, T. G.
J. Amer. Med. Assoc.
1924, lxxxii, 1515.

70. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1924, xxxix, 321.

71. Haden, R. L.
Orr, T. G.
Arch. Surg.
1925, xi, 859.

72. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1925, xli, 113.

73. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1925, xli, 119.

74. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1925, xli, 707.

75. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1926, xliv, 419.

76. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1927, xlv, 427.

77. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1927, xlv, 433.

78. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1927, xlvi, 419.

79. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1927, xlvi, 709.

80. Haden, R. L.
Orr, T. G.
J. Exper. Med.
1928, xlviii, 591.

81. Harding, V. J.
Drew, K.
J. Obst. & Gyn. Brit. Emp.
1923, xxx, 507.

82. Harding, V. J.
van Wyck, H. B.
Amer. J. Obst. & Gyn.
1926, xi, 1.

83. Harrison, G. A.
"Chemical Methods in Clinical Medicine".
2nd Edit. London, 1937.

84. Harrison, H. E.
Darrow, D. C.
J. Biol. Chem.
1936, cxiii, 515.

85. Hartmann, A. F.
Amer. J. Dis. Child.
1928, xxxv, 557.

86. Hartmann, A. F.
Elman, R.
J. Exper. Med.
1929, l, 387.

87. Hartmann, A. F.
Smythe, F. S.
Amer. J. Dis. Child.
1926, xxxii, 1.

88. Hartwell, J. A.
Hoguet, J. P.
J. Amer. Med. Assoc.
1912, lix, 82.

89. Hastings, A. B.
Eichelberger, L.
J. Biol. Chem.
1935, cix, 41.

90. Hastings, A. B.
Eichelberger, L.
J. Biol. Chem.
1937, cxvii, 73.

91. Hastings, A. B.
Murray, C. D.
J. Biol. Chem.
1921, xlvi, 223.

92. Heggs, F. M.
Lancet.
1937, ii, 1250.

93. Herrin, R. C.
J. Biol. Chem.
1935, cviii, 547.

94. Howell, W. H.
Amer. J. Physiol.
1901, vi, 181.

95. Ingvaldsen, T.
Whipple, A. O.
J. Exper. Med.
1924, xxxix. 117.

96. Keeley, J. L.
Gibson, J. G.
Surgery.
1939, v, 872.

97. Kerr, S. E.
J. Biol. Chem.
1926, lxvii, 689.

98. Kerr, A. B.
Lendrum, A. C.
Brit. J. Surg.
1935, xxiii, 615.

99. King, J. H.
Amer. J. Med. Sci.
1878, lxxv, 404.

100. Knight, G. C.
Slome, D.
Brit. J. Surg.
1936, xxiii, 820.

101. Langdon-Brown, W.
Lancet.
1935, i, 1069.

102. Larson, P. S.
Brewer, G.
J. Pharmac. & Therap.
1939, lxvii, 147.

103. Lim, R. K. S.
Ni, T. G.
Amer. J. Physiol.
1925, lxxv, 475.

104. MacCallum, W. G.
Lintz, J.
Bull. John Hopk. Hosp.
1920, xxxi, 1.

105. McCance, R. A. — Lancet. 1936, i, 643. " " i, 704. " " i, 765. " " i, 823.

106. McCance, R.A. Lawrence, R. D. — Quart. J. Med. 1935, nsiv, 53.

107. McClure, R. D. — J. Amer. Med. Assoc. 1907, xlix, 1003.

108. McEwen, O. R. — Lancet. 1935, i, 1015.

109. McGee, W. G. — Interstate Med. J. 1906, xiii, 279.

110. McIver, M. A. Gamble, J. L. — J. Amer. Med. Assoc. 1928, xci, 1589.

111. McKay, L. L. McKay, E. M. — Amer. J. Physiol. 1924, lxx, 394.

112. McVicar, C. S. — Amer. J. Med. Sci. 1925, clxix, 224.

113. McVicar, C. S. — J. Amer. Med. Assoc. 1929, xcii, 887.

114. Maddock, W. G. — Amer. J. Surg. 1939, xlvi, 426.

115. Marriott, W. M. — Physiol. Rev. 1923, iii, 275.

116. Mathison, G. C. — J. Physiol. 1911,xliii, 471.

117. Maury, J. W. D. — J. Amer. Med. Assoc. 1910, liv, 5.

118. Maycock, W. d'A. — Brit. J. Surg. 1938, xxv, 677.

119. Melnick, D. Cowgill, G. R. — Proc. Soc. Exper. Biol. & Med. 1936,xxxv, 312.

120. Nilson, H. W. — Amer. J. Physiol. 1937, cxviii, 620.

121. Oakley, W. — Lancet. 1935, ii, 187.

122. Orr, T. G. — Surgery. 1937, i, 838.

123. Orr, T. G. — Amer. J. Surg. 1939, xlvi, 70

124. Orr, T. G. Haden, R. L. — J. Amer. Med. Assoc. 1928, xci, 1529.

125. Owings, J. C. McIntosh, C. A. — Arch. Surg. 1928, xvii, 507.

126. Paine, J. R. Carlson, H. — J. Amer. Med. Assoc. 1933, c, 1910.

127. Parkins, W. M. Taylor, A. M. — Amer. J. Physiol. 1935, cxii, 581.

128. Patey, D. H. — Clinical Jour. 1935, i, 64.

129. Patey, D. H. Ashcroft, P. — Brit. Med. J. 1935, ii, 1197.

130. Peters, J. P. — Bull. New York Acad. Med. 1934, x, 415.

131. Pratt, G. — Amer. J. Surg. 1934, xxiii, 148.

132. Rachmilewitz, M. — Lancet, 1934, i, 78.

133. Rankin, F. W. Johnston, C. C. — Amer. J. Surg. 1939, xlvi, 411.

134. Robertson, J. D. — J. Physiol. 1935, lxxxiv, 393.

135. Schiff, L. Stevens, R. J. — Arch. Int. Med. 1939, lxiv, 1239.

136. Schloss, O. M. — Amer. J. Dis. Child. 1918, xv, 165.

137. Schloss, O. M. Stetson, R. E. — Amer. J. Dis. Child. 1917, xiii, 218.

138. Scudder, J. — Surgery. 1937, i, 74.

139. Scudder, J. Zwemer, R. L. — Annals of Surgery. 1938, cvii, 161.

140. Shier, L. — Surg. Gyn. Obs. 1936, lxiii, 523.

141. Shohl, A. T. Bennett, H. B. — J. Biol. Chem. 1928, lxxviii, 643.

142. Spencer, H. R. Filent, E. — Brit. Med. J. 1934, i, 75.

143. Stanbury, J. B. Warweg, E. — Amer. J. Physiol. 1936, cxvii, 230.

144. Steggerda, F. R. — Amer. J. Physiol. 1935, cxii, 559.

145. Stewart, J. D. Rourke, G. M. — J. Clin. Invest. 1936, xv, 692.

146. Sullivan, R. C. McLean, A. B. — Amer. J. Dis. Child. 1932, xliii, 1279.

147. Talbott, J. H. Michelson, J. — J. Clin. Invest. 1933, xii, 533.

148. Teitelbaum, M. — J. Lab. & Clinic. Med. 1938, xxiii, 689.

149. Thaler, J. I. — Proc. Soc. Exper. Biol. & Med. 1935, xxxiii, 368.

150. Thorn, G. W. Garbutt, H. R. — Proc. Soc. Exper. Biol. & Med. 1936, xxxv, 247.

151. Tileston, W. Confort, C. W. — Arch. Int. Med. 1914, xiv, 620.

152. Truszkowski, R. Zwemer, R. L. — Endocrinology. 1937, xxi, 40.

153. Underhill, J. Carrington, R. — Arch. Int. Med. 1923, xxxii, 31.

154. Van Beuren, F. T. — Annals of Surgery. 1935, cii, 605.

155. Van Slyke, D. D. — J. Biol. Chem. 1923, lviii, 53.

156. Vick, R. M. — Brit. Med. J. 1932, ii, 546.

157. Walters, W. Bollman, J. L. — J. Amer. Med. Assoc. 1926, lxxxvi, 186.

158. Walters, W.
Bollman, J. L. Arch. Surg.
1926, xiii, 578.

159. Wangansteen, O. H. Arch. Surg.
1933, xxvi, 933.

160. Wangansteen, O. H.
Chunn, S.S. Arch, Surg.
1928, xvi, 1242.

161. Warren, R. Brit. Med. J.
1936, i, 245.

162. Weiner, D. O.
Elman, R. Proc. Soc. Exper. Biol. & Med.
1936, xxxiv, 484.

163. Wheeler, W. de C. Brit. Med. J.
1932, ii, 545.

164. Whipple, G. H.
Cooke, J. V. J. Exper. Med.
1917, xxv, 461.

165. Whipple, G. H.
Cooke, J. V. J. Exper. Med.
1917, xxv, 479.

166. Whipple, G. H.
Cooke, J. V. J. Exper. Med.
1916, xxiii, 717.

167. Whipple, G. H.
van Slyke, D. D. J. Exper. Med.
1918, xxviii, 213.

168. White, J. C.
Bridge, E. M. Boston Med. & Surg. J.
1927, cxcvi, 893.

169. Wohl, M. G.
Brust, R. W. J. Lab. & Clin. Med.
1935, xx, 1170.

170. Wilkie, D. P. D. Brit. Med. J.
1932, ii, 545.

171. Wright, S. "Applied Physiology".
7th Edit. London. 1940.

172. Yannet, H.
Darrow, D. C. J. Biol. Chem.
1935, cxii, 477.

173. Zwemer, R. L.
Scudder, J. Surgery.
1938, iv, 510.

174. Zwemer, R. L.
Sullivan, R. C. Endocrinology.
1934, xviii, 97.

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