SYNDROMES AFTER PARTIAL GASTRECTOMY.

VOLUME I.

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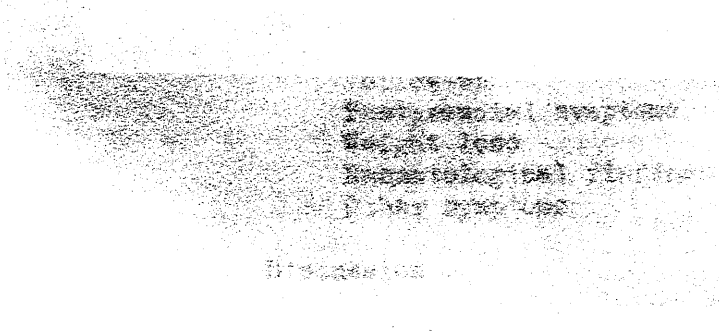
" I do not design, in the following remarks, to present any thing like a treatise on digestion. Works of this kind, treating of the subject both physiologically and pathologically, have so multiplied of late, as to render an attempt on my part, entirely a work of supererogation, even if I believed myself qualified for the task. I consider myself but a humble inquirer after the truth - a simple experimenter. "

William Beaumont - 1833.



VOLUME I.

CONTENTS. PAGES.

PAGES.

OUTLINE OF THESIS.

This Thesis presents a study of the altered physiology which may follow resection of part of the stomach. The incidence and types of clinical syndromes from which the patient may suffer after partial gastrectomy have been defined by a careful follow-up study and investigations of gastrointestinal motility, cardiovascular and electrocardiographic changes in response to a meal of glucose and absorption of glucose and of iron by the intestine have been carried out. The results of these investigations are discussed in relation to the various types of post-gastrectomy syndromes.

In Volume I, the main body of the Thesis, a description of the methods used and a statement and discussion of the results are presented. Volume II is an appendix to the main Thesis and includes details of the various studies.

ACKNOWLEDGEMENTS.

This work has been carried out during the past 3 years in the Professorial Surgical Unit at the Western Infirmary, Glasgow. It is with great pleasure that I thank Professor C.F.W. Illingworth for his keen interest and for making facilities available for this research.

I gratefully acknowledge the assistance of several colleagues in parts of the study, notably that of Dr. N.J. McKellar in the radiological investigations. Mr. W.T.Irvine stimulated my interest in the subject and helped in the pilot studies. Dr. J.W. Kerr took part in the electrocardiographic readings and Mr. J. Aitchison of the Mathematics Department of the University of Glasgow advised on the statistical analysis. The figures were the work of the Medical Illustration Department at the Western Infirmary - Mr. Gabriel Donald (Director), Miss K.J.Graham, Miss J. Brodie, Miss R. Henderson and Mr. A. Fotheringham. The execution of the final manuscript was by Miss A.R.Hamilton.

Finally, I wish to express my thanks to the patients who co-operated so willingly throughout the period of testing.

INTRODUCTION.

REVIEW OF LITERATURE.

INTRODUCTION.

REVIEW OF THE LITERATURE.

Partial gastric resection in man was first recorded in 1879 by Péan who performed the operation for a gastric cancer. Most of the early gastrectomies were for neoplasms but after the turn of the century it was advocated for peptic ulcer (Von Haberer, 1915). Since then it has become the most common surgical approach to the peptic ulcer problem, though, at present, it is challenged by the operation of gastrojejunostomy combined with subdiaphragmatic vagotomy.

While removal of two thirds or more of the stomach may relieve ulcer symptoms, it may give rise to many side-effects, some of which are most troublesome. One of the disappointing features is the occurrence of symptoms after eating and so the dyspeptic may be denied the long-awaited pleasure of having a normal meal with no undesirable consequences. In the first few weeks, many patients have a feeling of fullness in the epigastrium which may amount to discomfort. A minority have, in addition, faintness, tiredness, palpitations, sweating, pallor or subjective flushing, and sometimes nausea and bilious vomiting. This symptom complex is usually known as the "Dumping" Syndrome and it may last for years though many have spontaneous recovery within the first few months following operation. The symptoms often begin 15 to 20 minutes after eating and last up to an hour. Relief is usually obtained by lying down. Those patients /

/patients having trouble soon after a meal, were said to have the Early Postgastrectomy or "Dumping" Syndrome (Adlersberg and Hammerschlag, 1947; Zollinger and Hoerr, 1947) in contradistinction to a rarer group who had hypoglycaemic symptoms two to three hours after eating and were said to have the Late Postgastrectomy Syndrome. The physiological alterations in relation to the "Dumping" Syndrome are the main subjects for this study.

All symptoms are not necessarily present in the same patient and the attacks vary in severity for no apparent reason. There is a marked difference in the amount and type of food required to provoke an attack. In some cases a cup of tea is sufficient although sweet foods are more usually the cause. Mild degrees of the syndrome may easily be overlooked in routine follow-up examination but the full picture is unmistakable. Thus, the incidence reported will depend, to some extent, on the interest shown in eliciting the less clamant cases. This fact may account, to some extent, for the wide range to be found in the literature from nil (Henley, 1952) to about 75 per cent (Muir, 1949: Goligher and Riley, 1952). The majority of authors find 10 to 30 per cent affected (Allen and Welch, 1946: Bruusgaard, 1946: Custer et al., 1946: Butler, 1951: Palumbo et al., 1952: Pulvertaft, 1952: Wallensten and Gothman, 1953: Anderson et al., 1955).

The interest felt in the "dumping" syndrome is reflected in the number of reviews of its aetiology in recent years (Smith et al., 1953: Ferguson, 1955: Rosenberg and Matzner, 1957: Randall, 1958). Ample scope is afforded /

/afforded for these by the divergent opinions which have been held on the subject. A sure sign of the lack of true understanding is the multiplicity of explanations offered and the main outline of the more important will be summarised in the following order:-

Physical factors.

Stimulation of the autonomic nervous system.

Blood sugar changes.

Serum potassium changes.

Alterations in blood volume.

PHYSICAL FACTORS.

As is suggested by the name "Dumping", rapid emptying of the stomach with "dumping" of the meal into the small bowel was first thought to be the cause and, though there is disagreement about the secondary effects produced, it is still held that precipitate gastric emptying is of some importance in pathogenesis.

In 1913 the symptoms described above were noted in 20 patients after gastrojejunostomy by Hertz who attributed the upset to jejunal dilatation. Another patient with similar symptoms after gastrojejunostomy was recorded by Mix (1922) who used the expression "dumping" stomach. In patients after gastrectomy, the theory that jejunal distension was a causative factor was supported by Adlersberg and Hammerschlag (1947) and /

/and by Muir (1949). Rapid gastric emptying in patients with symptoms was shown radiographically by Machella (1949, 1950) who also produced symptoms, which he called "dumping", by the inflation of a balloon in the jejunum. He felt that jejunal distension was the cause of the "dumping" syndrome, a theory with which Goligher and Riley (1952) agreed.

However, Butler (1951) found no difference in the tendency to develop symptoms between dumping patients and a control group following jejunal distension with a balloon. Also against the jejunal distension were Custer and his colleagues (1946) who postulated that sudden emptying of the meal into the small bowel caused, not a distension, but rather a hypermotility. Increased motor activity of the jejunum and ileum also impressed Glazebrook and Wellbourn (1952). They were able to demonstrate hypermotility by means of kymographic and radiological studies and noted further that ganglion blockade with hexamethonium abolished the overactivity and the symptoms.

Stretching of the gastric remnant was considered to be a cause by Butler (1951) as a result of radiographic studies with patients in various postures. He observed that the symptoms occurred when the meal was still in the stomach and could be reproduced by putting a mercury-filled bag in the stomach. He thought that the main cause of the trouble was the loss of the supporting structures of the gastric remnant and had good results with operations designed to remedy this fault. Relief was also /

/also obtained by Capper (1951) in eight out of nine patients with severe "dumping" symptoms by reconstructing the peritoneal supports of the residual part of the stomach. He was of the opinion that gastric stretch was important in the aetiology of the syndrome. The contrary view was held by Abbott (1952) who was able to reduce the incidence of "dumping" by mobilising the gastric remnant, so having good results with a stomach almost totally deprived of its peritoneal supports.

The type of anastomosis used to reconstitute the alimentary canal after partial gastrectomy has been thought to be important by many authors and numerous claims have been made that the gastroduodenal anastomosis of the Billroth I operation gives rise to less "dumping" than the gastrojejunal one of the Polya and related procedures (Bruusgaard, 1946; Custer et al., 1946; O'Neill, 1950; Butler, 1951; Ross and Meadows, 1952; Wallensten, 1954). In fact, the change from gastrojejunal to gastroduodenal anastomosis for severe "dumping" has been recommended (Perman, 1947; Bohmansson, 1950; Tanner, 1951). As seems inevitable on the subject of "dumping", this opinion is disputed by others who maintain there is no difference in incidence between the two types of anastomosis, though the symptoms seem to be less severe in the patients with a Billroth I resection (Moore and Harkins, 1952; Walters et al., 1957).

One mechanical effect on which there is fairly general agreement is that there is a higher incidence of /

/of "dumping" the more radical the gastrectomy (Perman, 1947; Goligher and Riley, 1952; Pulvertaft, 1952; Babb et al., 1953; Johnson and Orr, 1953).

STIMULATION OF THE AUTONOMIC NERVOUS SYSTEM.

An autonomic nervous system reflex triggered by the rapid entry of the meal into the small bowel was suggested as a cause of "dumping" symptoms by Hoffman (1939) who thought that a hyperaemia of the splanchnic bed resulted with consequent symptoms from slight cerebral anoxia. A similar hypothesis was advanced by Perman (1947). He held that an insufficiency of the circulation was caused by a reflex from the intestine and that patients with "dumping" had an imbalance of the sympathetic nervous system. Other workers who have considered stimulation of the sympathetic important are Irvine (1948), Capper (1951), Smith (1951) and Roberts et al., (1954). Further evidence of the part played by the sympathetic nervous system was obtained by Butler and Capper (1951) in giving relief to some patients by means of a splanchnic block. As has been noted already, Glazebrook and Welbourn (1952) made use of hexamethonium for the treatment of symptoms. This drug, however, has a wide ganglion blocking action and cannot be said to be confined to the sympathetic nervous system alone.

Many of the symptoms produced by a meal in the susceptible patient, for example, sweating, tachycardia and /

/and dizziness, are compatible with adrenaline release and Smith and his colleagues (1953) reproduced these symptoms by adrenaline administration. The role of adrenaline was supported by Pontes and Neves (1953) by finding at the time of symptoms haematological and biochemical changes which they interpreted as favouring the hypothesis of adrenal stimulation. A rise in blood catechols was observed by Pulvertaft (1954) but this could not be correlated with the symptoms. Against the theory of adrenaline release was the work of Rauch and Bieter (1953) and of Smith and his colleagues (1953) which failed to show any relief in the patients with the syndrome after the administration of adrenolytic drugs and also the fact that giving ephedrine had benefitted some sufferers (Custer et al., 1946; Gilbert and Dunlop, 1947; Capper, 1951).

Some negative evidence concerning the possible pathway of the reflex was given by Moore (1947) when he recorded "dumping", which lasted for eight weeks, in a patient after vagotomy and concluded that the sensory arc of the reflex was not carried by the vagus. Sensory impressions from the bowel can be conveyed by sympathetic nerves, as bilateral splanchnicectomy abolishes sensations caused by distension of the small bowel with a balloon (Ray and Neill, 1947) so that the afferent route from the gut in "dumping" could well be via the sympathetic nervous system. /

/system.

BLOOD SUGAR CHANGES.

It has long been known that attacks of hypoglycaemia can follow a meal in some patients after gastrectomy (Evensen, 1942) and this was considered to be a likely cause of "dumping" by Gilbert and Dunlop (1947). However, it has been shown that the symptoms and the fall in the blood sugar do not coincide except in the Late Postgastrectomy Syndrome (Adlersberg and Hammerschlag, 1947; Irvine, 1948; Schechter and Necheles, 1949).

Other alterations in the blood sugar level have been blamed. Alimentary hyperglycaemic shock was the term used by Glaessner (1940) to describe the effect of the rise in the blood sugar which he observed at the time of symptoms. He was able to produce symptoms in these patients by giving glucose or sucrose but not with the more slowly absorbed laevulose. This theory was further supported by later work (Glaessner, 1945; Winklestein, 1947). Doubt was cast on the role of increased blood sugar by Schwartz and his co-workers (1942) and Schechter and Necheles (1949) expressed the opinion that the concept of hyperglycaemic shock was not valid, there probably being no relationship between symptoms and the abnormal glucose tolerance curves. The use of insulin to reduce the high blood sugar after a meal failed to prevent "dumping" (Custer et al., 1946). Finally, a connection between the rate of absorption of sugar and the vasomotor symptoms and the /

/the electrocardiographic changes was postulated by Pulvertaft (1954).

POTASSIUM CHANGES.

A fall in the serum potassium during "dumping" attacks was noted along with electrocardiographic and myographic changes suggestive of impaired muscle function (Smith, 1951). The intravenous administration of potassium reversed these electrocardiographic changes, prevented the muscle weakness and abbreviated the attacks. Further work (Smith et al., 1953) suggested that rapid absorption of sugar and release of adrenaline might both contribute to the later fall in serum potassium, although Pulvertaft (1954) found that symptoms usually occurred prior to the hypokalaemia. No correlation could be found by Roberts and her co-workers (1954) between symptoms and lowered levels of the serum potassium.

ALTERATIONS IN BLOOD VOLUME.

More recently, attention has been focussed on circulatory changes and a decrease in the blood volume was demonstrated at the time of symptoms induced by a glucose meal in patients after gastrectomy (Roberts et al., 1954; 1955; Walker et al., 1955), concomitant with electrocardiographic changes similar to those reported by Smith (1951). Hypotension accompanied these findings in most cases and it was thought that the fall in blood /

/blood pressure might result in stimulation of the sympathetic from pressoreceptors in the arch of the aorta. The electrocardiographic changes were explained on the basis of coronary insufficiency. Similar observations in blood volume were made by Amdrup and Jørgensen (1956a; 1957), a fall being noted in patients with symptoms and a rise in those not affected. Another study on this aspect of the problem (Hinshaw et al., 1957) revealed that some patients had a decrease in blood volume without "dumping" symptoms and digital plethysmography demonstrated a simultaneous increase in peripheral blood flow only in the patients with marked symptoms. It was suggested that, by this means, a peripheral intravascular shift of considerable quantities of blood could occur resulting in reduced flow to central vital areas such as the brain and the heart. Some inconsistency between the magnitude of plasma volume decrease and the severity of the "dumping" symptoms was found by Everson and Abrams (1958). The change in blood volume is usually attributed to the osmotic effect of the hypertonic meal withdrawing fluid into the lumen of the intestine, an event which Amdrup and his co-workers (1958) claim to have demonstrated radiographically.

Various other influences have been suggested - jejunitis (Porges, 1947; Adlersberg and Hammerschlag, 1949), allergy due to the release of antibodies in the gut wall by the meal (Zeldis and Klinger, 1951) and psychosomatic factors (Alvarez, 1949; Bell, 1953; Harvey et al., 1953). /

In short, it can be seen that several changes are said to occur in the response to a meal after the operation of gastrectomy. The gastric and small bowel motility, the cardio-vascular effects (including the electrocardiogram), the blood sugar and the serum potassium may all be altered. Little general agreement exists in the literature concerning the mechanism or the significance of these altered responses or their relationship to the "Dumping" Syndrome, with the one exception that rapid gastric emptying is fairly generally agreed to be the first in the chain of events.

Another side effect of gastrectomy is an increased susceptibility to Hypochromic Anaemia. On this subject the reported incidence again varies widely but is almost uniformly greater in women than in men. The range, for women, is from 19 per cent (Gaviser, 1948) to 82 per cent (Dedichen, 1934), the usual figure being 40 to 60 per cent (Larsen, 1934; Drablos et al., 1951) and for men, from nil (Gaviser, 1948) to 58 per cent (Morley and Roberts, 1928) usually about 30 per cent (Bruusgaard, 1946; Blake and Rechnitzer, 1953). One reason for this scatter is the different criteria used to define when a patient is anaemic. The problem of anaemia after gastrectomy has also been the subject of reviews (Lyngar, 1950; Baird et al., 1959) and a summary of the main trends of thought now follows.

It has been generally suggested that the main cause for the hypochromic anaemia after partial gastrectomy is a defect /

/defect in the absorption of iron. This is based on the finding that maximal absorption takes place in the duodenum, diminishing rapidly along the small bowel (Hahn et al., 1943; Endicott, 1949; Granick, 1949; Stewart et al., 1950). This important region can be disturbed in two ways by the operation. The first is by the increased speed of gastric emptying, which conveys the iron rapidly away from the sites of greatest assimilation, an action which has been said to cause malabsorption (Owren, 1952). The second is by the short-circuiting of the duodenum in the cases with a gastrojejunal anastomosis. Both factors were thought to be important by Larsen (1952). Other evidence of the role of this area is found, indirectly, in the lower incidence of anaemia in the patients with a gastroduodenal anastomosis where the duodenum is left in continuity than in those who have the duodenum by-passed when a gastrojejunal reconstitution is used. (Morley and Roberts, 1928; Bohmansson, 1950; Wallensten, 1954). A fall in gastric acidity after operation was blamed by Bruusgaard (1946) but Watson (1947), following a study in which he used fractional test meals, expressed the belief that hypochromic anaemia was not related to the presence or absence of free hydrochloric acid. By using serum iron curves to assess iron absorption Grace and Doig (1953) showed that the degree of gastric acidity was without influence on the absorption of iron, findings which were in keeping with those of Moore (1955) following studies in which he used radioactive iron to estimate the absorption rate. /

/rate.

Although there has been much theorising on the subject of iron absorption after gastrectomy, it is only recently that reliable information has become available. Studies of the absorption of radioactive iron salts in patients before and after partial gastrectomy (Baird et al., 1957; Smith and Mallett, 1957) has shown no significant difference between the two groups. In a similar project, making use of organic iron labelled with radioactive iron, Baird and Wilson (1959) also noted no change in iron assimilation after operation but found that, while most anaemic patients with intact stomachs have an increased iron absorption, those who were anaemic after gastrectomy still absorbed an amount which was within the normal range. They believed that this failure to respond to a fall in the body iron stores was important in the susceptibility to anaemia. Poor absorption of food iron after gastrectomy was also found by Chodos and others (1957) and Pirzio-Biroli and his colleagues (1958).

Contributory factors which have been mentioned are a poor intake of iron in the diet (Baird et al., 1959) and continued occult blood loss (Witts, 1956).

In brief, it may be said that most authors believe that rapid passage of iron through the duodenal loop and the upper jejunum, or past the duodenum and through the upper jejunum, is the main cause of the hypochromic anaemia after partial gastrectomy. This opinion is based largely on the high incidence of the /

/the anaemia, and the theoretical consideration that iron absorption is maximal in the duodenum and upper jejunum. The information from the iron absorption studies shows that the assimilation of inorganic salts is not influenced by the operation but there is some failure to increase the absorption from organic complexes when anaemia is present. It is well established clinically that there is a lower incidence of anaemia after the Billroth I operation than after the Polya procedure and an investigation into the possible mechanism behind this fact comprises the final part of this thesis.

From the review of the literature it can be seen that several changes in the normal response of the body to a meal have been observed in patients after partial gastrectomy. The alterations may well have some bearing on the aetiology of the "Dumping" Syndrome and on the increased susceptibility to hypochromic anaemia but no clearly defined relationship has been found.

SYMPTOMS SIX YEARS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

A FOLLOW-UP STUDY.

SYMPTOMS SIX YEARS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

In order to clarify the relative importance of the symptoms which may be present after partial gastrectomy and to observe some of their natural history, a consecutive series of patients who underwent operation in 1952 was studied. The 6-year period was chosen to allow adequate time for late effects to make their appearance.

MATERIAL AND METHODS.

A series of 132 patients who underwent partial gastrectomy for peptic ulcer in Professor Illingworth's unit in 1952 was studied. All operations were of the Polya type with a gastrojejunal anastomosis; this was either ante- or retro-colic in position. Approximately two thirds of the stomach was removed. Patients were examined personally and a history of the events since operation compiled by interrogation aided by consultation of routine hospital records. Patients were weighed and particular note made of the presence of postprandial symptoms and evidence of blood loss. The weight at the interview was compared with the average weight in health before operation.

The following groups of postprandial symptoms were recognized:

Epigastric fullness - A complaint of fullness and of distension in the epigastrium, usually beginning about 10 to 20 minutes after a meal.

"Dumping" or early postgastrectomy syndrome - To qualify for inclusion in this group, the patients had to experience some vasomotor upset - faintness, dizziness, sweating, palpitations, pallor, or subjective flushing. Epigastric fullness was usually present and bilious vomiting might occur. Patients complaining of "dumping" were subdivided into two grades - "moderate" and "severe" - according to the severity of the symptoms and the degree of inconvenience caused to the patient. Symptoms were classified as severe only when they were of daily occurrence and when they resulted in loss of work. Otherwise they were classed as moderate. Although this assessment obviously depends on the personality of the patient and his desire, or lack of desire to work, it is appropriately based on the effect of the symptoms on his ability to lead a normal life.

Bilious vomiting - Vomiting or regurgitation of bile, with or without vasomotor symptoms, were included under this heading which was subdivided into severe and moderate on the same criteria as above.

Several patients were having symptoms which were listed under more than one title. In order to keep the factor of suggestion as low as possible, the following question was asked routinely:

"Since your operation in 1952, have you had any trouble after your meals, for example, a fullness in your stomach, any feeling of faintness, tiredness, sweating, dizziness, any /

/any vomiting or any pain?"

In this way no particular emphasis was put on any one complaint. The time of onset of symptoms, the presence of any possible precipitating factor and the progress of the symptoms over the years were recorded.

Possible chronic blood loss was specifically enquired about and in particular the patient was asked if he had ever noticed bleeding from the rectum.

As far as could be judged at one interview, a rough assessment was made of the personality of the patient.

All patients were clinically examined, the haemoglobin (Hb. alkaline haematin method of Gibson and Harrison, 1945) and packed cell volume (PCV: 3000 r.p.m. for 30 min.) were estimated and the mean corpuscular haemoglobin concentration (MCHC) calculated. When patients had haemoglobin levels under 12.32 g. per cent, films of the peripheral blood were examined.

CONTROL STUDY.

To provide a suitable standard for comparison, the Hb., PCV and MCHC were determined on 40 men, aged 35 to 65 years, who had no history of haematological disorder or blood loss. The control readings were intermingled with those performed in the patients after gastrectomy so that each batch of estimations was individually controlled.

STATISTICAL ANALYSIS.

The difference in the findings between the various groups of patients were compared by standard statistical methods (Fisher, 1930: Moroney, 1953) using χ^2 and Student's "t" distributions as measures of significance (Fisher and Yates, 1948).

The following abbreviations have been used:

S.D.	-	Standard deviation
S.E. Mean	-	Standard error of the mean
S.E. Diff. Mean	-	Standard error of the difference between means
P.	-	Probability
d.f.	-	Degrees of freedom
t.	-	Student's "t"

RESULTS.

FOLLOW-UP.

Details of the follow-up are shown in Table I. Only three of 132 patients could not be traced and eleven had died in the 6-year interval since operation. Three patients were under treatment for pulmonary tuberculosis in sanatoria and nineteen were too far away to attend (London, Rhodesia, U.S.A.). Ninety-six patients were available for follow-up and all were personally interviewed.

TOTAL NO. OF PATIENTS OPERATED UPON		132
PATIENTS UNTRACED AT FOLLOW-UP		3
PATIENTS TRACED AT FOLLOW-UP		129
NOT AVAILABLE FOR INTERVIEW		33
DEAD	11	
TOO DISTANT	19	
SANATORIA	3	
AVAILABLE FOR INTERVIEW		96

TABLE I.

ANALYSIS OF 132 PATIENTS WHO HAD PARTIAL GASTRECTOMY FOR PEPTIC ULCER IN 1952.

RESULTS.

POSTPRANDIAL SYMPTOMS.

Symptoms of this type were present in a total of seventy-six patients (79 per cent) but in only nine patients (9.4 per cent) were the symptoms considered severe (Table II).

Epigastric fullness occurred in seventy-two patients (75 per cent).

"Dumping" or early postgastrectomy symptoms - Forty-five patients (46 per cent) complained of "dumping" at some time since the operation. In seven the symptoms were severe and further operative measures were necessary in three cases. The onset and duration of these symptoms were variable. Eleven of the forty-five patients who had "dumping" no longer had trouble at the time of the follow-up, the last attack being more than 6 months previously. In a further twenty-five cases the attacks had been troublesome for the whole 6 years since operation. In most of these patients the severity and frequency of attacks had diminished with the passing of time and many, when seen, were having an attack only about once a month. Several patients had no such improvement and in five the symptoms were still severe. The remaining nine patients (20 per cent of the forty-five with "dumping" symptoms) had not complained of the syndrome until 1 to $4\frac{1}{2}$ years after operation (Table III). The time of onset of symptoms could not be correlated with any other factor from the history.

Fifteen patients had bilious vomiting as well as the /

POSTPRANDIAL SYMPTOMS	NO. OF PATIENTS	PER CENT OF TOTAL SERIES OF 96 PATIENTS
EPIGASTRIC FULLNESS	72	75
DUMPING SYMPTOMS	45	46
SEVERE	7	7.3
BILIOUS VOMITING	30	31
SEVERE	5	5.2
TOTAL POSTPRANDIAL SYMPTOMS	76	79
TOTAL SEVERE	9	9.4

TABLE II.

ANALYSIS OF POSTPRANDIAL SYMPTOMS IN 96 PATIENTS FOLLOWED-UP 6 YEARS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

PATIENTS WITH "DUMPING" SYMPTOMS	NO.	PER CENT OF TOTAL
INITIAL "DUMPING" - LATER REMISSION	11	24
ATTACKS PERSISTING THROUGHOUT 6 YEARS	25	56
LATE ONSET OF SYMPTOMS	9	20
TOTAL	45	100

TABLE III.

DURATION AND ONSET OF "DUMPING" SYMPTOMS IN 45 PATIENTS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

/the "dumping" symptoms.

Women were affected by this syndrome less commonly than men; only two of eleven women complained of "dumping" compared with forty-five of eighty-five men. This difference is not significant in the number of patients studied (Table IV).

The incidence of "dumping" was the same (46 per cent) whether the anastomosis was antecolic or retrocolic in position.

The mean haemoglobin level in those men who had "dumping" symptoms did not differ from the level in those not affected (Table V). The incidence of anaemia (Hb < 12.32 per cent) in those patients with "dumping" symptoms was no higher than that in the series as a whole (Table VI).

Although the estimate of personality was a rough one, patients with severe symptoms were found to be of poorer type than those without symptoms. It was not possible to determine to what extent this contributed to, or resulted from the "dumping" syndrome.

Bilious vomiting. The incidence of bilious vomiting in the patients followed up was less than that of "dumping" symptoms and it occurred in only thirty patients (31 per cent of the whole series) (Table II). In five cases bilious vomiting was severe; in three of these patients severe "dumping" was also present. Bilious vomiting had ceased by the time of the interview in ten patients but had continued throughout the 6 years since operation in twenty, although generally they became less severe over the years. The onset of bilious vomiting was delayed (1 to 5 years) in /

	TOTAL	NO. WITH DUMPING SYMPTOMS	PER CENT OF TOTAL	χ^2	d.f.	P
WOMEN	11	2	19	2.07	1	> 0.1
MEN	85	43	51			

TABLE IV.

RELATIVE PROPORTIONS OF WOMEN AND MEN AFFECTED BY "DUMPING" SYMPTOMS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

DUMPING SYMPTOMS	NO. OF PATIENTS	MEAN Hb g. PER CENT
PRESENT	43	12.7 ± 0.21 *
ABSENT	42	12.8 ± 0.26 *
MEAN DIFFERENCE		0.1 ± 0.33 **

* S.E. Mean

** S.E. Diff. Means

TABLE V.

MEAN HAEMOGLOBIN LEVELS IN 85 MEN AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER, COMPARING THOSE WITH AND THOSE WITHOUT "DUMPING" SYMPTOMS.

ANAEMIA (Hb.< 12.32 g.)	NO. OF PATIENTS			χ^2	d.f.	P
	DUMPING SYMPTOMS		TOTAL			
	PRESENT	ABSENT				
PRESENT	16	11	27	0.86	1	> 0.3
ABSENT	27	31	58			

TABLE VI.

THE INCIDENCE OF "DUMPING" SYMPTOMS IN RELATION TO THE PRESENCE OF ANAEMIA IN 85 MEN AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

/in eight patients (27 per cent of those having bilious vomiting) (Table VII). The incidence of bilious vomiting was not related to sex or to the type of anastomosis performed.

It is worthy of comment that two thirds of the patients who had "dumping" or bilious vomiting following gastrectomy had noted an improvement in their symptoms over the years. In many of those patients in whom the symptoms persisted for 6 years, they occurred only about once a month and rarely caused great inconvenience.

WEIGHT LOSS.

A satisfactory comparison of pre- and post-operative weights was achieved in eighty-nine patients of whom sixty had lost weight, sixteen remained the same and thirteen gained weight (Table VIII). Twenty-four patients were at least 10 per cent below their average weight in health before operation and of these fourteen were suffering from "dumping" symptoms (Fig. 1). This proportion is not significant ($\chi^2 = 1.50$ d.f. = 1, $P > 0.2$). The mean weight change in the whole series was a fall of 5.1 per cent. The mean weight loss in the seven patients with severe "dumping" symptoms was 10.1 per cent, compared to that of 5.0 per cent in the remainder of the series. This difference is not statistically significant whether arithmetic or logarithmic values are used (Table IX).

PATIENTS WITH BILIOUS VOMITING	NO.	PER CENT OF TOTAL WITH BILIOUS VOMITING
INITIAL BILIOUS VOMITING - LATER REMISSION	10	33
ATTACKS PERSISTING THROUGHOUT 6 YEARS	12	40
LATE ONSET OF SYMPTOMS	8	27
TOTAL	30	100

TABLE VII.

DURATION AND ONSET OF BILIOUS VOMITING IN 30 PATIENTS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

	NO. OF PATIENTS	PER CENT OF 89 PATIENTS
WEIGHT LOST	60	67
WEIGHT STATIC	16	18
WEIGHT GAINED	13	15
TOTAL	89	100

TABLE VIII.

CHANGES IN WEIGHT RELATIVE TO THE AVERAGE WEIGHT IN HEALTH OF 89 PATIENTS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

+ S.E. Mean ++ S.E. Diff. Means.

Mean weight loss in patients after Partial Gastrectomy for Peptic Ulcer, comparing those with severe dumping symptoms and the remainder of the series.

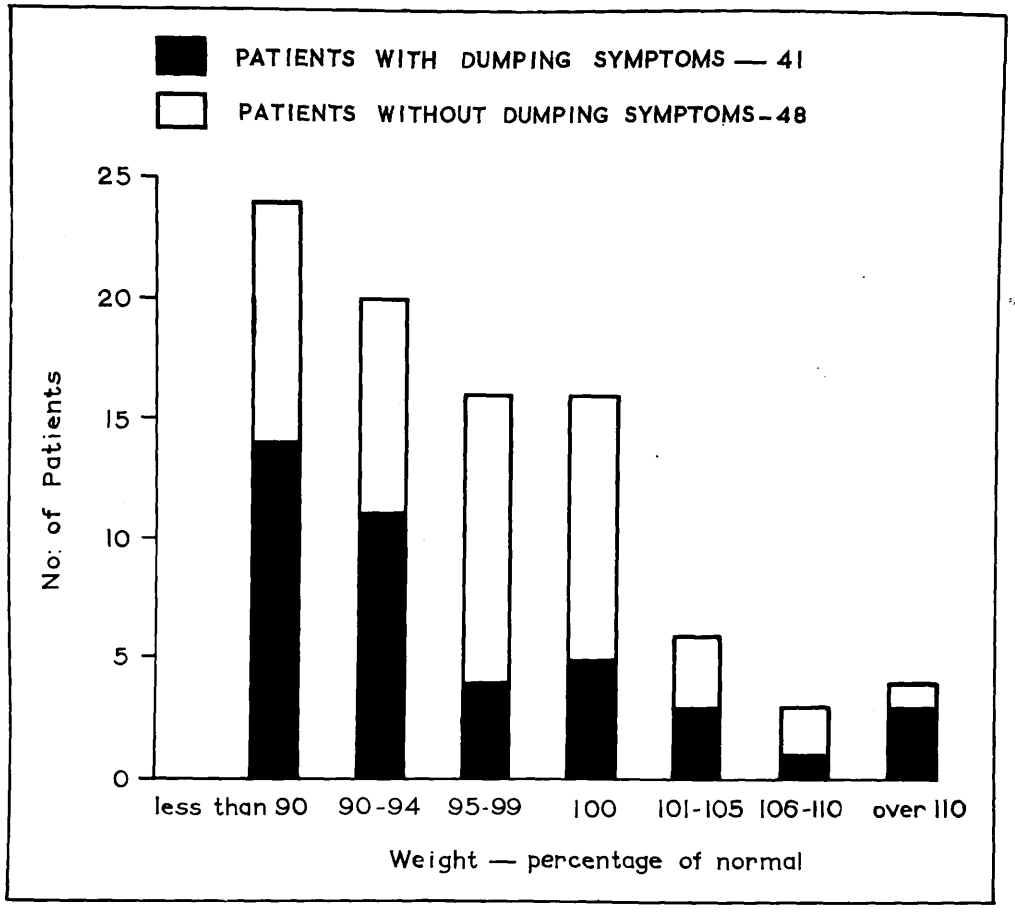


FIG. 1. DISTRIBUTION OF WEIGHTS OF 89 PATIENTS 6 YEARS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER COMPARED WITH THEIR AVERAGE WEIGHT IN HEALTH BEFORE OPERATION.

	NO. OF PATIENTS	MEAN WEIGHT LOSS PER CENT
SEVERE DUMPING SYMPTOMS	7	10.1 ± 1.53*
REMAINDER OF SERIES	82	5.0 ± 0.95*
MEAN DIFFERENCE		5.1 ± 3.19**
t		1.59
P		> 0.05

* S.E. Mean

** S.E. Diff. Means

TABLE IX.

MEAN WEIGHT LOSS IN PATIENTS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER, COMPARING THOSE WITH SEVERE DUMPING SYMPTOMS AND THE REMAINDER OF THE SERIES.

(As the distribution of the logarithm of the weight is more nearly normal than that of the weight itself, the above relationship was checked using logarithms and $t = 1.90$, $d.f. = 88$, $P > 0.05$).

HAEMATOLOGICAL FINDINGS.

MEN.

The age of the control group of forty men (average 47.0 years S.D. ± 8.45) is closely comparable to that of the eighty-five men studied after gastrectomy (average 47.6 years S.D. ± 9.54). In the control group the mean level of Hb was 14.7 g. per cent (S.E. Mean ± 0.20). The lowest normal Hb on the basis of the 95 per cent confidence limits, is 12.32 g. per cent. Patients with an Hb reading below this level were regarded as anaemic.

The mean level of Hb in the eighty-five men who were studied after gastrectomy was 12.75 g. per cent (S.E. Mean ± 0.16). This is significantly different from the level in the controls (Table X). The two groups are compared in Figure 2. Using the criterion defined above, twenty-seven of the eighty-five men after gastrectomy were anaemic. Sixteen of these had "dumping" symptoms, a proportion which is not significant (χ^2 = 2.0 d.f. = 1, P > 0.1). In the patients with anaemia the PCV and MCHC were significantly lower than those in the control group confirming that the anaemia was hypochromic in type (Table XI). Evidence of macrocytic anaemia was not found in the blood films of any patient.

A history of blood loss, mainly from haemorrhoids, was found in ten of the twenty-seven men who were anaemic and in eleven of the fifty-eight men who were not anaemic after /

PATIENTS	NO.	MEAN Hb g. PER CENT
CONTROLS	40	14.70 ± 0.20*
AFTER GASTRECTOMY	85	12.75 ± 0.16*
MEAN DIFFERENCE		1.95 ± 0.27**
t		7.22
P		< 0.001

* S.E. Mean

** S.E. Diff. Means

TABLE X.

MEAN HAEMOGLOBIN LEVEL IN 85 MEN AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER COMPARED WITH THE MEAN LEVEL IN A CONTROL SERIES OF 40 MEN.

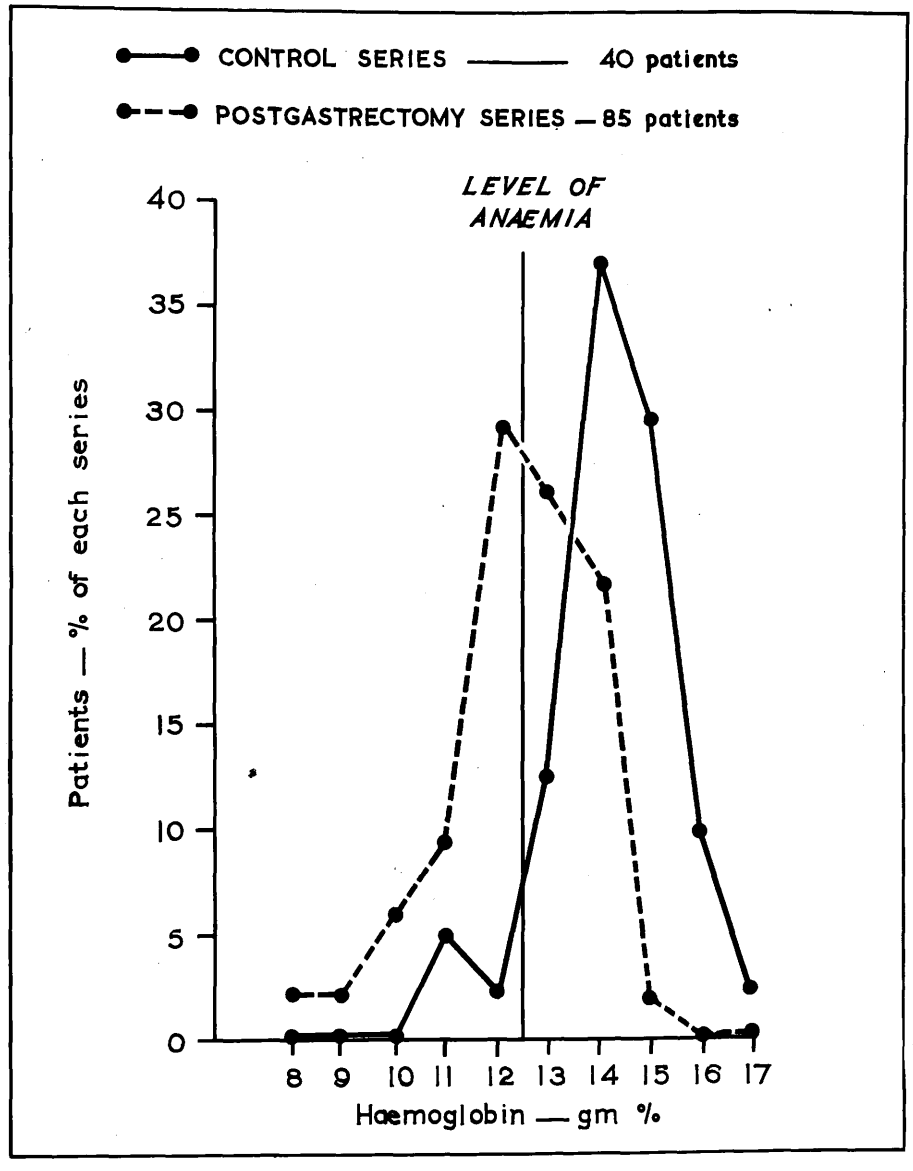


FIG. 2. HAEMOGLOBIN LEVELS OF 85 MEN 6 YEARS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER COMPARED WITH THOSE OF 40 "NORMAL" MEN OF COMPARABLE AGE.

PATIENTS	NO.	PCV PER CENT	MCHC PER CENT
CONTROLS	40	45.0 ± 0.61*	31.9 ± 0.36*
ANAEMIC AFTER GASTRECTOMY	27	40.3 ± 0.73*	29.4 ± 0.57*
MEAN DIFFERENCE		4.7 ± 0.90**	2.5 ± 0.62**
t		5.22	4.03
P		< 0.001	< 0.001

* S.E. Mean

** S.E. Diff. Means

TABLE XI.

MEAN PCV AND MCHC LEVELS IN 28 MEN ANAEMIC AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER COMPARED WITH THE LEVELS IN A CONTROL SERIES OF 40 MEN.

/after gastrectomy. The larger proportion in the former group is statistically significant ($\chi^2 = 4.02$ d.f. = 1 $P < 0.05$). This finding is illustrated in Figure 3.

WOMEN.

In the eleven women who were studied after gastrectomy the mean values were: Hb 11.47 g. per cent, PCV 39.2 per cent, and MCHC 29.4 per cent.

OTHER SYMPTOMS.

Two cases (2.1 per cent) had proven recurrent ulceration on radiological or gastroscopic examination. Symptoms of recurrent ulcer were present in four other patients although the investigations were negative.

The cause of death in three of the eleven patients who had died in the 6 years since operation was pulmonary tuberculosis. Three additional patients were in sanatoria and two others had been diagnosed, one of whom had a left lower lobectomy in 1955. Thus eight of 110 patients traced (7.3 per cent) had pulmonary tuberculosis after operation.

DISCUSSION.

The most important symptoms of altered physiology after partial gastrectomy for peptic ulcer are of the postprandial type. Their incidence is high, 79 per cent of patients being affected /

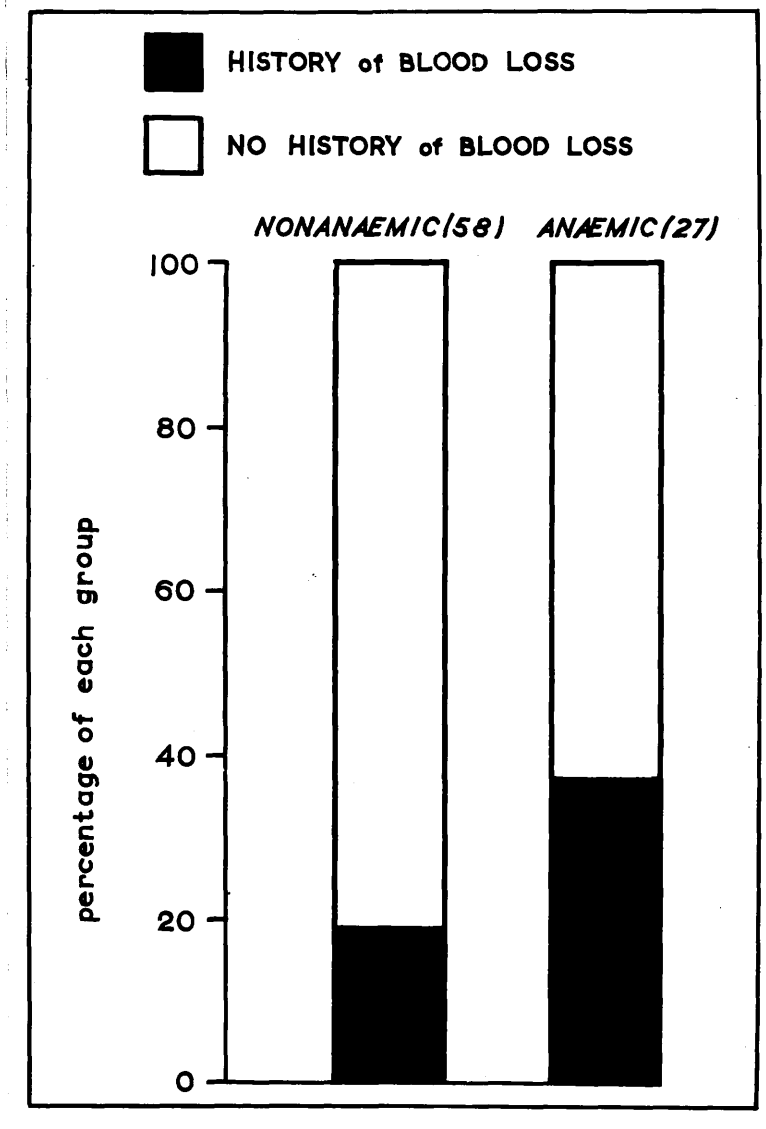


FIG. 3. INCIDENCE OF A HISTORY OF BLOOD LOSS IN 85 MEN 6 YEARS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER COMPARING PATIENTS WITH AND WITHOUT ANAEMIA (Hb 12.32 g. PER CENT).

/affected to some extent. The vasomotor symptoms of the "dumping" or early postgastrectomy syndrome cause greatest upset. Patients are little disturbed by a feeling of fullness in the epigastrium; but the uncertainty induced by faintness or weakness, especially in an active occupation, can be most incapacitating. Seven people were severely affected by this syndrome. Their disability is as great as that from ulcer symptoms before operation and has resulted in loss of working time. Strangely enough, despite its social inconvenience, bilious vomiting usually caused less disability. Attacks were more predictable and were quickly relieved, the patient being ready to resume his activities almost immediately afterwards. Weight loss in itself may be a little disquieting but does not lead to financial loss. Hypochromic anaemia also accounts for lack of full well-being after this operation. It all too frequently goes undetected and prophylactic iron therapy is often omitted after the first year.

"DUMPING" SYMPTOMS.

The different incidence of "dumping" in various reported series of patients after gastrectomy is more apparent than real for it depends on the definition of the syndrome. The highest incidence after partial gastric resection, namely about 75 per cent, was reported by Muir (1949) and by Goligher and Riley (1952). All types of postprandial upset are included under this heading by Muir, while Goligher and Riley extend their /

/their interpretation of the term "dumping" to cover some patients who may have only epigastric fullness or bilious vomiting. In the present series the figure for all postprandial symptoms was 79 per cent which is similar to that which these writers found for "dumping". Authors reporting an unduly low incidence may have overlooked the less obvious cases in routine follow-up or may have been dealing with patients treated with a less extensive operation.

The relatively high incidence of "dumping" symptoms in the present series (46 per cent) may be due to specific enquiry for the minor upsets. The "hard core" of patients with severe symptoms was 7.2 per cent which is within the range reported by most authors (Muir, 1949 - 12 per cent; Capper and Butler, 1951 - 4.7 per cent; Goligher and Riley, 1952 - 10 per cent; Pulvertaft, 1952 - 13 per cent; Anderson et al., 1955 - 5.5 per cent).

It is generally agreed that the early postgastrectomy syndrome tends to become less evident as time passes. The present results indicate that the number of complete remissions is not as great as generally thought and that many patients who will say that they are "better" when first asked are found on closer questioning to mean better rather than cured, and still to have definite though infrequent attacks. In this investigation 25 per cent had a complete remission but the remainder were having some attacks at the time of interview, although /

/although diminished in number and severity in most cases. Previous workers have observed that "dumping " rarely starts after the first few months (Moore and Harkins, 1954) and in fact 3 months has been put as the limit by Muir (1949). In the present study a fifth of the patients who had "dumping" first noticed the attacks from 1 - 4½ years after operation. Pulvertaft (1952) mentioned that this could occur but did not give any indication of its frequency.

The relationship of iron deficiency to "dumping" was investigated by Fehr and Ott (1950) by means of serum iron curves. They believed that iron deficiency could play a part in the complex picture of the "dumping" syndrome. In a later study, Remy et al., (1953) found "dumping" symptoms were more prevalent in those patients who were anaemic after gastrectomy than in those whose haemoglobin level was within normal limits. Treatment with iron gave relief of symptoms in some cases (Ott and Jasinski, 1954). The results reported here do not show any significant association between the occurrence of "dumping" and anaemia.

HYPOCHROMIC ANAEMIA.

To judge when patients are anaemic, previous authors have taken an arbitrary standard for the haemoglobin values (for example Morley and Roberts, 1928; Gordon Taylor, 1929; Wells and Welbourn, 1951), have used a range of ulcer patients as controls (Lyngar, 1950) or have accepted the values in a large /

/large population as their standard (Blake and Rechnitzer, 1953). In the present series a control study was done on "normal" patients of the same age group. The control observations were carried out simultaneously with those of the patients after gastrectomy, providing a control for each batch of estimations and a check on any local variation in the haemoglobin level with age. Using the lower of the 95 per cent confidence limits of the control series as the criterion for anaemia, 32 per cent of the mean after gastrectomy were found to be anaemic. Despite the fact that this level for anaemia is lower than that usually chosen, the incidence is comparable to that reported by others.

An abnormally high percentage of patients with anaemia were suffering from chronic blood loss, indicating the importance of this factor in the production of the anaemia. This finding agrees with the suggestion of Witts (1956) that the blood loss is an important aetiological factor and contrasts with the work of Baird and co-workers (1959) who were unable to find any history of blood loss in the anaemic patients after gastrectomy.

No comparisons have been made of the findings of the eleven women in the present study as any subdivision of this group gave very small numbers.

OTHER RESULTS.

Weight loss after partial gastrectomy is common /

/common and was present in two-thirds of the patients studied in this series. Twenty-six per cent of the patients were 10 per cent or more below their normal weight. These figures are in keeping with previous reports (Zollinger and Ellison, 1954). The failure to maintain weight has been attributed to a poor dietary intake consequent to postprandial symptoms (Muir, 1949; Brain and Stammers, 1951; McLean et al., 1954) but Shingleton and his colleagues (1957) discovered no correlation between dietary calorific intake and weight after gastrectomy. In this present follow-up study, the incidence of weight loss in those with "dumping" symptoms did not differ from that in the patients without symptoms.

The incidence of proven recurrent ulceration is in the usual range for Polya partial gastrectomy (Spira, 1956).

The incidence of pulmonary tuberculosis (7.3 per cent) in this study is greater than that previously reported (Pulvertaft, 1952; Anderson et al., 1955). This apparent increase may be due to the recent campaign in tuberculosis diagnosis in the West of Scotland rather than to a true increase.

CONCLUSIONS.

The "dumping" or early postgastrectomy syndrome was found in forty-five of ninety-six patients (46 per cent) available for follow-up 6 years after partial gastrectomy for peptic ulcer. /

/ulcer. It was notable that symptoms persisted during the entire period in 55 per cent of those affected and that, in a further 21 per cent, symptoms first occurred more than one year after operation. Symptoms were severe in seven patients of the total series 7.3 per cent). The type of anastomosis and the degree of anaemia did not affect the incidence.

Anaemia was present in twenty-seven of eighty-five men after operation (32 per cent) using a comparable series of forty men as controls. A history of blood loss was more common in the anaemic group and the difference was probably significant.

A loss of weight was observed in two-thirds of eighty-nine patients who had comparable estimations before and after operation. This loss was more than 10 per cent of the normal weight in 26 per cent of patients. Loss of weight was not significantly greater in those with "dumping" symptoms.

Proved recurrent ulcer occurred in 2.1 per cent of gastrectomy patients. Pulmonary tuberculosis developed in 7.3 per cent.

It must be stressed, that the symptoms detailed in this section, were elicited after repeated questioning at previous routine follow-up interviews often by observers other than the present. It follows that symptoms, real or imagined, have been fully documented here. In view of this, one may fail to appreciate that most of these symptoms were well tolerated, indeed many patients hardly thought fit to mention them. When compared to the loss of time from work before operation the amelioration after gastrectomy in many cases was striking.

GASTROINTESTINAL MOTILITY

AFTER PARTIAL GASTRECTOMY.

GASTROINTESTINAL MOTILITY AFTER PARTIAL GASTRECTOMY.

Various opinions have been held on the relationship of changes in the rate of passage of food through the alimentary tract after gastrectomy to "dumping" symptoms. This part of the thesis details an investigation of the effect of partial gastric resection on bowel motility with particular reference to the "dumping" syndrome. This study has been done in collaboration with Dr. N.J.McKellar, Radiologist, Western Infirmary, Glasgow.

MATERIAL AND METHODS.

A study has been made of thirty-five patients who had been treated previously by either a partial gastrectomy (Polya in twenty-nine cases and Billroth I in two), or gastrojejunostomy with vagotomy (four patients). Controls were provided by the use of non-flocculating barium suspension in routine radiological screening lists and five normal cases were given the Raybar-glucose meal described below.

After an overnight fast patients were given an opaque meal of non-flocculating barium sulphate microsuspension (50 per cent W/V: Raybar - Damancy & Co.). This material was used in preference to ordinary barium sulphate to allow clear visualisation of the small bowel pattern without clumping. The two types of Raybar meal were given - 200 ml. Raybar suspension and 150 ml. Raybar with 75 g. glucose (total volume 200 ml.). Initially both meals were given to all patients after operation but it was soon found that /

/that Raybar alone gave small bowel appearances no different from those in patients without operation and so the main part of the study was done with only the Raybar-glucose meal. In addition, eight patients were given 150 ml. Raybar with 50 ml. added water (to give the same dilution effect as in the Raybar-glucose meal), the mixture being cooled to 5-10°C. before ingestion.

With the exceptions noted below screening was carried out in the erect position and films taken at 5, 10, 20, 30, 45 and 90 minutes. Films were not necessarily taken at all these times but only if a significant change had occurred in the appearances. The exposures were frequently made with the patient recumbent as this gave better quality definition; the upright position was resumed immediately after the exposure.

Five patients were studied throughout in the horizontal position. The Raybar-glucose mixture was introduced through a transnasal intrajejunal tube in two patients and through a feeding jejunostomy in a further two cases. The fifth patient took the meal by mouth.

ASSESSMENT OF THE APPEARANCE OF THE SMALL BOWEL.

The rate of transit of the opaque medium through the intestine was assessed by the time of entry into the caecum. The pattern of the small bowel following the ingestion of Raybar in the postoperative groups of patients was compared with that in the normal subjects. If the pattern was similar, the postoperative patient /

/patient was classified as having "normal small bowel appearances". A loss of definition was common in the postoperative patients following the Raybar-glucose meal and this was assessed as +, ++ or +++ according to the extent of the change. In some instances, it was difficult to estimate the degree of abnormality, but there was never any difficulty in recognising that loss of definition was present. This can be, at best, only a subjective impression and this is borne in mind in the interpretation of the results which have not been submitted to statistical analysis.

ASSESSMENT OF LARGE BOWEL MOTILITY.

The motility of the large bowel after ingestion of Raybar was also estimated in both normal and postoperative patients. This was classified as being normal, medium or rapid. In those patients with "rapid" motility the barium reached the distal descending colon or rectum within 90 minutes after the ingestion of the meal. If the meal reached the transverse colon by 90 minutes the motility was assessed as"medium"; if this segment had not been reached motility was recorded as "normal".

RECORDING OF SYMPTOMS.

Dumping symptoms were said to be present when there was some vasomotor upset following the meal, for example, faintness, dizziness, sweating or palpitations. This was usually accompanied by epigastric fullness and bilious vomiting might occur. Neither of these last two symptoms were assessed as "dumping" symptoms, /

/symptoms, unless they occurred with vasomotor disturbance.

RESULTS.

The five normal patients showed no untoward reaction to the Raybar-glucose mixture and gastric and intestinal motility were the same as with Raybar alone.

Full details of the findings following the ingestion of the Raybar-glucose meal in thirty-five patients after operation are recorded in Volume II. A general description of these findings with representative figures only will be presented here.

Gastric emptying was rapid in the majority of the twenty-nine patients studied after Polya gastrectomy and took 10 minutes or less in twenty-two patients. The rate of gastric emptying in those patients with "dumping" symptoms did not differ from that of those in whom no such symptoms were present (Table XII). In three patients with symptoms, gastric emptying was initially fast but ceased with the onset of "dumping" when about half of the Raybar was still in the stomach (Fig. 4).

Intestinal motility was increased in most cases studied (Table XIII). An example is illustrated in Figure 5. This hypermotility was very noticeable on screening, the bowel writhing and twisting as the opaque medium was passed rapidly on to the caecum and the large bowel. Hypermotility of small gut was found in some patients who had no "dumping" symptoms (Fig. 6) but there was only one occasion on which there were symptoms without /

PATIENTS	GASTRIC EMPTYING			TOTAL
	0-10 min.	11-30 min.	31-60 min.	
DUMPING SYMPTOMS	9	1	3	13
NO DUMPING SYMPTOMS	12	2	2	16
TOTAL	21	3	5	29

TABLE XII.

GASTRIC EMPTYING FOLLOWING THE INGESTION OF 200 ml. RAYBAR-GLUCOSE MIXTURE IN 29 PATIENTS AFTER POLYA GASTRECTOMY.

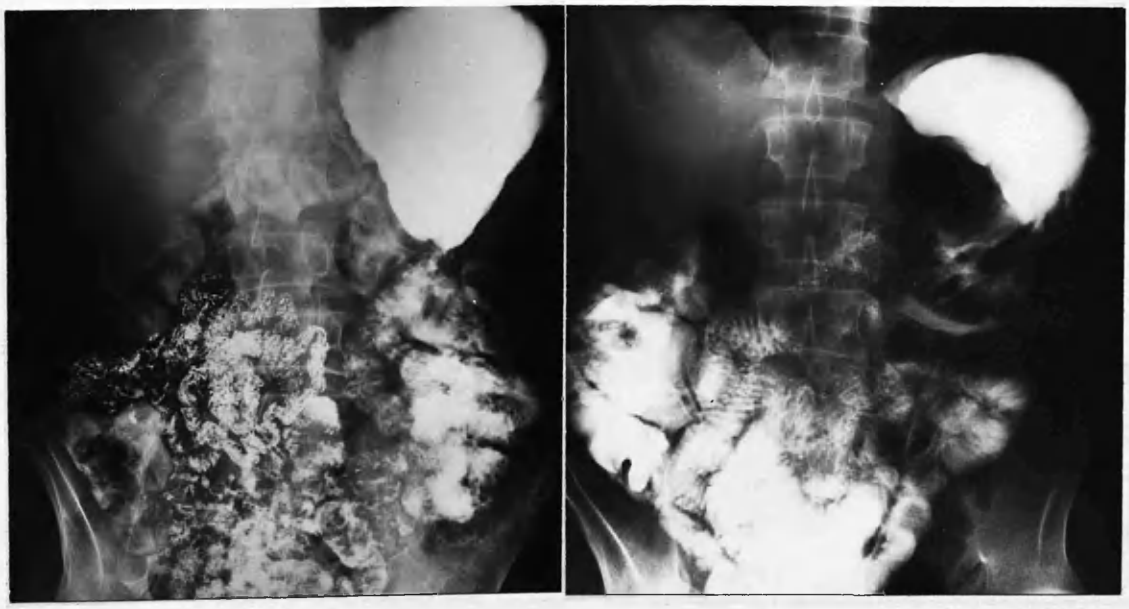


FIG. 4. A

PATIENT 67 AFTER POLYA GASTRECTOMY. RADIOGRAPH 90 MINUTES AFTER THE INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING CESSATION OF GASTRIC EMPTYING WITH STILL ABOUT HALF THE MEAL IN THE STOMACH. "DUMPING" SYMPTOMS WERE PRESENT.

B

PATIENT 17 AFTER POLYA GASTRECTOMY. RADIOGRAPH 15 MINUTES AFTER THE INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING HYPERMOTILITY WITH RAYBAR AT THE HEPATIC FLEXURE AND STILL ABOUT HALF THE MEAL IN THE GASTRIC REMNANT. "DUMPING" SYMPTOMS WERE PRESENT.

	SMALL BOWEL MOTILITY TIME TO REACH CAECUM (MIN.)					TOTAL
	0-10	11-20	21-30	31-60	OVER 60	
DUMPING SYMPTOMS	2	7	3	1	0	13
NO DUMPING SYMPTOMS	1	5	3	1	6	16
TOTAL	3	12	6	2	6	29

TABLE XIII.

SMALL BOWEL MOTILITY FOLLOWING THE INGESTION OF 200 ml. RAYBAR-GLUCOSE MIXTURE IN 29 PATIENTS AFTER POLYA GASTRECTOMY.

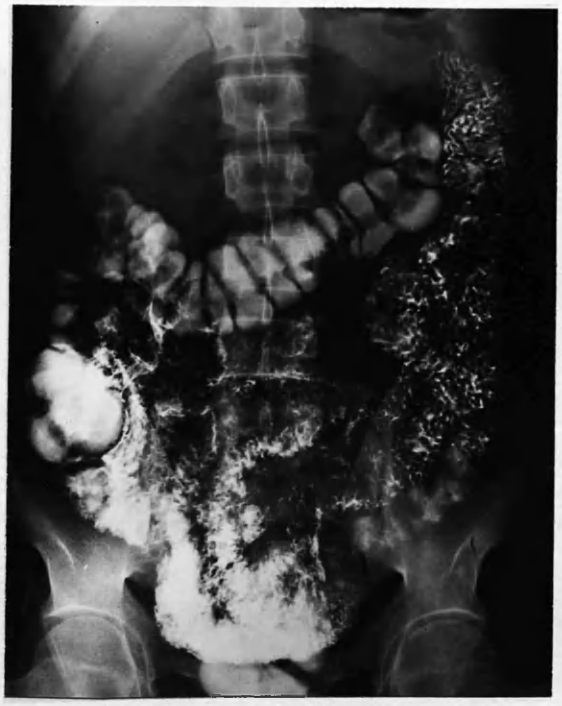


FIG. 5. PATIENT 22 AFTER POLYA GASTRECTOMY. RADIOGRAPH 60 MINUTES AFTER THE INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING HYPERMOTILITY WITH RAYBAR IN THE RECTUM. "DUMPING" SYMPTOMS OCCURRED.

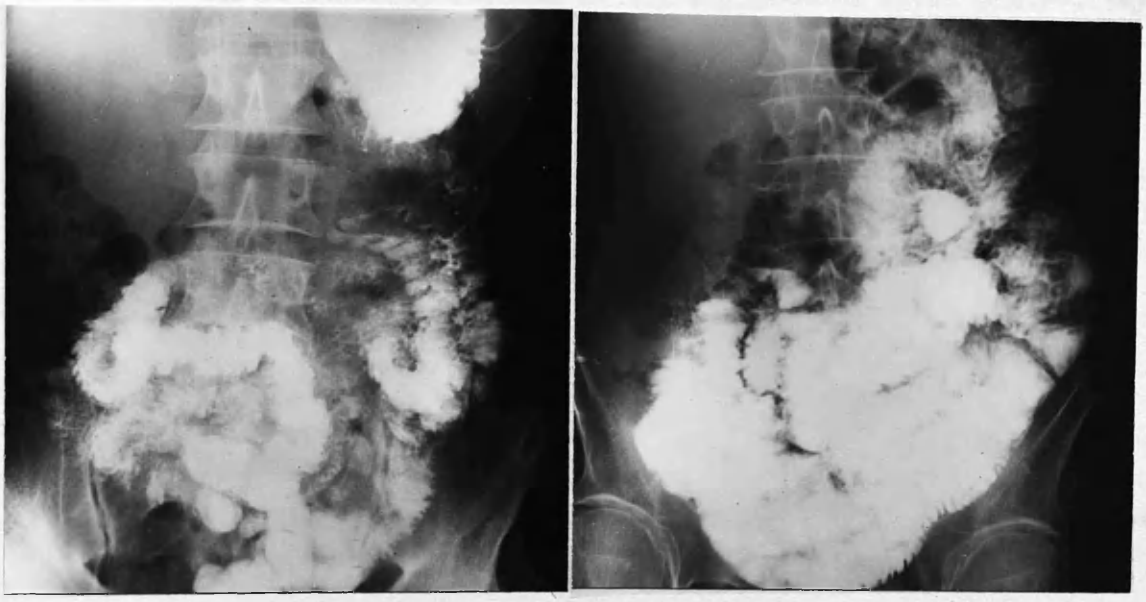


FIG. 6. A

PATIENT 5 AFTER POLYA GASTRECTOMY. RADIOGRAPH 10 MINUTES AFTER THE INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING THE LOSS OF DEFINITION IN THE SMALL BOWEL WHICH WAS ASSESSED AS +++. "DUMPING" SYMPTOMS OCCURRED.

B

PATIENT 13 AFTER POLYA GASTRECTOMY. RADIOGRAPH 10 MINUTES AFTER THE INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING THE LOSS OF DEFINITION IN THE SMALL BOWEL WHICH WAS ASSESSED AT +++. NO "DUMPING" SYMPTOMS OCCURRED.

/without hypermotility. In patients without symptoms hypermotility tended to pass off by the time the caecum was reached, while in the patients with "dumping" attacks it persisted in the large intestine (Table XIV). Only one patient with no symptoms had rapid motility in the large bowel.

An outstanding feature of the appearances after gastrectomy was the lack of definition of the small bowel, especially the ileum. The outline was blurred and the Raybar poorly concentrated, findings which were more noticeable in the cases with marked hypermotility. Loss of detail of small bowel accompanied by hypermotility in two patients, only one of whom had "dumping" symptoms is seen in Figure 6; Figure 7 shows the different appearances following a meal of Raybar alone and following one of Raybar-glucose. Comparison of the time taken for the meal to reach the caecum and the degree of loss of definition (Table XV) indicated that poor definition of the small bowel was associated with increased motility. The apparent correlation was further studied by administering the cooled Raybar to eight patients. In three patients the cooled meal stimulated hypermotility of the small bowel and there was also lack of definition of the radiological pattern. An example is shown in Figure 8 where it is compared with the effect of the Raybar-glucose mixture in the same patient. This patient had typical "dumping" with the Raybar-glucose and only slight symptoms with the cooled meal. The other five of the eight patients given the cooled /

	LARGE BOWEL MOTILITY			TOTAL
	NORMAL	MEDIUM	RAPID	
DUMPING SYMPTOMS	2	5	6	13
NO DUMPING SYMPTOMS	4	5	1	10*
TOTAL	6	10	7	23*

*In the remaining six cases after Polya gastrectomy the Raybar did not reach the caecum in the period of study.

TABLE XIV.

LARGE BOWEL MOTILITY FOLLOWING THE INGESTION OF 200 ml. RAYBAR-GLUCOSE MIXTURE IN 23 PATIENTS AFTER POLYA GASTRECTOMY.

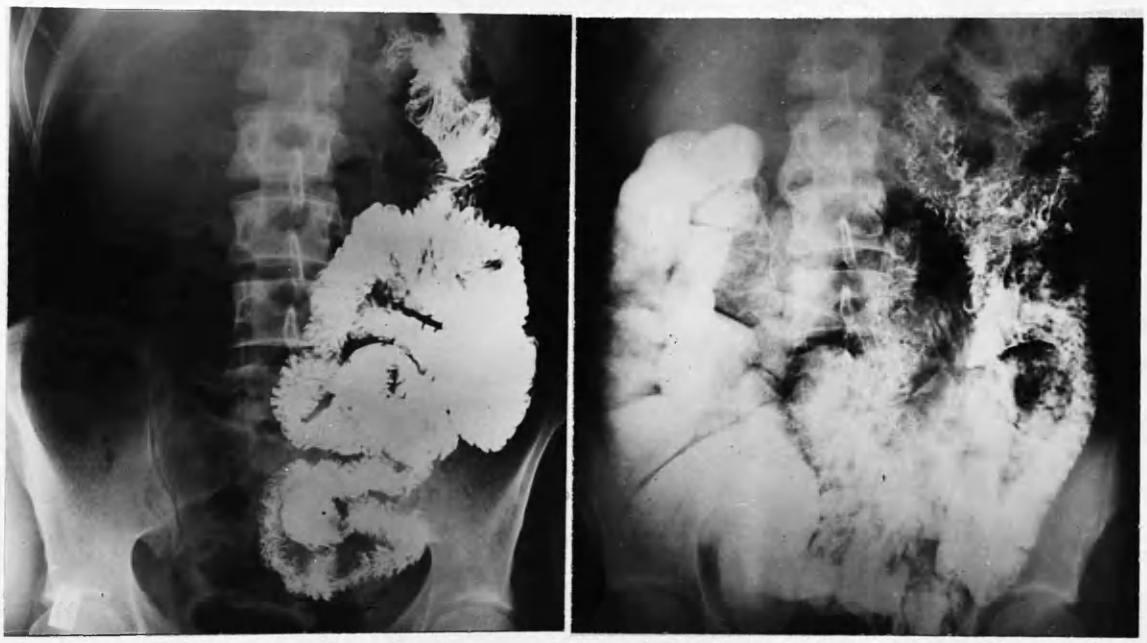


FIG. 7. A

PATIENT 29 AFTER POLYA GASTRECROMY. RADIOGRAPH 90 MINUTES AFTER THE INGESTION OF 200 ml. RAYBAR SHOWING THE MAIN MASS OF THE MEAL IN THE PROXIMAL SMALL BOWEL WITH A NORMAL SMALL BOWEL PATTERN. NO "DUMPING" SYMPTOMS OCCURRED.

B

RADIOGRAPH 90 MINUTES AFTER THE INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING INCREASED MOTILITY COMPARED WITH (A), THE MEAL BEING IN THE TRANSVERSE COLON AND SOME LOSS OF DEFINITION OF THE SMALL BOWEL.

LOSS OF DEFINITION IN THE SMALL BOWEL	SMALL BOWEL MOTILITY TIME TO REACH CAECUM (MIN.) 0-11	11-20	21-30	OVER 30	TOTAL NO. OF PATIENTS
+	0	0	0	2	2
++	0	2	6	4	12
+++	3	12	0	6	21
TOTAL	3	14	6	12	35

TABLE XV.

COMPARISON OF THE LOSS OF DEFINITION AND THE MOTILITY OF THE SMALL BOWEL FOLLOWING THE INGESTION OF 200 ml. RAYBAR-GLUCOSE MIXTURE IN 35 PATIENTS AFTER OPERATION.

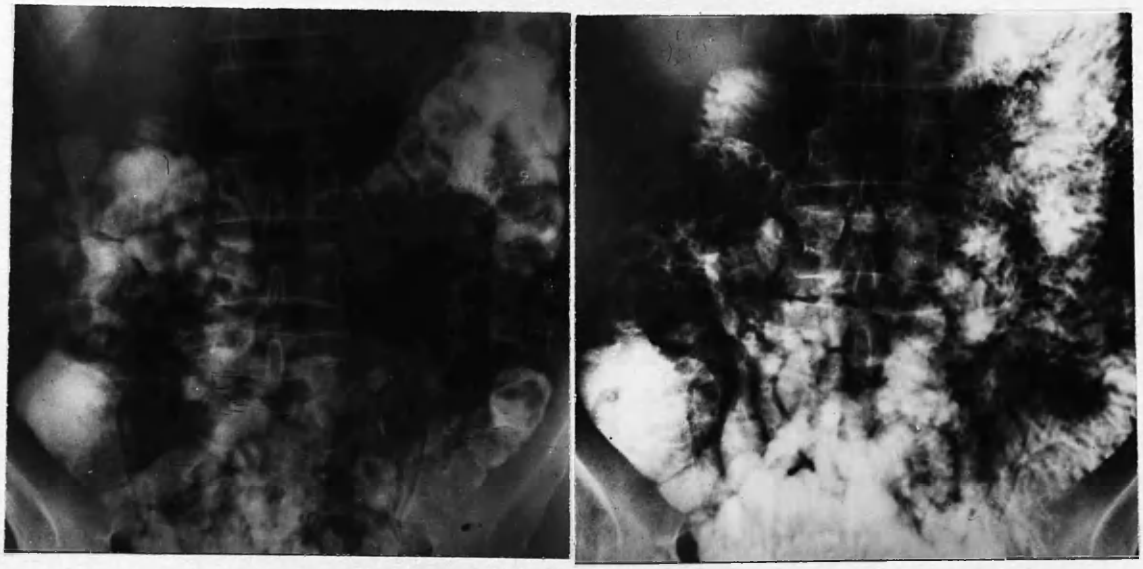


FIG. 8. A

PATIENT 66 AFTER POLYA GASTRECTOMY. RADIOGRAPH 20 MINUTES AFTER INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING HYPERMOTILITY (RAYBAR IN DESCENDING COLON) AND +++ LOSS OF SMALL BOWEL DEFINITION.

B

RADIOGRAPH 20 MINUTES AFTER INGESTION OF 200 ml. COOLED RAYBAR SHOWING HYPERMOTILITY (RAYBAR IN CAECUM) AND +++ LOSS OF SMALL BOWEL DEFINITION.

/cooled Raybar had normal motility and a normal small bowel pattern (Table XVI).

Fluid levels were often seen in the small bowel when films were taken with the patient in the upright position (Fig. 9). In a few cases the proximal jejunum had a wider lumen than usual (Fig. 10) though there was no marked jejunal distension. Both these findings were equally common in patients with and without "dumping" symptoms. No areas of spasm were observed in the bowel.

Experiments in which Raybar-glucose was given with the patients in the recumbent posture showed that this position did not abolish the hypermotility. In two patients in whom the Raybar-glucose meal was given by means of a transnasal intrajejunal tube and in one of the two patients in whom it was introduced into a jejunostomy, increased motor activity of the bowel was noted. The other patient with a jejunostomy showed no hypermotility. The remaining patient who took the meal by mouth in a recumbent position is worthy of description in detail. His stomach remnant emptied slowly in the first 10 minutes although the Raybar which reached the jejunum was quickly passed on (Fig. 11a). At this time he was turned prone and the gastric stump emptied in the next 10 minutes. The small bowel was very active (Fig. 11b), but no symptoms occurred although this patient had been found previously to have attacks of "dumping" which commenced 15 to 20 minutes after eating. When 30 minutes had elapsed from /

PATIENT	SMALL BOWEL	
	LOSS OF DEFINITION	MOTILITY
66	+++	20 MIN. TO CAECUM
77	+++	13 MIN. TO CAECUM
79	+++	30 MIN. TO CAECUM
64	NORMAL APPEARANCE	40 MIN. TO JEJUNUM
70	" "	60 MIN. TO ILEUM
80	" "	45 MIN. TO JEJUNUM
81	" "	60 MIN. TO ILEUM
82	" "	45 MIN. TO JEJUNUM

TABLE XVI.

COMPARISON OF THE APPEARANCE AND THE MOTILITY OF THE SMALL BOWEL FOLLOWING THE INGESTION OF 200 ml. COOLED RAYBAR IN 8 PATIENTS AFTER POLYA GASTRECTOMY.

FIG. 9. PATIENT 69 AFTER POLYA GASTRECTOMY. RADIOGRAPH 30 MINUTES AFTER INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING FLUID LEVELS IN SMALL BOWEL. NO "DUMPING" SYMPTOMS OCCURRED.

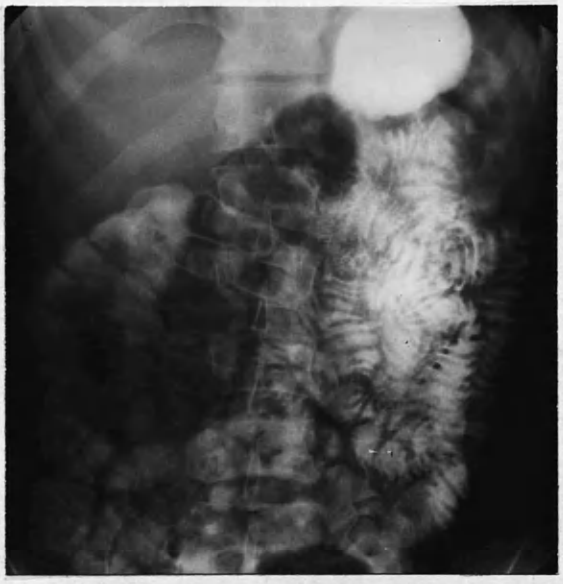


FIG. 10. PATIENT 39 AFTER POLYA GASTRECTOMY. RADIOGRAPH 30 MINUTES AFTER INGESTION OF 200 ml. RAYBAR-GLUCOSE SHOWING INCREASE IN WIDTH OF JEJUNUM ALONG WITH HYPERMOTILITY AND LOSS OF SMALL BOWEL DETAIL.

FIG. 11. A

PATIENT 25 AFTER POLYA GASTRECTOMY FOLLOWING INGESTION OF 200 ml. RAYBAR-GLUCOSE WHEN RECUMBENT. RADIOGRAPH AT 10 MINUTES SHOWING MOST OF MEAL IN GASTRIC REMNANT BUT WHAT HAD ENTERED JEJUNUM WAS WELL ROUND INTO ILEUM.

B

RADIOGRAPH AT 20 MINUTES (10 MINUTES AFTER TURNING PRONE) SHOWING EMPTY STOMACH AND MEAL AT CAECUM.

/from the ingestion of the Raybar-glucose he stood upright and had typical "dumping" symptoms within 3 minutes. Further evidence that lying down does not inhibit hypermotility of the small bowel was provided by its persistence in patients who developed "dumping" symptoms following the ingestion of a meal of Raybar-glucose in the erect posture and who required recumbency for relief of symptoms.

The occurrence of "dumping" symptoms after gastrojejunostomy with vagotomy did not relate to the pattern of bowel motility. The two patients who were studied after Billroth I gastrectomy had rather slower gastric and intestinal motility than was usual after Polya gastrectomy.

DISCUSSION.

The importance of rapid emptying of the gastric remnant in contributing to "dumping" symptoms has been stressed since the time of Mix (1922). In the present study rapid emptying of the stomach was found with equal frequency in those patients with and without symptoms and three patients with symptoms had relatively slow emptying. The cessation of gastric emptying with the onset of symptoms in three patients would support the contention of Capper and Butler (1951) that stretching of the gastric stump is an important factor in producing "dumping" were it not for the fact that in these three patients hypermotility was also present. In addition, other patients had "dumping" attacks with no evidence of "hold-up" in the gastric remnant. /

/remnant.

It has also been suggested that hypermotility of the small intestine is a causative factor in the production of "dumping" symptoms (Glazebrook and Welbourn, 1952). The results of this study indicate that greatly increased bowel activity (even to the extent of the opaque medium reaching the caecum in 10 minutes) can occur in patients without symptoms. On the other hand, hypermotility was invariably noted in the patients after gastrectomy who had "dumping" although one patient with a gastrojejunostomy and vagotomy had a typical attack with no sustained hypermotility. Hypermotility of the large bowel was seen almost exclusively in patients with "dumping" symptoms.

Further evidence that hypermotility does not necessarily give rise to symptoms is found in the studies done in the recumbent posture. In four of five patients increased motor activity of the bowel occurred without symptoms while in the fifth patient motility was normal. When the upright position was assumed "dumping" occurred in the one patient who had been previously subject to attacks, suggesting that hypermotility per se cannot produce symptoms even in the susceptible patient.

The osmotic effect of a hypertonic meal in the lumen of the gut has been considered the cause of "dumping" symptoms (Roberts et al., 1954; Amdrup and Jørgensen, 1956c, 1957). This osmotic effect was believed to induce dilution of bowel contents with blurring of mucosal pattern and the presence of fluid levels when an opaque meal containing glucose was ingested. Blurring /

/Blurring of the pattern occurred only in patients who had "dumping" attacks (Amdrup et al., 1958). In the present investigation, the administration of a Raybar-glucose meal was followed by a lack of definition and the presence of fluid levels in an equal proportion of patients with and without "dumping" symptoms. A similar incidence of blurring (believed to indicate dilution of bowel contents) was also observed by Pulvertaft (1953) in both groups of patients.

The association of lack of definition of the small bowel and hypermotility is shown by the results following the ingestion of the Raybar-glucose meal. Additional support comes from the studies with the cooled Raybar, for with this material blurring of the small bowel pattern occurs only when hypermotility is present. These two groups of findings suggest that an osmotic factor is not the main cause of the loss of detail in the small bowel. In addition, it must be remembered that the cooled Raybar had no glucose added and so the blurred appearances resulted from a meal of "normal" tonicity. It might be argued that fluid was, in fact, attracted into the small bowel not by osmotic pressure but as a result of the low temperature of the meal. That this is unlikely is shown by the normal appearances in the four patients who had no hypermotility and in whom the meal remained in the proximal small bowel with no distension or loss of definition. These results indicate that the loss of definition of the small bowel pattern may well be due to dispersion of the meal and to /

/to failure of absorption of fluid resulting from hypermotility rather than to addition of secretions from the bowel wall.

CONCLUSIONS.

Rapid gastric emptying and hypermotility of the small bowel occur after the ingestion of a Raybar-glucose meal independently of the occurrence of "dumping" symptoms.

Hypermotility of the small intestine continuing into the large intestine is more common in patients who have "dumping" symptoms than in those who have none.

The hypermotility of the small bowel is not abolished by lying down and the upright posture is essential before the hypermotility is associated with "dumping" symptoms.

A lack of definition of the small bowel is seen as often in patients without "dumping" attacks as in those affected after operation. This poor definition is associated with hypermotility of the small bowel.

CARDIOVASCULAR CHANGES FOLLOWING A MEAL

IN PATIENTS AFTER GASTRECTOMY.

CARDIOVASCULAR CHANGES FOLLOWING A MEAL IN PATIENTS AFTER PARTIAL GASTRECTOMY.

The follow-up study of 132 patients has emphasised the importance of the vasomotor symptoms of the "dumping" syndrome after gastrectomy. In this section a study of the alterations in the cardiovascular responses to a meal after operation with particular emphasis on a possible relationship to vasomotor symptoms is reported.

MATERIAL.

Fifty-five patients between the ages of 35 and 65 years were studied, the majority being 40 to 50 years old. A total of ninety-six studies are reported, fifteen on patients before operation, sixty-four after Polya gastrectomy, eight after Billroth I gastrectomy and nine after gastrojejunostomy with vagotomy. Control tests were done on a further twenty patients who were in hospital for some minor complaint, e.g. an inguinal hernia. The postoperative experiments were carried out at an interval varying from 8 weeks to 10 years after operation.

METHODS.

Patients were studied in the sitting position as recumbency is well known to alleviate "dumping" symptoms. Subjects had been fasting and at rest in bed for 12 hours and were allowed 30 minutes /

/minutes to settle after assuming the sitting posture. They sat in an upright chair which was comfortable enough to prevent restlessness at the end of 2 hours. Active movement was forbidden as this is known to cause tachycardia and if excessive, a decrease in plasma volume (Kaltreider and Meneely, 1940; Collumbine and Koch, 1949). Change of posture may also have a similar effect (Hallock and Evans, 1941). No patient whose haemoglobin (Hb - alkaline haematin method of Gibson and Harrison, 1945) was lower than 12.32 g. per cent was admitted to the study.

During the experiment, the pulse rate, blood pressure, plasma volume, packed cell volume, blood sugar and electrocardiograms were measured at frequent intervals during the hour before and the hour after a test meal of 100 ml. 50 per cent glucose. It has frequently been observed that sweet foods are liable to produce dumping attacks in susceptible patients after gastrectomy, and hypertonic glucose has been previously used as a method of inducing "dumping" experimentally, although the dose is usually larger than that which we have used (150 ml. 50 per cent glucose - Roberts et al., 1954; Amdrup and Jørgensen, 1957; Everson and Abrams, 1958). The smaller dose used by us was selected to provide a more delicate test and one which would only produce symptoms in patients who were regularly troubled by "dumping" symptoms.

Several other types of test meals were also given to a number of patients. These were 100 ml. 50 per cent fructose /

/fructose (three patients) 100 ml. 50 per cent suspension of mannitol (mannose-sugar - a six carbon polyhydric alcohol slowly absorbed from the gut and poorly utilised - two patients) and a meal of 120 g. protein (one patient). The meals were usually taken by mouth in the sitting position, but in five patients studied in recumbency the meal was given by transnasal intrajejunal tube.

In eight tests 1 mg. adrenaline hydrochloride was administered subcutaneously as a substitute for the meal.

During the two hours of the test any symptoms were recorded on a similar plan to that used in the follow-up study. A diagnosis of "dumping" was made only when vasomotor upset was present, and epigastric fullness alone was not regarded as significant evidence of this syndrome. The severity of the attacks were assessed clinically, and were recorded as "severe" when the symptoms were sufficiently marked completely to incapacitate the patient. Definite but less marked vasomotor symptoms were assessed as moderate.

METHODS OF ESTIMATION.

Packed cell volume. Heparinised blood was centrifuged in a haematocrit tube at 3,000 r.p.m. for 30 minutes.

Plasma volume. In the main human serum albumen labelled with radioactive iodine (131_I) (RHSA) was used to estimate plasma volume by the dilution principle. Additional studies were made using Evans Blue dye (EB).

RADIOIODINATED HUMAN SERUM ALBUMEN (RHSA)*

Approximately 10 microcuries of RHSA in 9 ml. were injected intravenously from a calibrated syringe and the plasma volume was estimated by the deviation from the control disappearance slope. To prevent uptake of any free ^{131}I (maximum 2 per cent of radioiodine content of RHSA) by the thyroid gland 45 minins of Lugol's Iodine were given daily for two days before the test. Specimens were taken at 15-minute intervals from the antecubital vein with the arm at heart level and with the minimal venous occlusion. Clotting was prevented with heparin. Plasma was obtained by centrifugation and 2 ml. samples were estimated for radioactivity in a well type scintillation counter with an automatic scaler (Ecko N350A). A standard 1 in 1,000 dilution of the RHSA dose was counted simultaneously. The readings were done in duplicate and an average taken. Individual readings did not vary by more than 2 per cent. The log. average values in counts per millilitre were plotted against time and their deviation from the regression line was expressed as a percentage. It has been shown by Pritchard and others (1955) that following the intravenous injection of RHSA there is a constant disappearance of radioactivity during the first 2 hours. In the method used a continuous estimate of radioactivity of the peripheral blood /

* Specific activity not less than 5 microcuries per g. protein, obtained from The Radiochenical Centre, Amersham.

/blood was made by directing its flow through a scintillation counter. To determine if a similar relationship was shown when intermittent samples of plasma were counted, twenty normal patients were studied. The results are shown in Table XVII.

On a semi-logarithmic scale the disappearance curve approximates closely to a straight line (Fig. 12). Readings more than twice the standard deviation from the mean were taken as significant. Typical examples of disappearance slopes following the ingestion of 100 ml. 50 per cent glucose before and after operation are shown in Figure 13. The control value for plasma volume was found by extrapolation of the disappearance slope to zero time.

$$\text{Plasma Volume} = \frac{\text{Radioactivity of Dose Given}}{\text{Radioactivity of Plasma per ml. at zero time}}$$

The accuracy of this method in five patients was estimated at 2 per cent (Abrams et al., 1957).

EVANS BLUE DYE (EB).

The Evans Blue dye was given in repeated doses of 5 to 10 ml. of a 0.1 per cent solution into an arm vein from a burette as described by Plentl and Gelfand (1954) and illustrated in Fig. 14. This method allows the administration of frequent accurate doses of EB and the washing through of the tubing with 5 per cent dextrose ensures that all the dye reaches the circulation. Plasma samples were obtained as above exactly /

MINUTES AFTER INJECTION OF RHSA	PER CENT DEVIATION FROM CONSTANT DISAPPEARANCE RATE OF RADIOACTIVITY (MEAN OF 20 CONTROLS)
15	-0.77 ± 1.84*
30	+0.20 ± 1.37*
45	+0.43 ± 0.86*
60	+0.33 ± 1.36*
75	-0.06 ± 1.16*
90	-0.72 ± 0.86*
105	-0.48 ± 0.98*

* S.D.

TABLE XVII.

MEAN PER CENT DEVIATION FROM A CONSTANT DISPPEARANCE RATE OF RADIOACTIVITY FROM THE PLASMA FOLLOWING AN INJECTION OF RHSA IN 20 CONTROL PATIENTS.

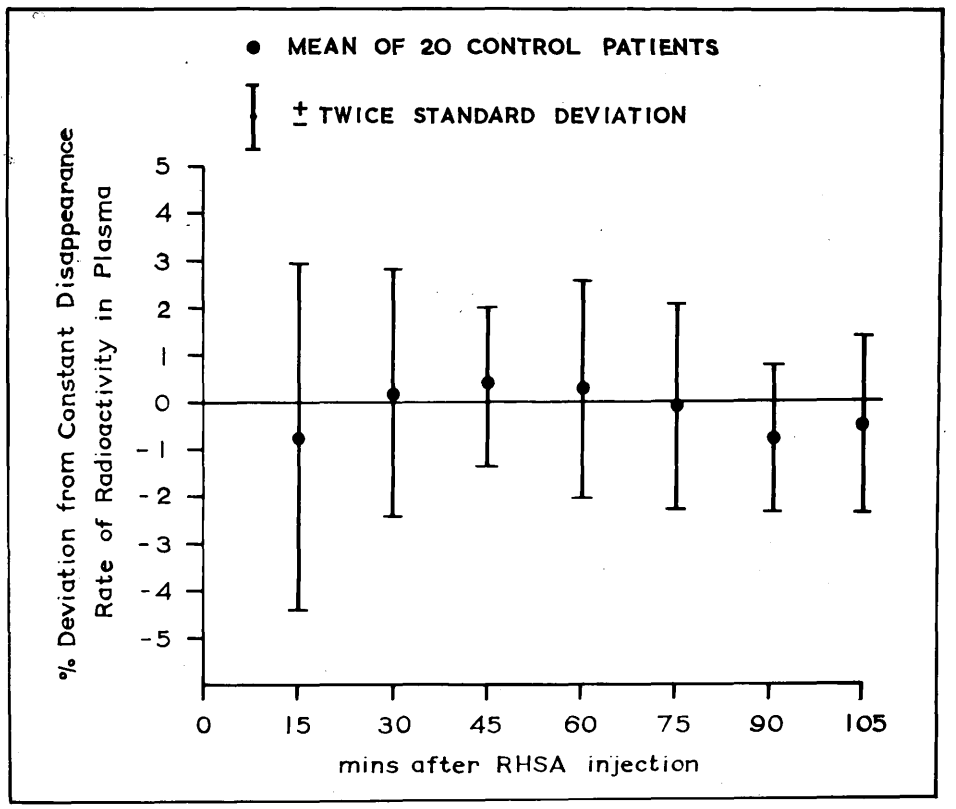


FIG. 12. MEAN PER CENT DEVIATION FROM A CONSTANT DISAPPEARANCE RATE OF RADIOACTIVITY FROM PLASMA FOLLOWING AN INJECTION OF RHSA IN 20 CONTROL PATIENTS.

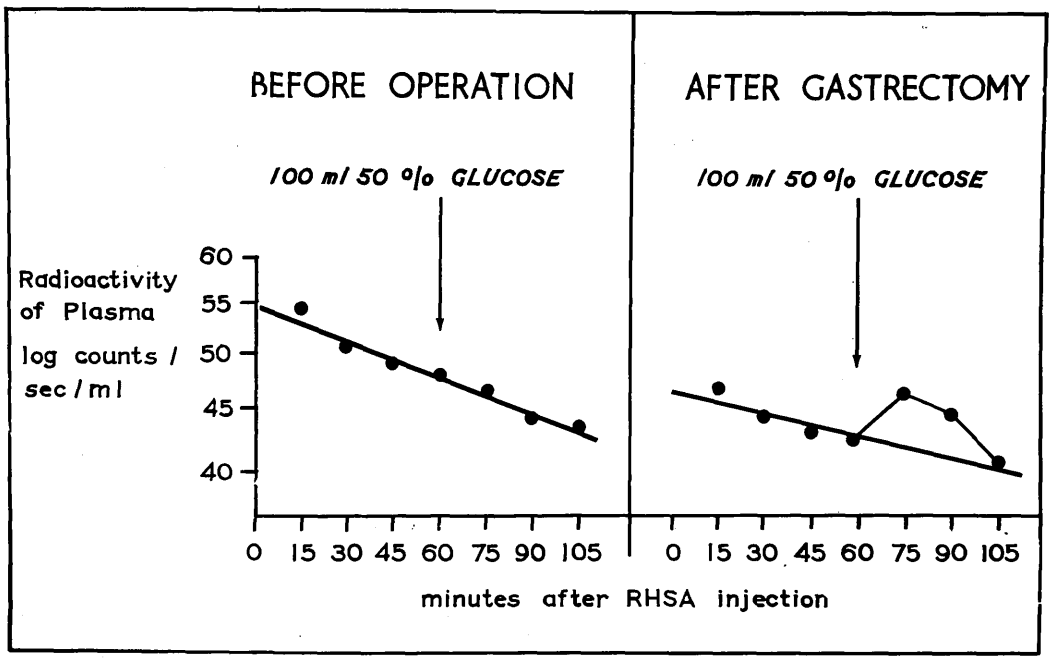


FIG. 13. DISAPPEARANCE SLOPES OF RADIOIODINATED HUMAN SERUM ALBUMEN FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE BEFORE AND AFTER OPERATION (PATIENT 3).

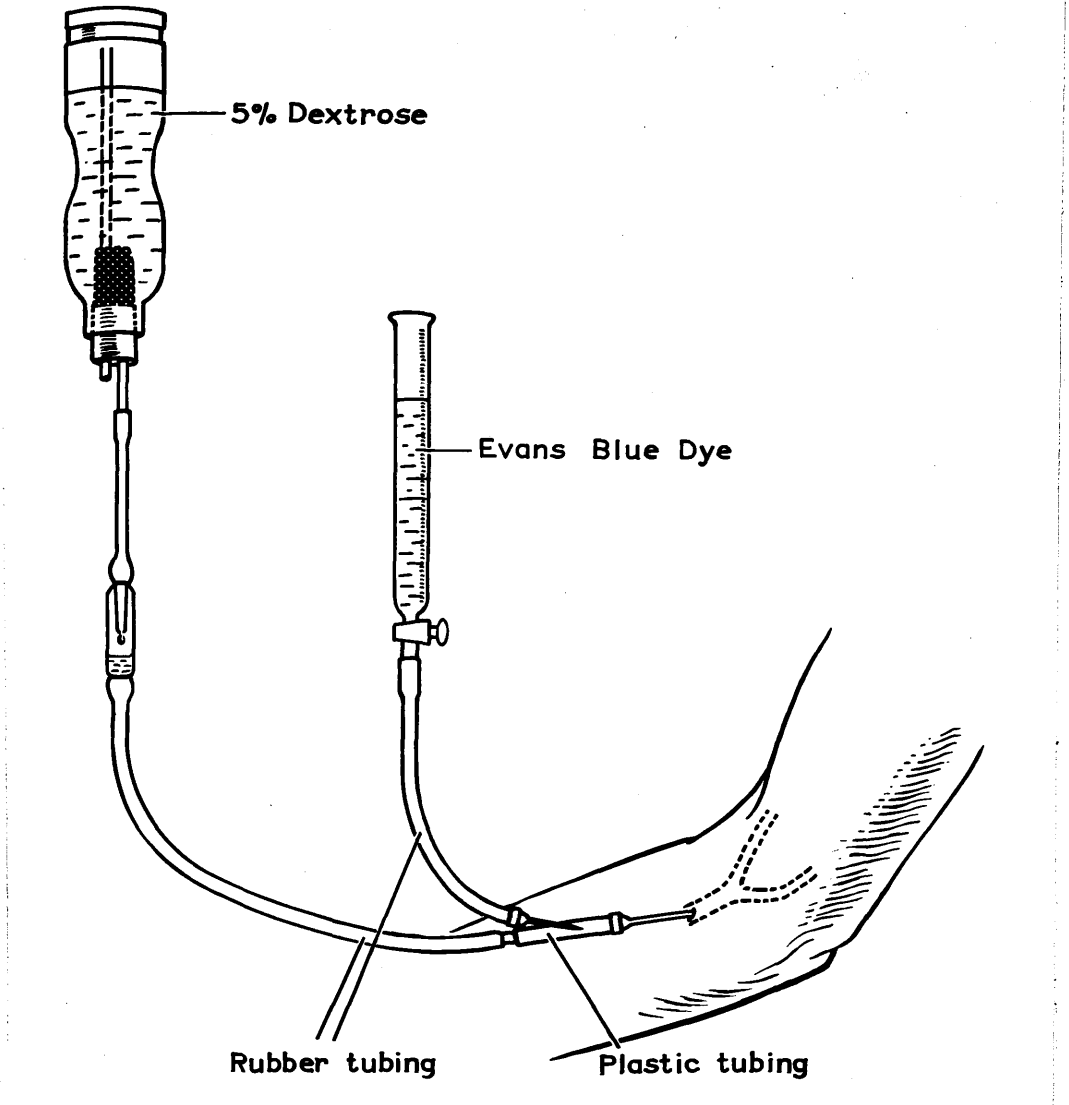


FIG. 14.

APPARATUS FOR GIVING REPEATED DOSES OF EVANS BLUE DYE.

/exactly 15 minutes after each injection. The EB in each specimen was estimated by the method of Bedwell and his co-workers (1955). Samples of 2 ml. plasma are run through a column of Celite (Johns Manville, No. 545) supporting a layer of freshly precipitated cellulose acetate approximately 0.5 cm. in depth. The dye is adsorbed on the cellulose and the column is washed with a solution of 1 in 200 Teepol (Shell Chemicals Ltd., Grade 530) in physiological saline until no protein is present in the washings tested with 20 per cent salicylsulphonic acid. The EB is then eluted with 5 to 6 ml. 35 per cent acetone and the volume made up to 7 ml. before reading in a spectrophotometer (Unicam SP 350). Adsorption and elution are carried out at a temperature of 45°C. The maximal absorption for EB in 35 per cent acetone was found to be at 604 millimicrons. In our hands the recovery rate was 92.8 per cent (S.E.Mean ± 0.25) in 20 controls. This was not as high as that of the original authors. At each determination a further recovery was done using 20 micrograms EB and allowance was made for the loss in the calculation of the plasma volume. A single determination took approximately 1½ hours and no more than three columns could be run in parallel. Plasma volume was obtained from the formula

$$\text{Plasma Volume} = \frac{\text{O.D.EB standard} \times \text{Dose of EB given}}{\text{O.D. EB adsorbed from plasma} \times \frac{\text{Amount}}{\text{EB standard}}}$$

POSSIBLE SOURCES OF ERROR.

FACTORS AFFECTING PLASMA VOLUME ESTIMATION.

Apart from the usual experimental error in measurements, for instance in pipetting, there are other factors which might influence the accuracy of the above methods as estimates of the true plasma volume.

THE RELATIONSHIP OF THE "SPACE" MEASURED TO THE PLASMA VOLUME.

Both the above methods of estimating the plasma volume depend on marking the albumen fraction of the plasma proteins, either with RHSA or with EB, and then measuring the concentration of the marker. This assumes that there is no undue loss out of the circulation through the capillary walls and that the slow removal of the marker from the blood stream is a function of the normal protein metabolism. Any leak of circulating albumen would result in an erroneously high value for the plasma volume. Several authors have noted that the estimation of blood volume by methods which mark the serum albumen result in a reading 10 - 40 per cent higher than that obtained by methods labelling the red blood cells. In a full review of the subject von Porat (1951) suggests that this discrepancy may be due to variations in the PCV (from trapped plasma or from the difference between the peripheral and the whole body haematocrit) and to escape /

/escape of albumen from the circulation. This latter factor has also been emphasised by Finnerty et al., (1957) who believe that the space measured by EB is partly extra-vascular. These authors make no mention of PCV errors. Attention is drawn to the escape of albumen from the circulation over a period of several hours by Thistlethwaite and his colleagues (1957).

The use of a control disappearance curve, as in the present study with RHSA, will allow for escape of albumen and, though the reading may give too large an estimate of the absolute plasma volume, the relative measurements will be comparable. The results from the repeated doses of EB will also bear comparison as long as the rate of leakage does not change between doses. The occurrence of a "blocking" effect on the reticuloendothelial system by the first dose of EB was shown in the cat by Cruickshank and Whitfield (1945) and the rate of leakage of the albumen marked with EB was less in the subsequent doses. This has been shown not to apply to man. The subject is reviewed by von Porat (1951) and Plentl and Gelfand (1954) provide further evidence supporting this conclusion.

HAEMOLYSIS AND PLASMA TURBIDITY.

These factors are frequently troublesome in any method in which the optical density of plasma is measured. Care in obtaining specimens was taken to avoid haemolysis. Using Evan's Blue, errors from both haemolysis and plasma turbidity are obviated by the adsorption method. Estimations of optical density are not required in the RHSA method and neither factor is likely to cause error. However, in either method, marked haemolysis would cause inaccuracy by dilution of the plasma and any specimen with haemolysis visible to the naked eye was discarded.

FACTORS AFFECTING THE PCV.

It is well known that the peripheral venous haematocrit does not give a true sample of the proportion of red cells to plasma in the whole body (Chaplin et al., 1953) and is affected by the amount of plasma trapped in the column of red cells (Chaplin and Mollison, 1952). Correction factors for these differences are available though they show some variation from author to author. Rather than introduce this possible error by converting plasma volume to total blood volume, the plasma volumes themselves were used in this study. The PCV results are considered separately.

SIGNIFICANCE OF APPARENT CHANGE IN PLASMA VOLUME.

As will be seen in the results, the main change noted /

/noted was a decrease in the plasma volume resulting from ingestion of a glucose meal. Using the RHSA disappearance method, this decrease implies that the concentration of radioactive albumen has increased relative to the expected disappearance slope. This occurs if there is a loss of fluid but no albumen from the circulation. Stasis of blood alone would not influence the disappearance curve unless there was also fluid loss. On the other hand, pooling might be detected by the EB method using intermittent doses, as the later doses of the dye might not mix with the blood in the areas of sluggish circulation. Under these circumstances, the concentration of EB in the circulating plasma would be greater than with the earlier injections and the measured plasma volume would decrease. When both methods are used simultaneously, an agreement in the results would suggest that there was no stasis, while a greater fall with the EB method than that with RHSA would be compatible with pooling of blood.

RESULTS.

The results given here are mean values. Full details of the experiments and their correlation with the other findings following the test substances are to be found in Volume II. The changes in pulse rate, blood pressure and plasma volume are reported first followed by the PCV results. Electrocardiographic and blood sugar results are discussed in later sections.

RESPONSE TO THE GLUCOSE MEAL.

BEFORE OPERATION.

In eleven patients studied before operation, the ingestion of 100 ml. 50 per cent glucose gave rise to a slight increase in plasma volume. All were studied again after operation (which was a gastrectomy in eight and a gastrojejunostomy with vagotomy in three patients) when the changes were greater (Table XVIII). In two patients "dumping" symptoms occurred after operation although no preoperative response could be correlated with this finding.

AFTER POLYA GASTRECTOMY.

Forty patients who were tested after gastrectomy had a marked tachycardia with a fall in plasma volume after the glucose meal. (Table XIX). The changes in plasma volume at 15 and 30 minutes were significantly different from those in identical tests in the control patients (Table XX). No difference was observed between the twenty pairs who had "dumping" attacks and the other twenty who had no upset (Table XXI). Typical examples of the results before and after operation are shown in Figure 15. A fall in plasma volume of 10 per cent or more was found in eight patients with "dumping" and in five of the patients without "dumping". This difference is not significant ($\chi^2 = 0.11$ d.f. = 1, $P > 0.9$). The /

ELEVEN PATIENTS	MAXIMAL RISE IN PULSE RATE BEATS/MIN.	MAXIMAL RISE IN PULSE RATE PER CENT	BLOOD PRESSURE	MAXIMAL FALL IN PLASMA VOLUME PER CENT
BEFORE OPERATION	6.5±1.30*	8.7±1.79*	NO CHANGE 11	2.8±0.68*
AFTER OPERATION	16.7±2.93*	25.1±4.38*	RISE 4 NO CHANGE 6 FALL 1	6.7±0.70*
t	3.04	2.90		3.72
P	< 0.01	< 0.01		< 0.01

* S.E.MEAN

TABLE XVIII.

MEAN CARDIOVASCULAR CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 11 PATIENTS STUDIED BEFORE AND AFTER OPERATION.

PATIENTS	MAXIMAL RISE IN PULSE RATE		BLOOD PRESSURE	MAXIMAL FALL IN IN PLASMA VOLUME PER CENT
	BEATS/MIN.	PER CENT		
PRE-OP. 11	6.5±1.30*	8.7±1.79*	NO CHANGE 11	2.8±0.68*
POLYA GASTRECTOMY 40	20.8±1.79*	30.3±2.57*	RISE 17 NO CHANGE 18 FALL 5	7.4±0.55*
COMPARED WITH 11 PRE-OP.				
t	4.03	4.25	-	3.98
P	< 0.001	< 0.001	-	< 0.001
GASTROJEJUN-OSTOMY AND VAGOTOMY 8	13.7±1.52*	20.7±2.64*	RISE 3 NO CHANGE 5	6.4±1.12*
COMPARED WITH 11 PRE-OP.				
t	3.43	3.69	-	2.67
P	< 0.01	< 0.01	-	< 0.02

* S.E.MEAN

TABLE XIX.

MEAN CARDIOVASCULAR CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 11 PATIENTS BEFORE OPERATION, 40 PATIENTS AFTER POLYA GASTRECTOMY AND 8 PATIENTS AFTER GASTROJEJUNOSTOMY WITH VAGOTOMY.

PATIENTS	NO.	MEAN CHANGE IN PLASMA VOLUME PER CENT MIN. AFTER 100 ml. 50 PER CENT GLUCOSE 15	30	45
PRE-OP	11	-2.3±1.01*	-1.8±0.68*	0.1±0.0*
POLYA GASTRECTOMY	40	-6.3±0.54*	-5.9±0.62*	-2.4±0.63*
t		3.07	2.93	1.64
P		< 0.01	< 0.01	> 0.1

* S.E. MEAN

TABLE XX.

MEAN CHANGE IN PLASMA VOLUME FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 11 PATIENTS BEFORE OPERATION AND 40 PATIENTS AFTER POLYA GASTRECTOMY.

"DUMPING" SYMPTOMS	RISE IN PULSE RATE PER CENT	BLOOD PRESSURE		MAXIMAL FALL IN PLASMA VOLUME PER CENT	
PRESENT 20 PATIENTS	31	RISE NO CHANGE FALL	9 8 3	8.3±0.82*	t = 1.23
ABSENT 20 PATIENTS	30	RISE NO CHANGE FALL	8 10 2	7.0±0.63*	P > 0.2

* S.E.MEAN

TABLE XXI.

MEAN CARDIOVASCULAR CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 40 PATIENTS AFTER POLYA GASTRECTOMY, COMPARING 20 WHO HAD "DUMPING" ATTACKS WITH 20 WHO HAD NO SYMPTOMS.

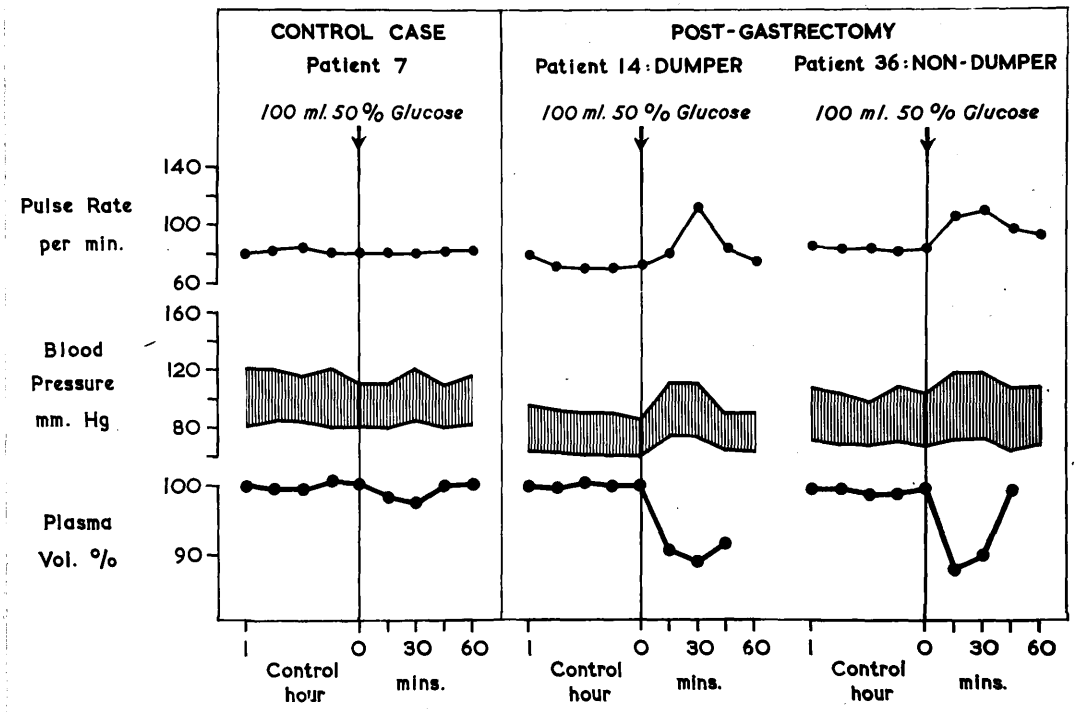


FIG. 15. CARDIOVASCULAR CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE BEFORE OPERATION AND AFTER POLYA GASTRECTOMY WITH AND WITHOUT "DUMPING" SYMPTOMS.

/The eighteen patients with a rise in blood pressure following glucose had a greater fall of plasma volume than those in whom the blood pressure was unaltered (Table XXII). The increase in pulse rate was most marked in the thirteen patients with a fall in plasma volume greater than 10 per cent (Table XXIII). Both these differences are significant.

A similar degree of response was obtained when tests were repeated on the same patient. An example is shown in Volume II, pages 24 and 25.

AFTER CONVERSION FROM POLYA TO BILLROTH I GASTRECTOMY.

This further procedure was performed in seven patients, five of whom were suffering from "dumping" symptoms and two from bilious vomiting. Comparison of the tests before and after the operation (Table XXIV) shows that there was little change in response. This is illustrated in Figure 16. However, three of the five patients with "dumping" symptoms benefitted by having less trouble after normal meals and one was cured of bilious vomiting.

AFTER GASTROJEJUNOSTOMY WITH VAGOTOMY.

Similar, but less well marked, changes were observed in eight patients who were studied after this operation (a rise in pulse rate, a slight rise in blood pressure in three cases and a fall in plasma volume which was not significant in two patients). The mean values are shown in Table XIX. Four /

BLOOD PRESSURE	NO. OF PATIENTS	MEAN FALL IN PLASMA VOLUME PER CENT
RISE	17	-9.2 ± 1.15*
NO CHANGE	18	-6.2 ± 0.91*
t	2.40	
P	< 0.05	

* S.E. MEAN

TABLE XXII.

MEAN FALL IN PLASMA VOLUME FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN PATIENTS AFTER POLYA GASTRECTOMY COMPARING PATIENTS WITH AND WITHOUT A RISE IN BLOOD PRESSURE.

FALL IN PLASMA VOLUME	NO. OF PATIENTS	RISE IN PULSE RATE PER CENT
10 PER CENT OR MORE	13	42 ± 1.05*
LESS THAN 10 PER CENT	27	24 ± 2.85*
t	6.04	
P	< 0.001	

* S.E. MEAN

TABLE XXIII.

MEAN RISE IN PULSE RATE FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN PATIENTS AFTER POLYA GASTRECTOMY COMPARING 13 WITH A FALL IN PLASMA VOLUME OF 10 PER CENT OR MORE AND 27 WITH A FALL OF LESS THEN 10 PER CENT.

PATIENT		PULSE RATE		BLOOD PRESSURE		FALL IN PLASMA VOLUME PER CENT	"DUMPING" SYMPTOMS
		BEFORE GLUCOSE	AFTER GLUCOSE	BEFORE GLUCOSE	AFTER GLUCOSE		
14	P	72	112	85/ 60	110/ 75	11	Severe
	BI	68	148	90/ 60	110/ 70	19	Severe
15	P	84	120	105/ 75	120/ 80	8	Severe
	BI	88	126	115/ 85	145/ 90	5	Severe
22	P	64	100	135/100	160/105	17	Moderate
	BI	68	76	110/ 75	130/ 85	15	Moderate
44	P	84	102	140/105	90/ 60	6	Moderate
	BI	78	100	115/ 80	85/ 65	11	Moderate
45	P	88	106	95/ 65	85/ 55	12	Moderate
	BI	76	80	95/ 70	85/ 60	10	Moderate
34	P	86	120	105/ 70	120/ 75	11	Bilious vomiting.
	BI	96	130	90/ 75	140/ 90	6	Nil
41	P	60	68	120/ 75	135/ 80	7	Bilious vomiting.
	BI	60	78	110/ 65	125/ 80	6	Nil

P = Polya gastrectomy BI = Billroth I gastrectomy

TABLE XXIV.

CARDIOVASCULAR CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 7 PATIENTS COMPARING THE RESULTS AFTER POLYA GASTRECTOMY WITH THOSE AFTER CONVERSION TO BILLROTH I GASTRECTOMY

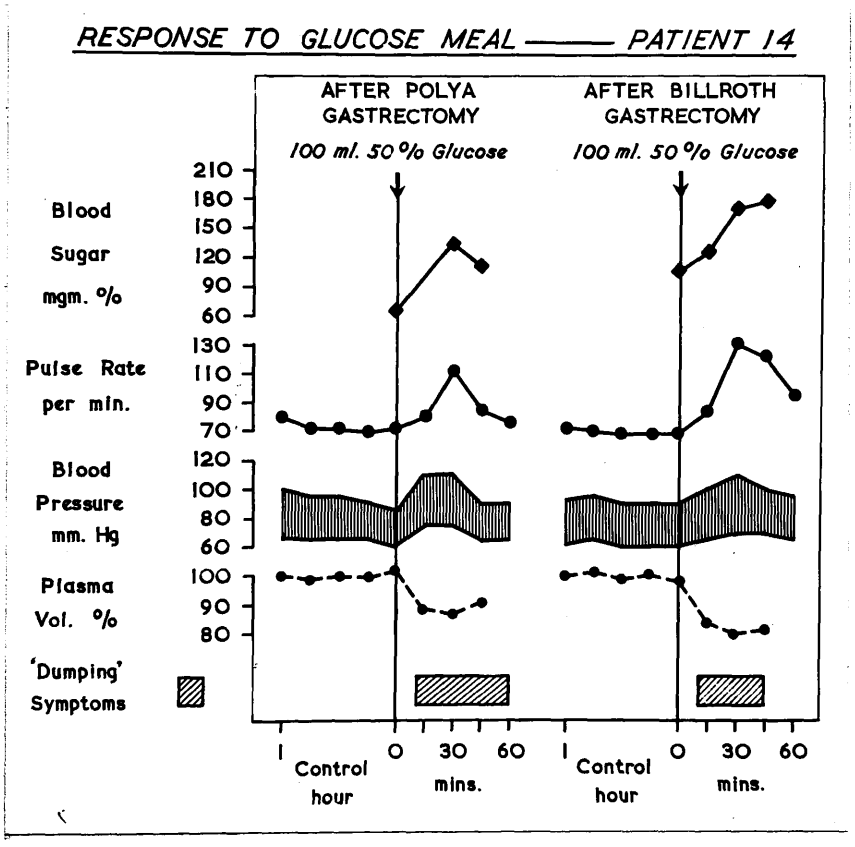


FIG. 16. CARDIOVASCULAR CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE AND AFTER CONVERSION FROM POLYA TO BILLROTH I GASTRECTOMY.

/Four patients with moderate "dumping" attacks had a response which was no different from the other four.

RESPONSES TO OTHER MEALS.

FRUCTOSE.

In patient No. 10 the response after a meal of fructose was the same as after glucose both as regards symptoms and cardiovascular changes. Another patient (No. 18) had similar symptoms after both meals but the cardiovascular changes were much more marked after glucose than with fructose. The last patient of this sub-group (No. 51) had symptoms with fructose and not with glucose, yet the cardiovascular changes were greater with the glucose. These findings further confirm the lack of correlation of the symptoms with other aspects of the response.

MANNITOL.

This meal was given to two patients after gastrectomy and both had symptoms and a cardiovascular response which was greater than that after glucose.

PROTEIN.

"Dumping" symptoms occurred in the patient (No. 35) to whom this meal was given. He gave a history of being regularly upset by a meal of minced meat and this produced an attack during the experiment. It also induced cardiovascular /

/cardiovascular alterations which were less in degree than after the glucose meal.

ADDITIONAL EXPERIMENTS.

EFFECT OF ADRENALINE.

The subcutaneous injection of 1 mg. adrenaline hydrochloride (1 in 1,000 solution) in four normal patients caused a tachycardia and a rise in blood pressure. Two patients had a significant fall in plasma volume. The same dose given to four patients after operation (Polya gastrectomy in three, gastrojejunostomy in one) resulted in changes similar to those which they had experienced after the standard glucose meal (Fig. 17). All eight patients given adrenaline had symptoms similar to vasomotor symptoms of the "dumping" symptoms.

REPEATED DETERMINATIONS OF PLASMA VOLUME WITH EVANS BLUE.

In an effort to detect any pooling of blood within the cardiovascular system, the plasma volume after the glucose meal was measured by repeated doses of EB. In two cases this was done as a separate experiment and in six concurrently with an RHSA determination. The mean fall in plasma volume was slightly greater with the EB (7.1 per cent as against 6.8 per cent with RHSA). In two patients with "dumping" symptoms the shift observed by the EB method was much greater than that when /

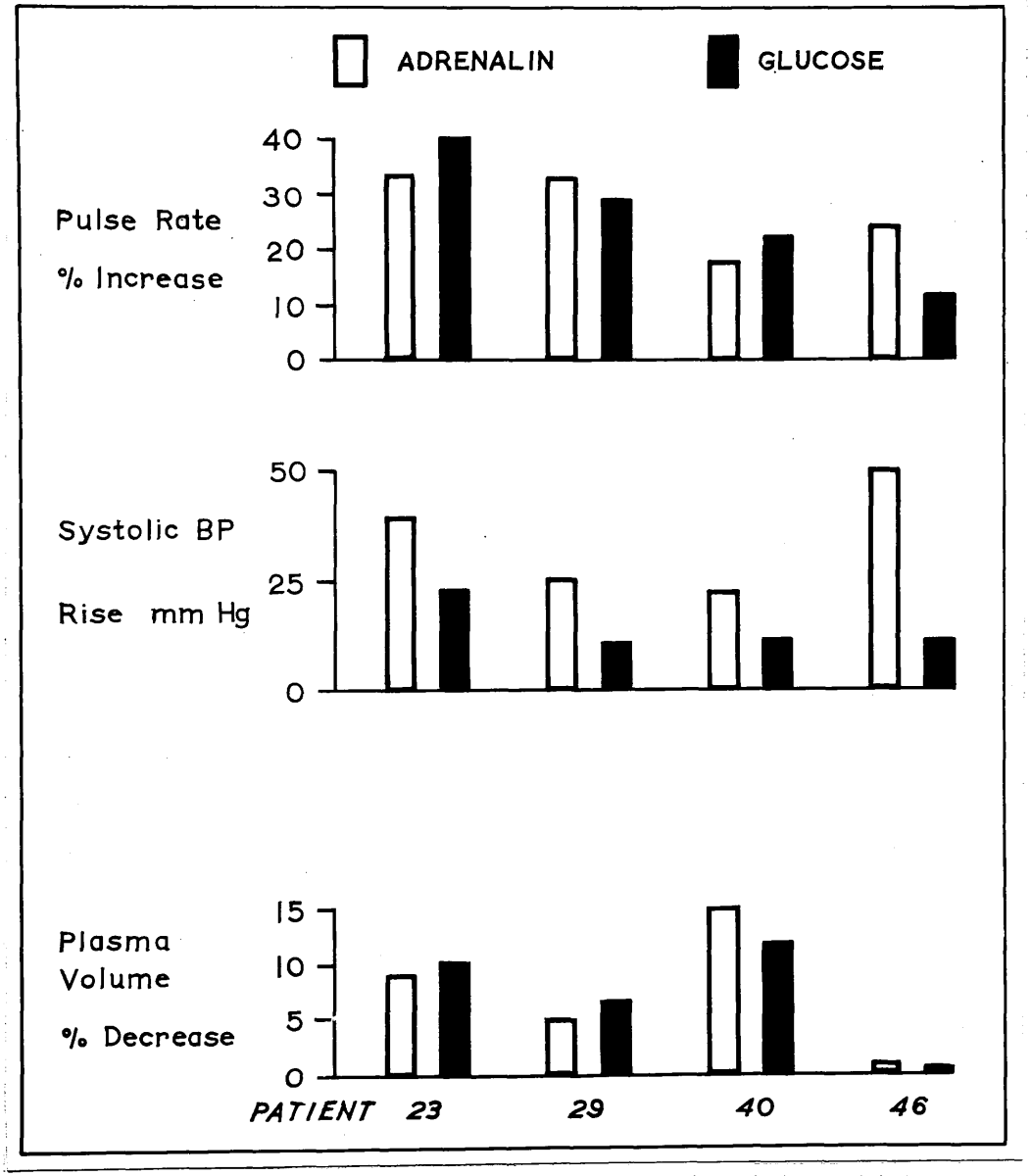


FIG. 17. COMPARISON OF CARDIOVASCULAR CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE AND THE SUBCUTANEOUS INJECTION OF 1 mg. ADRENALINE HYDROCHLORIDE IN 4 PATIENTS AFTER OPERATION.

/when RHSA was used (Fig. 18).

EFFECT OF POSTURE ON THE CARDIOVASCULAR CHANGES AFTER A GLUCOSE MEAL.

In five patients after gastrectomy the experiment with the glucose meal was repeated with the subject recumbent. The glucose was given by a transnasal intrajejunal tube to avoid delay in gastric emptying, which was blamed for a diminished response by Amdrup and Jørgensen (1956b). The cardiovascular responses were much less in each case than when the same meal was given in the sitting position. Plasma volume changes are shown in Figure 19.

EFFECT OF INTRAVENOUS INFUSION OF PLASMA.

Six patients who had symptoms after the glucose meal were given a further test in which 500 ml. plasma or 6 per cent Dextran in physiological saline were infused rapidly in 3 - 4 minutes, just before the time that the symptoms had begun on the previous occasion. Three patients felt that they had less severe symptoms after the infusion than after glucose alone but in the others symptoms were unchanged. Repeated estimations with EB in two cases showed that the fall in plasma volume was compensated by the infusion.

CHANGES IN PACKED CELL VOLUME (PCV).

In order to assess whether there was pooling of blood /

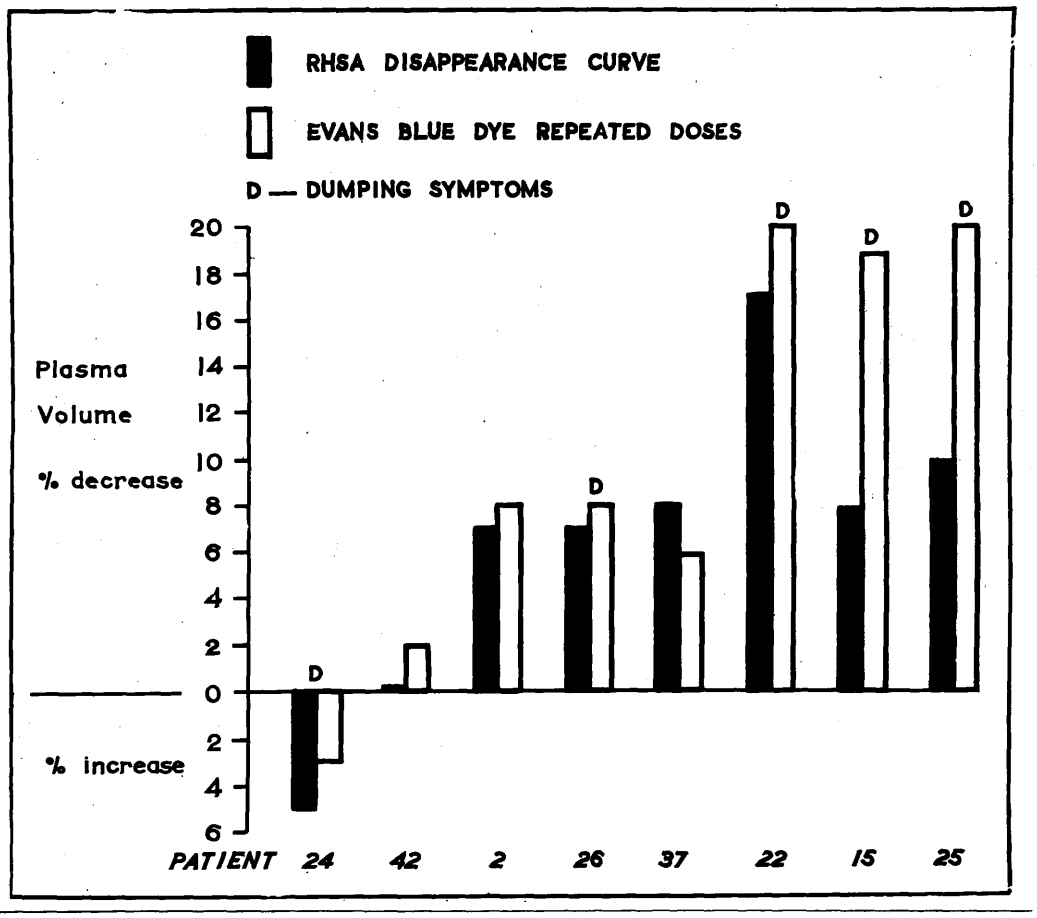


FIG. 18. COMPARISON OF PLASMA VOLUME CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE USING BOTH RADIOIODINATED HUMAN SERUM ALBUMEN (RHSA) AND EVANS BLUE DYE IN 8 PATIENTS AFTER GASTRECTOMY.

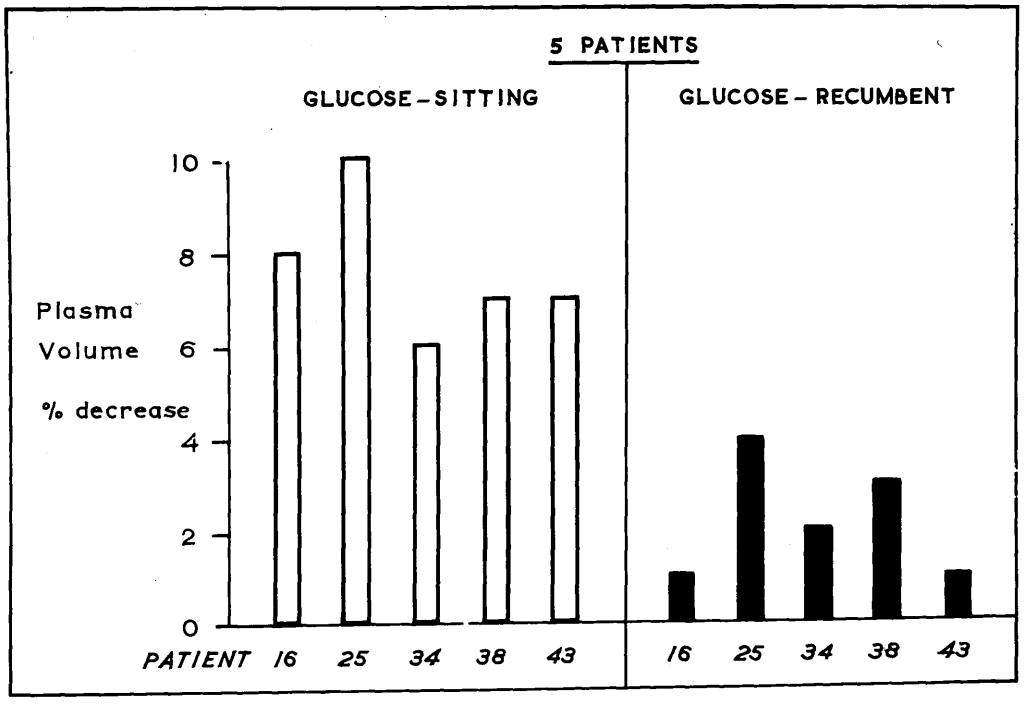


FIG. 19. COMPARISON OF PLASMA VOLUME CHANGES FOLLOWING 100 ml. 50 PER CENT IN THE LYING AND SITTING POSITIONS IN 5 PATIENTS AFTER GASTRECTOMY.

/blood in any part of the circulation, a comparison was made between the recorded PCV and that calculated from the control PCV and the change in plasma volume. It was assumed that the red cell mass would not change in the 2 hours of the experiment. With this assumption, the calculated PCV was obtained from the formula:

$$PCV_x = \frac{100\ PCV_c}{K\ (100 - PCV_c) + PCV_c}$$

Where PCV_x is the packed cell volume at time x after the meal.

PCV_c is the packed cell volume in the control period.

K is the ratio of the plasma volume at time x to be control plasma volume.

The derivation of this formula is shown in Volume II, page 100. The mean difference between the two values should be zero if the amount of red blood cells in the peripheral blood remained constant during the test. In eleven patients before operation and in eighteen patients after gastrectomy who had no "dumping" symptoms, the readings were not significantly different from zero, but in the seventeen patients after gastrectomy who had "dumping" symptoms, the recorded PCV at 15 and 30 minutes after the glucose meal were significantly less than the calculated value (Table XXV). These findings suggest that some red cells are being trapped in patients with symptoms, perhaps by pooling of blood of insufficient extent to be detected in other than two /

MEAN DIFFERENCE OF MEASURED AND CALCULATED PCV	PATIENTS		
	11 PREOP.	18 POST-GASTRECTOMY NO "DUMPING"	17 POST-GASTRECTOMY "DUMPING"
15 MIN. AFTER GLUCOSE	-0.27±0.27*	-0.19±0.26*	-0.65±0.23*
t	0.99	0.75	2.92
P	>0.3	>0.4	< 0.02
30 MIN. AFTER GLUCOSE	+0.17±0.42*	-0.06±0.25*	-0.72±0.32*
t	0.40	0.24	2.28
P	>0.6	>0.8	< 0.05
45 MIN. AFTER GLUCOSE	-0.14±0.20*	+0.53±0.31*	-0.32±0.30*
t	0.71	1.75	1.06
P	>0.5	>0.05	> 0.3

* S.E.MEAN

TABLE XXV.

MEAN DIFFERENCES OF THE MEASURED PCV AND THE CALCULATED PCV FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 11 PATIENTS PREOPERATIVELY, 18 PATIENTS AFTER GASTRECTOMY HAVING NO "DUMPING" SYMPTOMS AND 17 PATIENTS AFTER GASTRECTOMY WHO HAVE "DUMPING" ATTACKS.

/two patients by intermittent EB method for plasma volume estimation. All patients had a variability in PCV readings after a glucose meal which may be an index of circulatory changes.

It has been suggested that repeated sampling of blood per se causes a fall in the haematocrit (Bonnycastle, 1947; Huber, 1947). Allen and Semple (1951) found that, if allowance was made for the volume of blood withdrawn, no significant difference in packed cell volume occurred. This latter suggestion is confirmed in the two groups showing no change in the present series. As the same volume of blood was withdrawn in each case, the fall in haematocrit in those patients with "dumping" symptoms cannot be explained by blood loss alone. It would appear that in these patients there is a deviation of some red cells from the peripheral circulation.

DISCUSSION.

The results of this section have shown that patients after gastrectomy show an exaggerated response to a glucose meal compared with subjects before operation. This is characterised by a greater rise in pulse rate and an obvious fall in plasma volume. The blood pressure changes are variable. This response is common to all patients after surgery and is not specifically related to the presence of "dumping" symptoms.

Conflicting opinions have been held on the role of the fall in plasma volume in the production of these symptoms. The /

/The suggestion that a diminution of plasma volume was primarily responsible was advanced by Roberts et al., (1954) and by Amdrup and Jørgensen (1957). The prevention of symptoms by the infusion of 500 ml. plasma expander supported this hypothesis (Walker et al., 1955). Other workers have found no direct relationship between the degree of diminution in plasma volume and the occurrence of symptoms (Hinshaw et al., 1957: Everson and Abrams, 1958). The present study indicates that a decrease in plasma volume is not always associated with "dumping" symptoms but is rather the usual response to which some patients appear to be unduly sensitive. This can be seen both from the finding of large decreases in plasma volume when no symptoms are present and by the failure of the infusion of plasma expander to prevent symptoms.

The importance of hypotension in the aetiology of the vasomotor symptoms has also been emphasised by Roberts et al., (1954). In the series reported here, the blood pressure was more often slightly raised in the presence of "dumping" symptoms. Thus, the faintness and dizziness are not necessarily due to hypotension. It was observed that the largest fall in blood pressure did occur in patients with severe symptoms and this may indicate a failure of compensatory mechanisms.

The fall in plasma volume, and hence in circulating blood volume, has been ascribed to loss of fluid into the bowel lumen due to the osmotic pressure of the meal (Roberts et al., 1954: /

/Amdrup and Jørgensen, 1956c: Peddie et al., 1957). The additional studies with fructose and mannitol confirm that equivalent changes can occur with meals of the same osmotic tension as the glucose, but the fact that a meal of protein can also be followed by plasma volume changes indicates that some other factor must also be responsible.

Assuming, for the moment, that osmotic pressure takes part in the reduction in plasma volume, the observation that lying down diminished the symptoms and the tachycardia is not surprising. It is well known that a subject can tolerate the loss of a pint of blood better when supine. What is surprising is that the decrease in plasma volume should be less with the patient lying than sitting, even though the glucose was introduced into the jejunum. If the osmotic pressure theory be accepted, some other factor must also contribute to the changes in the erect patient. Alteration of posture from lying to standing results in a drop in circulating blood volume (Hallock and Evans, 1941). This is to believed to be due to pooling of blood in the dependent parts of the body with some fluid loss into the tissues. Such pooling of blood may be the additional factor which causes the greater decrease in plasma volume in the seated patient in this study, this effect being neutralised when the body is horizontal. This suggestion is supported by the larger diminution in circulating plasma volume measured by the EB technique when EB and RHSA methods were simultaneously used on the same patient (especially in the two cases who had a marked difference). Further corroborative /

/corroborative evidence is provided by the reduction of PCV estimated by haematocrit compared with that calculated from the plasma volume in the patients with "dumping", indicating a deviation of red cells from the peripheral circulation in the arm.

Another possible effect of the alteration in posture from supine to sitting erect is stimulation of the sympathetic nervous system (Scherf and Schlachman, 1948a). The administration of adrenaline can cause the same response - tachycardia and a fall in plasma volume - as occurs after this postural change and also after the glucose meal. Thus a parallel mechanism may operate. A direct comparison of the results in four patients after operation showed a close similarity of the cardiovascular changes brought about by adrenaline and the glucose meal. A fall in plasma volume took place in three patients and the remaining one had no change. In four control patients who were given adrenaline the response varied. A decrease in plasma volume occurred in two patients, a variation which has been noted previously in normal subjects (Kaltreider and Meneely, 1942). Others have failed to detect a fall greater than the experimental error (Parson et al., 1948). The close correlation of the response to adrenaline with that following the glucose in each case after operation suggests that some overactivity of the sympathetic nervous system also plays a part in the production of the cardiovascular changes.

Under these circumstances, it is not difficult to appreciate that mechanical alteration of the gastrectomy stoma from /

/from gastrojejunal to gastroduodenal would not cause much improvement in the changes following a glucose meal except in so far as the rate of gastric emptying is concerned. Nor would it be expected that division of the gastric branches of the vagus, as in subdiaphragmatic vagotomy, would abolish the responses to a meal which remains only for a short time in the stomach.

The sequence of events which follows the ingestion of a meal of glucose in patients after gastrectomy appears to be a loss of fluid from the circulation with a pooling of blood in some part of the body below the level of the heart. These changes reduce the circulating blood volume. This reduction would be compensated by a presso-receptor reflex, chiefly from the sino-aortic bodies, causing a tachycardia and a rise in cardiac output. In most cases this sympathetic overactivity prevents a fall in blood pressure; although in a few patients it may fail with resulting hypotension.

There is no clear explanation of the occurrence of "dumping" symptoms for no single factor characterises the cardio-vascular changes of the patients affected by symptoms e xcept the difference in PCV readings. It may be that the individuals who are subject to attacks have a more labile autonomic nervous system than normal which over-reacts to the stimulus of a glucose meal in the erect position. Such over-reaction not only maintains blood pressure but also causes side effects of faintness, sweating, pallor, tachycardia and palpitations. These are the clinical features of the "dumping" syndrome.

CONCLUSIONS.

The cardiovascular changes induced by a meal of glucose are more marked in patients after gastrectomy than in those with intact stomachs. These changes consist of a rise in pulse rate and a fall in plasma volume with a variable change in blood pressure.

Cardiovascular changes are similar in patients with and without "dumping" attacks and the conversion of a Polya to a Billroth I gastrectomy does not prevent their occurrence.

The response after gastrojejunostomy with vagotomy is only slightly less than that after gastrectomy, indicating that these changes are not mediated through the gastric branches of the vagus nerves.

The osmotic effect of the meal is not the only factor in the initiation of the cardiovascular changes and some pooling of blood in the body below the level of the heart may contribute to the changes.

Relative overactivity of the sympathetic nervous system plays a part in the aetiology of the cardiovascular changes.

ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL IN PATIENTS AFTER GASTRECTOMY.

ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL IN PATIENTS AFTER GASTRECTOMY.

Changes in the electrocardiogram after a meal in patients who have had gastrectomy have previously been noted (Smith, 1951; Smith et al., 1953; Pulvertaft, 1954; Roberts et al., 1954; and Peddie et al., 1957). It has not been suggested that these changes cause "dumping" symptoms but that they are caused by the same factors as the symptoms. A study has therefore been made of the electrocardiographic changes in order to investigate the mechanism of the "dumping" syndrome. In this section Part I contains a description of the form and incidence of the changes following a glucose meal and their relationship to other cardiovascular changes and to "dumping" symptoms. Part II details investigations into the factors producing the electrocardiographic changes. Detailed illustrations of the electrocardiograms appear in Vol. II and representative examples only are shown here.

It is generally agreed that subjective influences can play a part in the interpretation of electrocardiograms and care has been taken throughout to use the patient as his own control especially in Part II when the effects of other tests were compared with the changes after a glucose meal.

PART I.

THE FORM AND INCIDENCE OF ELECTROCARDIOGRAPHIC CHANGES, MATERIAL AND METHODS.

Twelve patients were studied before operation, thirty-six after Polya gastrectomy, seven after gastrojejunostomy with vagotomy and five after conversion from a Polya to a Billroth I gastrectomy.

As has been described, the electrocardiograms (ECG) were taken during a 2 hour experiment when the other cardiovascular parameters were measured. That is, patients were studied after a 12 hour fast sitting in a chair and there was a control period of one hour before and a test hour after the ingestion of 100 ml. 50 per cent glucose. Some patients only had ECG and blood pressure recordings.

ECGs were taken every few minutes using a Cambridge "Electrite" machine. Standard and augmented limb leads were recorded. After being placed the electrodes were not moved during the 2 hours. The patient remained sitting throughout as postural changes are known to alter the ECG (Myerson and Davis, 1942; Scherf and Schlachman, 1948a; Cameron, 1951). Measurement of the deflections, the time intervals and the QT ratio were by standard methods (Goldberger, 1953). When alterations were consistent in several consecutive complexes a change of 1 mm. or more was assessed as significant except the R and S waves when the lower limit was 2 mm. The /

/The machine was standardised at each tracing to show a deflection of 1 cm. for 1 millivolt.

CONTROL STUDIES.

To ensure that no changes occurred due to minor shifts of the electrodes or due to the development of poor contacts from drying of the electrolytic paste, repeated tracings were taken during the course of 2 hours when no test meal was given. No changes in the ECG were observed.

The ingestion of cold water has given rise to ECG changes (Wilson and Finch, 1923). Five patients were given 200 ml. water at room temperature by mouth. No alteration in the ECG took place. This indicated that any change following the glucose meal which was given at room temperature was not due to a cooling effect.

RESULTS.

FORM OF ECG CHANGES (Fig. 20).

The changes were as follows in order of frequency:-

An increase in heart rate varying from a few beats to 50 beats per minute.

A broadening and a lowering in potential of the T wave which was best seen in leads II, III and aVF and was less frequent and less obvious in lead I. Accompanying this was often a decrease in the negativity of the T wave in lead aVR. /

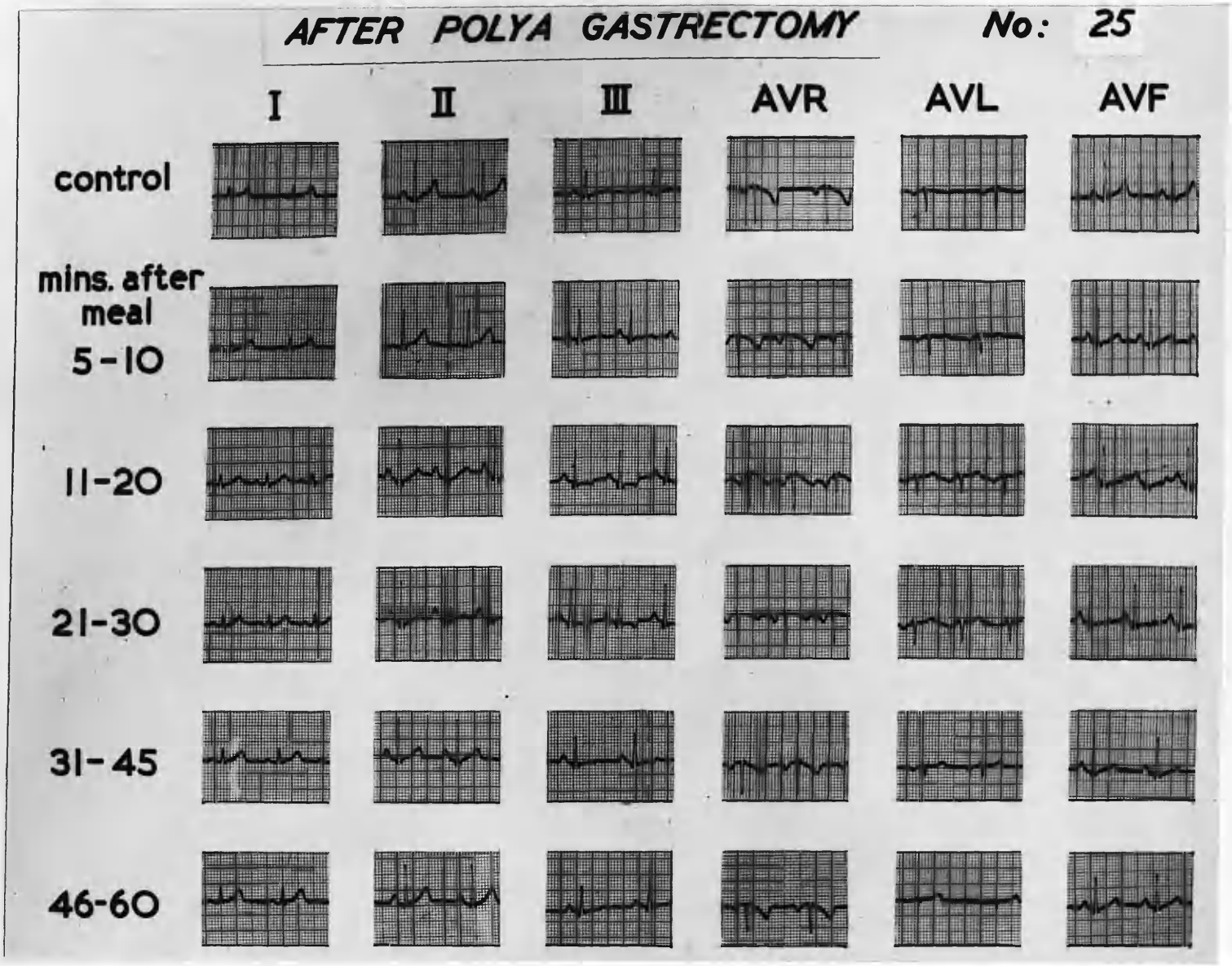

AFTER POLYA GASTRECTOMY
No: 25
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

FIG. 20.

FIG. 20. ELECTROCARDIOGRAPHIC CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN A PATIENT AFTER GASTRECTOMY.

PATIENT 3.

TRACINGS	-	Control and at 6, 12, 25, 40 and 60 mins.
HEART RATE	-	Increased from 70 to 100
P	-	Peaked in II, III and aVF at 12 - 40 mins.
QRS	-	R increased in III and aVF at 12 and 25 mins.
		S less negative in aVL at 40 and 60 mins.
RT	-	ST in III below isoelectric line at 12 and 25 mins.
T	-	Flatter in II at 12 - 40 mins., becomes inverted III at 12 and 25 mins., and biphasic aVF at 12 and 25 mins. Less negative in aVR and 12 - 40 mins., and becomes positive aVL at 12 and 25 mins.
U	-	Increased in II and aVF at 40 mins.
		Maximal changes at 25 mins.

/aVR.

The QT ratio was lengthened.

An increase in potential of the P wave with a peaking of its shape again occurring in leads II, III and aVF.

A U wave made its appearance or became more prominent if already present.

The ST segment take off was below the isoelectric line or the whole ST segment became negative.

The QRS complex was increased in voltage.

The onset was from 5 to 15 minutes after the glucose meal and they usually reached a maximum at about 20 to 30 minutes. Thereafter they reverted slowly towards the control and some residual changes were often still present at the end of the test hour. All of the above did not occur in each case; the first change was flattening of the T wave.

Reproducibility of the changes was shown by repeated tests on the same patient, an example being shown in Vol. II, pages 137 and 138.

INCIDENCE OF THE ECG CHANGES.

The incidence of changes is shown in Table XXVI. They occur more often in patients after operation, particularly after Polya gastrectomy.

One patient before operation exhibited slight T wave changes and two others had the full pattern. These two had similar changes after a Polya gastrectomy though beginning /

PATIENTS	NO. WITH ECG CHANGES	COMPARISON WITH PRE-OPERATIVE PATIENTS		
		χ^2	d.f.	P
PRE-OPERATIVE 12	3 (25 per cent)	-		
POLYA GASTRECTOMY 36	31 (86 per cent)	13.44	1	<0.001
GASTROJEJUNOSTOMY + VAGOTOMY 7	5 (71 per cent)	2.27	1	>0.1
TOTAL 55	39 (71 per cent)	-		

TABLE XXVI.

THE INCIDENCE OF ELECTROCARDIOGRAPHIC CHANGES IN PATIENTS BEFORE AND AFTER OPERATION.

/beginning a little sooner after the meal. Neither had "dumping" symptoms.

After Polya gastrectomy the ECG changes were well marked in twenty-six patients; the remaining five had only T wave changes.

The five patients who were studied after conversion to a Billroth I gastrectomy showed a pattern similar to that observed when they had a Polya anastomosis.

DETAILS OF THE ECG CHANGES. (Table XXVII).

RHYTHM.

Sinus rhythm was normally present but in two cases occasional ventricular extrasystoles occurred, while in a third, who fainted after an attack of bilious vomiting, the tracing showed the temporary onset of partial heart block of the Wenkebach type with dropped beats.

RATE.

The heart rate was increased(by 4 to over 50 beats per minute). Other changes in the ECG were not directly related to the tachycardia, for a rise in rate of only a few beats could be accompanied by obvious changes (Vol. II, pages 104, 111, 114, 122, 129, 131, 132, 141, 143, 145, 147).

CHANGES IN ELECTROCARDIOGRAM.		NO. OF PATIENTS. (Out of 44 with ECG changes)
T WAVE		
Flatter in II, III and aVF		43
(Flatter in 1 - 6)		
Less negative in aVR		27
QT RATIO		
Lengthened (II)		26*
P WAVE		
Peaking in II, III and aVF		25
U WAVE		
Prominent especially in II, III and aVF		23
ST SEGMENT		
Depression	7	
Low take off	6	18
Sagging	5	
QRS COMPLEX		
Increased in voltage in II, III and aVF		14

* Out of 31 patients with ECG changes after Polya gastrectomy.

TABLE XXVII.

DETAILS OF CHANGES IN THE ELECTROCARDIOGRAM FOLLOWING A GLUCOSE MEAL.

P WAVE.

In the 44 tracings showing changes after the glucose meal 25 had peaking of the P wave. In all cases this was best seen in leads II, III and aVF.

QT RATIO.

This was increased in twenty-six out of the thirty-one patients who had ECG changes after Polya gastrectomy.

QRS COMPLEX.

Increase in the voltage of the QRS complex occurred in 14 out of the 44 tracings showing changes following the glucose meal. It usually occurred in leads II, III and aVF and was accompanied by flattening or inversion of the corresponding T wave.

RT SEGMENT.

Depression of the ST segment take off was observed in six cases; in seven the whole of the ST segment was below the isoelectric line and five there was sagging of the ST segment from a positive or an isoelectric take off. Thus, some RT segment changes were present in 18 out of 44 tracings which had alterations following the glucose meal.

T WAVE.

Alterations in the T wave were the first to appear in all cases except one. A flattening and broadening of the deflection with progression to inversion in some cases was best seen in leads II, III and aVF. It was much less frequent in lead I (6 out of 44 tracings with changes) where it was also less marked. There /

/There was often corresponding lessening of the negativity of the T wave in lead aVR (27 out of 44 tracings with changes).

U WAVE.

This deflection made its appearance, or was increased in voltage in 23 of the 44 tracings with changes after the glucose. With the broadening and flattening of the T wave and with the tachycardia it was sometimes difficult to distinguish the T and U waves from one another.

ASSOCIATION OF ECG CHANGES WITH OTHER FINDINGS AFTER THE GLUCOSE MEAL.

No correlation was found between the ECG changes and change in blood pressure, pulse rate or the degree of the fall in plasma volume, as can be seen from the detailed reports of the experiments in Vol. II, pages 10 to 79.

The presence of "dumping" symptoms was not related to the ECG alterations (Table XXVIII).

DISCUSSION.

The changes in the ECG noted in this study are the same as those described after a meal in normal patients. These latter changes have been described by several authors since Gardberg and Olsen (1939) showed a 30 to 50 per cent decrease in the height of the T wave 30 minutes after a meal, a change which lasted for up to 2 hours. A statistical study of the effect of meals on the ECG by Simonson et al., (1946) revealed a tachycardia with a decrease in the amplitude of the T wave as the main change. /

ECG CHANGES	"DUMPING" SYMPTOMS	x^2	d.f.	P
PRESENT 31	17 (55%)	0.93	1	>0.3
ABSENT 5	1 (20%)			

TABLE XXVIII.

THE INCIDENCE OF "DUMPING" SYMPTOMS IN PATIENTS AFTER POLYA GASTRECTOMY WITH AND WITHOUT ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE.

/change. The QT interval, corrected for heart rate, was lengthened and the QRS complex increased in voltage. The ST segment changes were not pronounced and these authors expressed the belief that sympathetic stimulation might explain the T wave changes and did not think that diminished coronary blood flow was the cause. This work was amplified by Simonson and Keys (1950) who were of the opinion that the T wave changes were associated with a change in the ventricular gradient, a view which was upheld by Rosen and Gardberg (1957 a,b). Neither group further elucidated the cause of the changes. The use of a glucose meal (100 g.) elicited ECG changes in forty-seven out of eighty-five normal patients (Rochlin and Edwards, 1954). These alterations were mainly a decrease in the voltage of the T wave which was also observed in seventy-eight out of 2,000 healthy young men in a random study (Sears and Manning, 1958). From the above description and our results in the patients before operation it is clear that the ECG changes following a glucose meal in patients after gastrectomy have the same pattern as those in normal subjects except that the changes are more frequent, occur more rapidly and are more marked after operation.

In contradistinction to the work of Roberts et al., (1954) the present study has not shown any correlation between blood pressure change, amount of fall in plasma volume or "dumping" symptoms on the one hand with ECG changes on the other. /

/other.

CONCLUSIONS.

A pattern of changes in the electrocardiogram following a glucose meal in patients after operation is found which is the same as in normal people but is exaggerated after operation.

There is no correlation between the ECG changes and the other cardiovascular changes after the meal or to the incidence of "dumping" symptoms.

Changes occurring in patients before operation do not presage the development of "dumping" symptoms after operations.

PART II.

THE MECHANISM OF PRODUCTION OF THE ELECTROCARDIOGRAPHIC CHANGES.

The factors which have been thought to contribute to the ECG changes following a meal in patients after gastrectomy are:

Coronary arterial insufficiency	(Roberts et al., 1954)
Potassium deficiency	(Smith, 1951).
Rapid glucose absorption	(Pulvertaft, 1954)
Sympathetic overactivity	(Smith et al., 1953)

Additional experiments have been undertaken to evaluate the importance of these four factors and to find the effect of lying down on the ECG changes. In all studies the conditions were the same as in Part I only the test substance and the position being varied.

CORONARY ARTERIAL INSUFFICIENCY.

One of the main arguments used by Roberts et al., (1954) in favour of this explanation was that the ECG changes occurred in a period of hypotension. We have found typical changes when there has been slight hypertension.

It was suggested by Simonson et al., (1946) that the pattern of ECG changes after a meal does not necessarily indicate coronary arterial insufficiency, particular attention being drawn to the small changes in the ST segment. In the present investigation ST changes were not marked and the other changes /

/changes were always most pronounced in leads II, III and aVF, a finding which is also not in favour of coronary arterial insufficiency where one might reasonably expect some patient to show another distribution of the "ischaemic" changes. Furthermore part of the ST changes may be due to the tachycardia (Sjöstrand, 1950 b).

ADDITIONAL EXPERIMENTS.

Six patients after Polya gastrectomy were given rapid intravenous infusion of 500 ml. Dextran (6 per cent in saline) or 500 ml. plasma at a time when ECG changes were beginning to appear following the ingestion of 100 ml. 50 per cent glucose. In only one case was the duration of the changes shortened compared with the test meal given alone. The volume of plasma (or plasma expander) given was greater than the fall in plasma volume measured at the first experiment so that a decrease in the circulating blood volume should have been balanced. This suggests that a drop in coronary blood flow is not likely to be the main cause of the ECG changes.

Evidence against myocardial ischaemia also comes from the effect of intravenous injection of ergotamine tartrate 0.5 mg. which rapidly abolished the ECG changes in nine patients after operation (see page 108). This drug tends to constrict the coronary vessels (Katz and Linder, 1939).

POTASSIUM DEFICIENCY.

In patients after gastrectomy Smith (1951) suggested that a fall in serum potassium may be an important aetiological factor in the ECG changes. The pattern of changes recorded in Part I are similar to those described by Bellet et al., (1950) in hypokalaemia although the peaking of the P wave was not noted.

ADDITIONAL EXPERIMENTS.

Plasma potassium levels were estimated in twenty patients who had ECG changes after the glucose meal. The estimation was carried out on a 1 in 500 dilution of plasma using an EEL flame photometer. No constant trend was found in the test hour, the readings all being within the range of normal.

PLASMA POTASSIUM MG. PER CENT (MEAN OF TWENTY PATIENTS)				
CONTROLS		MIN. AFTER GLUCOSE		
		15	30	45
17.2	17.2	17.1	16.8	17.4

This does not exclude an intracellular potassium deficiency as Rochlin and Edwards (1954) noted when they found that 3 g. potassium chloride given orally prevented ECG changes after a glucose meal. However, potassium has a general /

/general tendency to reverse the direction of abnormal T waves regardless of the cause of the abnormality (Goldberger (1953).

Two patients with hypokalaemia (serum potassium 12 mg. and 13.5 mg. per cent) were given 0.5 mg. ergotamine tartrate intravenously with no effect on the ECG changes which were typical of potassium deficiency. This is in marked contrast to the amelioration in the nine patients who were given ergotamine after the glucose meal (page 108).

RAPID GLUCOSE ABSORPTION.

An association between the rate of absorption of glucose and the ECG changes in patients after gastrectomy was suggested by Pulvertaft (1954).

ADDITIONAL EXPERIMENTS.

The blood sugar levels were measured (Hagedorn and Jensen, 1923) in 27 patients after gastrectomy who had ECG tracings. There was no difference between the five who had no ECG changes and the twenty-two who had (Table XXIX).

Four patients were given 0.5 g. glucose per kg. intravenously as a 20 per cent solution and ECG tracings recorded. These patients all had typical changes with the glucose when given orally. The intravenous glucose produced equivalent blood sugar levels (Table XXX) but no ECG changes.

Three patients were given hypertonic saline (16.3 g. per 100 ml., isotonic with the glucose meal) by a /

	BLOOD SUGAR MG. PER CENT			
	CONTROL	MINS. AFTER GLUCOSE		
		15	30	45
NO ECG CHANGES 5	88.8±2.58*	128.5±5.61*	162.7±7.33*	172.1±9.29*
ECG CHANGES 22	97.0±5.39*	121.6±6.19*	165.4±14.62*	169.8±15.15*

* S.E. MEAN

TABLE XXIX.

MEAN BLOOD SUGAR LEVELS IN 27 PATIENTS AFTER GASTRECTOMY FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE, COMPARING THOSE WITH AND WITHOUT ELECTROCARDIOGRAPHIC CHANGES.

GLUCOSE TEST	BLOOD SUGAR MG. PER CENT			
	CONTROL	MIN. AFTER GLUCOSE 15	30	45
ORAL	92	137	191	193
INTRAVENOUS	96	209	206	195

TABLE XXX.

MEAN BLOOD SUGAR LEVELS IN 4 PATIENTS AFTER GASTRECTOMY COMPARING THE EFFECT OF 100 ml. 50 PER CENT GLUCOSE ORALLY AND 0.5 G. / Kg. INTRAVENOUSLY.

/a transnasal intrajejunal tube and ECG changes the same as those after the glucose meal were observed (Fig. 21). The pattern was identical in one, slightly less in one and more fully developed in the third.

The above evidence leads to the conclusion that the ECG changes are not related to the rise in blood sugar.

SYMPATHETIC OVERACTIVITY.

The pattern of ECG changes after a glucose meal in patients after gastrectomy is the same as that seen in normal patients after a meal (Part I) and this has been said to be due to sympathetic stimulation (Simonson et al., 1946).

ADDITIONAL EXPERIMENTS.

Methods to alter the balance between the sympathetic and the parasympathetic nervous systems were used to study this further:-

SYMPATHETIC NERVOUS SYSTEM.

Effect of adrenaline.

Effect of postural change.

Effect of sympatheticolytic drugs on the changes following the glucose meal.

PARASYMPATHETIC NERVOUS SYSTEM.

Effect of atropine.

Effect of ergotamine tartrate on the changes following the glucose meal.

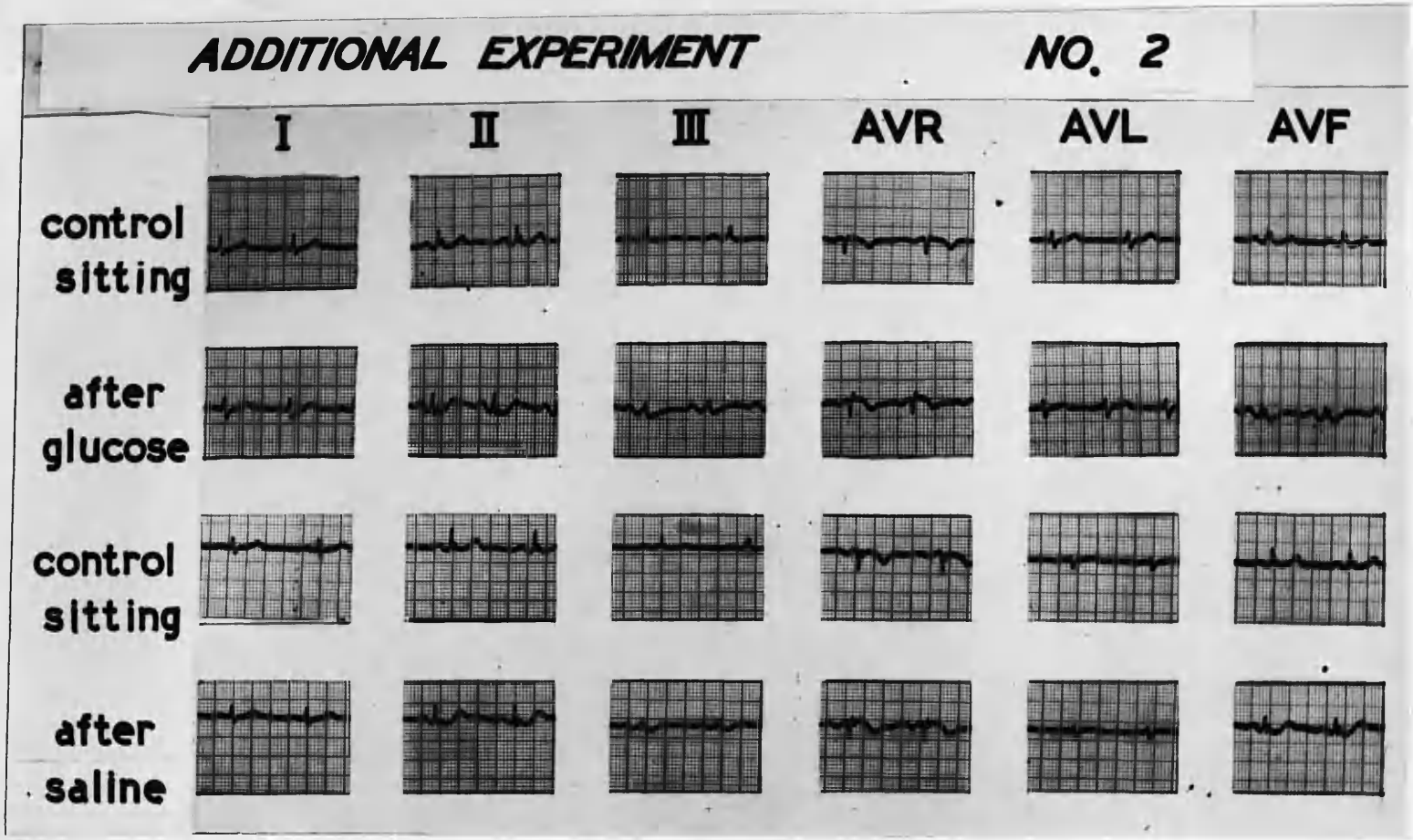

ADDITIONAL EXPERIMENT
NO. 2
I
II
III
AVR
AVL
AVF
control sitting
after glucose
control sitting
after saline

FIG. 21.

FIG. 21. ELECTROCARDIOGRAPHIC CHANGES FOLLOWING THE INGESTION OF GLUCOSE COMPARED WITH THOSE AFTER HYPERTONIC SALINE.

PATIENT 57.

GLUCOSE Tracings - Control and 18 mins.

Heart rate increased from 68 to 94: P:- N_o change from the control: QRS:- No change from the control: RT:- ST sagging below the isoelectric line II, III and aVF and take off ST aVR elevated: T:- broader and runs into U waves: U:- increased II, III and aVF.

HYPERTONIC SALINE Tracings - Control and 18 mins.

Heart rate increased from 66 to 75: P:- No change from the control: QRS:- No change from the control: RT:- ST sagging below isoelectric line II, III and aVF: T:- broader and runs into U waves: U:- increased II, III and aVF.

The results of these studies are then analysed.

SYMPATHETIC NERVOUS SYSTEM.

EFFECT OF ADRENALINE.

It was shown by Hartwell (1942) that adrenaline caused flattening of the T waves of the ECG and Smith et al., (1953) compared the changes after the subcutaneous injection of adrenaline and those after glucose. In the present study six patients after Polya gastrectomy were given 1 mg. adrenaline hydrochloride by subcutaneous injection and the effect on the ECG compared with that after the glucose meal. The general pattern was seen to be similar (Fig. 22) and the changes were almost identical in three cases. However, there was more tendency for ST changes with the adrenaline and the U wave changes were not so obvious. In addition, there was a tendency to arrhythmia, supraventricular extrasystoles being common and transient inversion of the P wave occurred in three patients.

EFFECT OF POSTURAL CHANGE ON THE ECG.

Alteration of posture changes the ECG and the changes have been attributed in part to stimulation of the sympathetic nervous system (Nordenfelt, 1941: Myerson and Davis, 1942: Scherf and Schlachman, 1948a). After gastrectomy nine patients who had ECG changes with the glucose meal were tested by taking a control ECG on a horizontal table and then tilting the table /

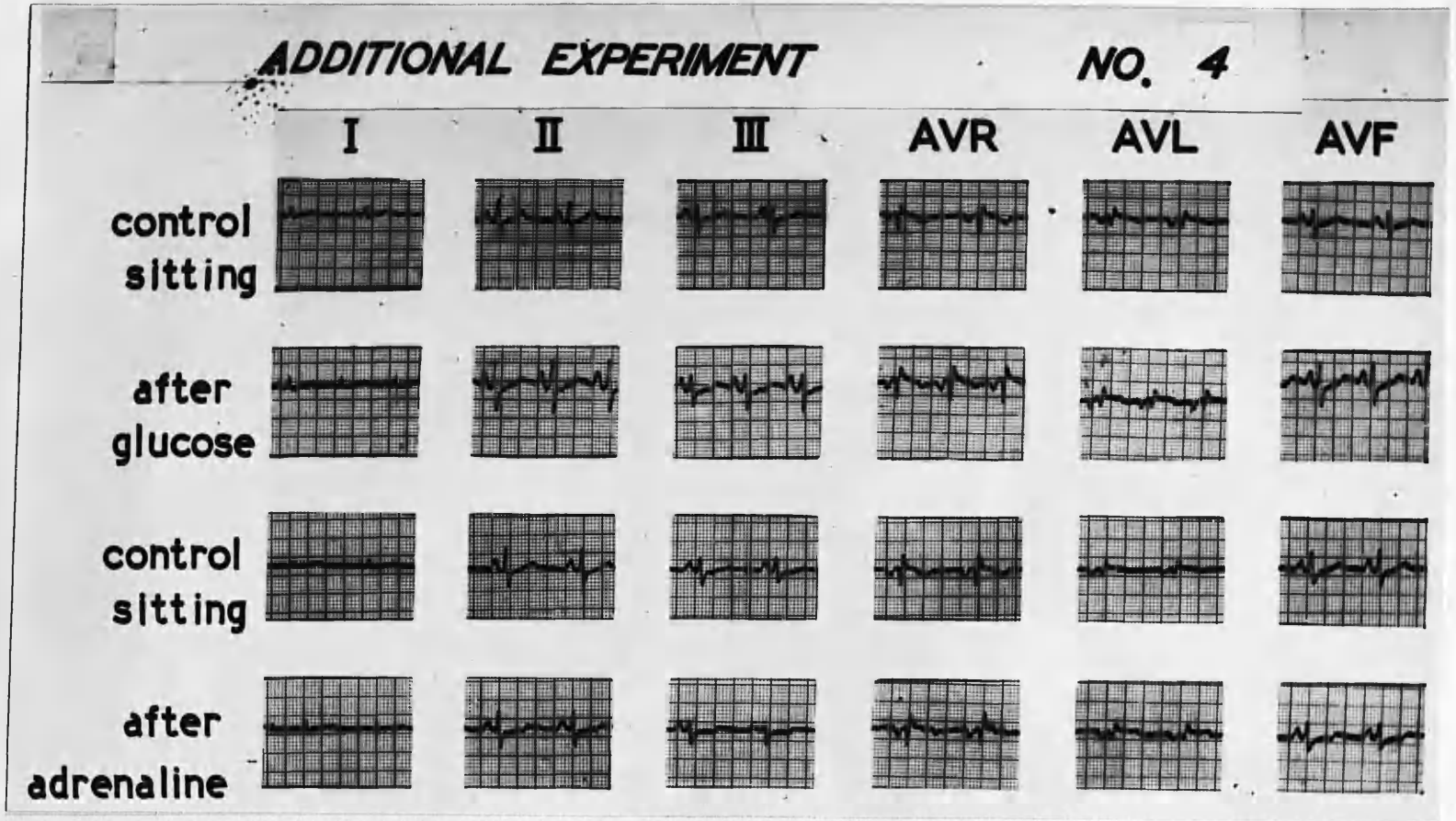

ADDITIONAL EXPERIMENT
NO. 4
I
II
III
AVR
AVL
AVF
control sitting
after glucose
control sitting
after adrenaline

FIG. 22.

FIG. 22. ELECTROCARDIOGRAPHIC CHANGES FOLLOWING THE INGESTION OF GLUCOSE COMPARED WITH THOSE AFTER SUBCUTANEOUS INJECTION OF 1 mg. ADRENALINE.

PATIENT 40.

GLUCOSE Tracings - Control and 30 mins.

Heart rate increased from 72 to 100: P:- peaked II, III and aVF: QRS:- increased II, III and aVF: RT:- ST below isoelectric line in II, III and aVF: T:- flatter and broader II, III and aVF, biphasic III, less negative aVR, biphasic aVL: U:- No c hange from the control

ADRENALINE Tracings - Control and 33 mins.

Heart rate increased from 72 to 76: P:- peaked aVF: QRS:- No change from the control: RT:- ST below isoelectric line II, III and aVF: elevated aVL: T:- biphasic II, III and aVR, aVL, and aVF: U:- No change from the control.

/table to an angle of 75 degrees to the horizontal. Tracings were taken at minute intervals up to 10 minutes when the patient was returned to the horizontal.

The pattern of changes was the same as after glucose (Fig. 23). In six cases the changes were closely similar and in the remaining three they were less well marked after the postural change than after the glucose meal. The changes began immediately on tilting and progressed to reach a maximum at three to seven minutes. This progression indicated the changes were not due solely to rotation of the heart.

EFFECT OF SYMPATHETICOLYTIC DRUGS ON THE ECG CHANGES FOLLOWING THE GLUCOSE MEAL.

The drugs used were phentolamine and phenoxybenzamine hydrochloride (dibenzyline*) which were given to patients after gastrectomy who were studied in the usual way. The drug was given when the ECG changes were present after the glucose meal. Five patients were given 10 mg. phentolamine intravenously and three were given 0.7 to 1.0 mg. dibenzyline per kg. by intravenous infusion. In two other patients a total of 140 mg. dibenzyline was given orally in the 12 hours preceding the test meal of glucose. In none of these experiments did the drugs used prevent the changes or accelerate their disappearance. If anything, there was slight /

* Kindly supplied by Smith, Kline and French Laboratories, Ltd., Philadelphia, Pa.

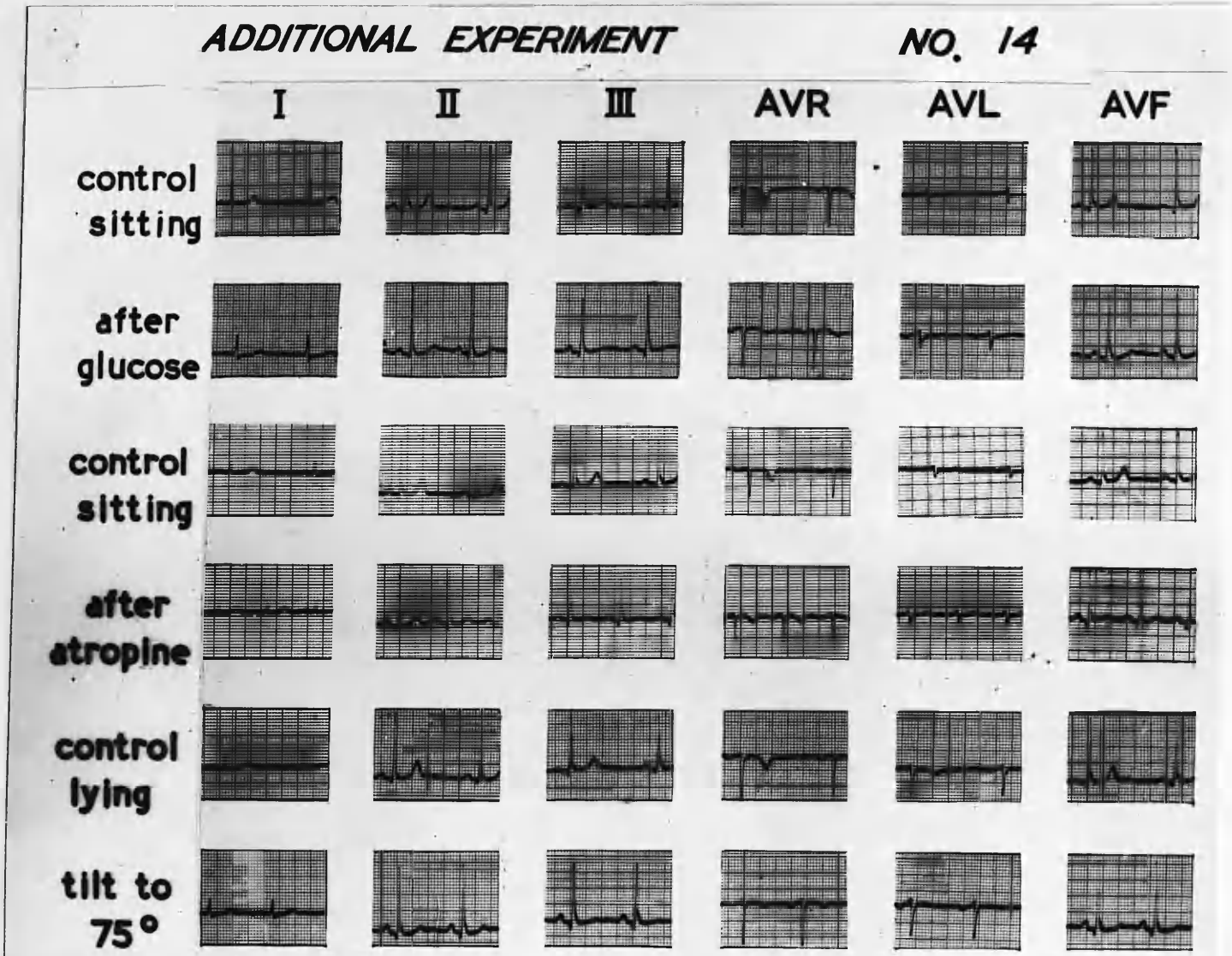

ADDITIONAL EXPERIMENT
NO. 14
I
II
III
AVR
AVL
AVF
control sitting
after glucose
control sitting
after atropine
control lying
tilt to 75°

FIG. 23.

FIG. 23. ELECTROCARDIOGRAPHIC CHANGES FOLLOWING THE INGESTION OF GLUCOSE COMPARED WITH THOSE AFTER INTRAVENOUS INJECTION OF ATROPINE AND AFTER POSTURAL CHANGE.

PATIENT 29.

GLUCOSE Tracings - Control and 38 mins.

Heart rate increased from 58 to 74: P:- peaked aVF: QRS:- increased R II and III: RT:- No change from the control: T:- flatter I, II, III and aVF and less negative aVR: U:- increased in II and aVF.

ATROPINE Tracings - Control and 15 mins.

Heart rate increased from 58 to 94: P:- peaked aVF: QRS:- No change from the control: RT:- No change from the control: T:- Flatter I, II, III and aVF and less negative aVR: U:- No change from the control.

POSTURE Tracings - Control and 5 mins.

Heart rate increased from 54 to 72: P:- peaked II, III and aVF: QRS:- No change from the control: RT:- No change from the control: T:- flatter I, II, III and aVF and less negative aVR and aVL: U:- increased in II.

/slight prolongation of the time before recovery began.

Both phentolamine and dibenzyline block the responses to the adrenergic mediators, adrenaline and noradrenaline (Goodman and Gilman, 1955) and it appears unlikely that the ECG changes are due to adrenaline release into the general circulation. However, dibenzyline does not prevent the compensatory cardiac reflexes and phentolamine itself may cause a "sympatheticomimetic" tachycardia (Goodman and Gilman, 1955) so that failure to reverse the ECG changes does not exclude an overactivity of the sympathetic nervous system.

PARASYMPATHETIC NERVOUS SYSTEM.

EFFECT OF ATROPINE ON THE ECG.

By blocking the vagus, atropine allows overactivity of the sympathetic nervous system and a tachycardia is one of the results. Atropine causes changes in the T wave of the ECG (Hartwell, 1942). Five patients after gastrectomy were given two doses each of 1/100 gr. atropine sulphate intravenously at 15 minute intervals under the usual test conditions. Comparison of the maximal changes obtained with those after the glucose meal showed that the pattern was similar (Fig. 23) in three patients and less well developed in two, when the maximal changes after atropine were the same as an earlier stage in the changes after the glucose. /

/glucose.

EFFECT OF ERGOTAMINE ON THE ECG CHANGES FOLLOWING A GLUCOSE MEAL.

Ergotamine tartrate 0.5 mg. was given by intravenous injection to nine patients after gastrectomy when they had developed ECG changes following a glucose meal. In all nine the changes were rapidly reversed (Fig. 24) and the ECG returned to the control except that U waves still tended to be increased.

To provide controls for this action three patients who had pathologically flattened T waves (two with a left heart strain pattern and one with an old posterior myocardial infarct) were given 0.5 mg. ergotamine tartrate intravenously, amyl nitrite being at hand should any worsening of the tracing occur. No restoration of the T waves occurred and there was further depression in one case. This responded rapidly to the inhalation of amyl nitrite. An improvement in organic T wave changes was reported in three out of thirteen patients who were given intravenous ergotamine by Scherf and Schlachman (1948b). As it can be seen the non-specific improvement is, at best, variable in contrast to the uniform reversion of the ECG changes after the glucose.

A further control was obtained from two patients who were given mephentermine sulphate 15 mg. intravenously at the same time interval as the ergotamine tartrate so that the vasoconstrictor effect of ergotamine might be imitated. No /

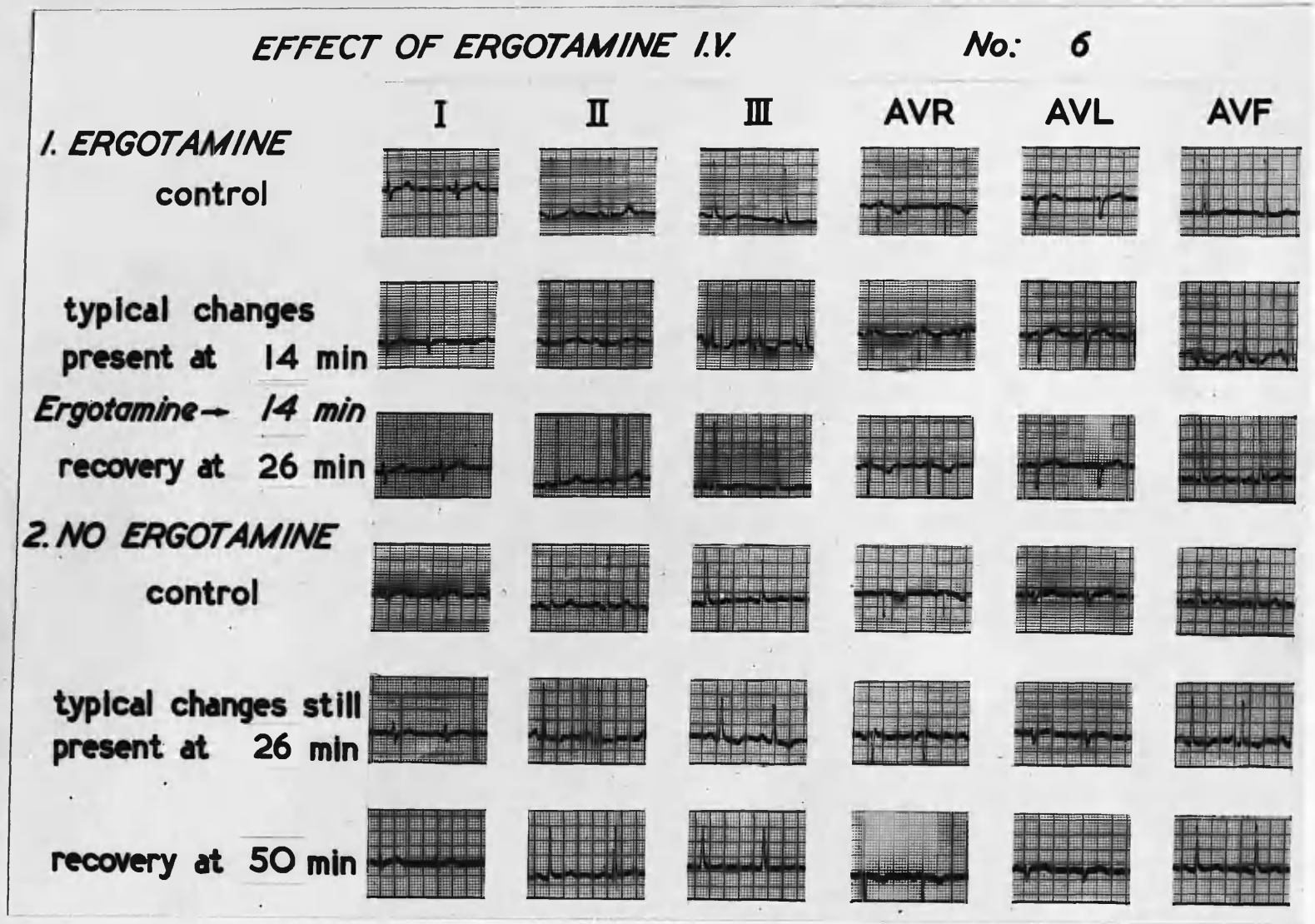

EFFECT OF ERGOTAMINE I.V.
No: 6
I
II
III
AVR
AVL
AVF
1. ERGOTAMINE
control
typical changes
present at 14 min
Ergotamine→ 14 min
recovery at 26 min
2. NO ERGOTAMINE
control
typical changes still
present at 26 min
recovery at 50 min

FIG. 24.

FIG. 24. ELECTROCARDIOGRAPHIC CHANGES FOLLOWING THE INGESTION OF GLUCOSE SHOWING THE EFFECT OF INTRAVENOUS INJECTION OF 0.5 mg. ERGOTAMINE TARTRATE.

PATIENT 60.

1. GLUCOSE AND ERGOTAMINE

14 mins. Heart rate increased from 65 to 94: P:- peaked II and aVF: QRS:- R II, III and aVF increased: RT:- No change: T:- flatter I, biphasic II, inverted III and aVF; less negative aVR and more positive aVL: U:- No change.

26 mins. Heart rate 75: P:- control: QRS:- R III and aVF still increased. T:- back to control voltage : U:- No change.

2. GLUCOSE AND NO ERGOTAMINE.

26 mins. Heart rate increased from 74 to 84: P:- peaked II and aVF: QRS:- No change: RT:- No change: T:- flatter I, biphasic II and aVR, inverted II and aVF: U:- No change.

50 mins. Heart rate 75: P:- as control: QRS:- No change: RT:- No change: T:- return to control voltage : U:- No change.

/No improvement in the ECG appearances resulted.

The dose of ergotamine tartrate given in this investigation is too small to cause a sympathetic block in man but exercises an effect on the cardiac reflexes by central vagal stimulation (Goodman and Gilman, 1955). This would be compatible with a relative sympathetic overactivity causing the ECG changes with restoration of the balance by the ergotamine.

ANALYSIS.

The preceding five groups of experiments point to sympathetic stimulation as having a part in the production of the ECG changes following a glucose meal. The pattern after glucose is similar to that after adrenaline, after atropine and after postural change and it is reversed by the vagal stimulation of ergotamine. Two sympatheticolytic drugs, phentolamine and dibenzyline, gave no improvement showing that adrenaline release is not the mechanism.

That sympathetic stimulation is not the whole explanation is shown by the greater change in some patients following the glucose than after atropine or postural change and by the partial relief with plasma expander in one case.

Analysis of the T wave changes was undertaken by plotting the height of the T wave against the corresponding pulse rate. Each was measured from at least four complexes in lead II which was chosen as a lead in which the changes were well marked. /

/marked. There is a rough linear relationship after atropine and postural changes (Fig. 25) which has been noted previously (Sjöstrand, 1950a). Glucose gives a steeper slope in four patients, although the linear correlation is not close in one of these cases (No. 57). This effect is similar to that observed after amyl nitrite which was explained on the basis of some haemodynamic factor by Sjöstrand (1950a).

It may be argued that these experiments which reproduce the pattern of changes do so because they all cause a tachycardia. That this is not the only basis for the ECG changes after glucose is shown by the patients who have typical changes with a rise in heart rate of only a few beats.

EFFECT OF LYING DOWN ON ECG CHANGES FOLLOWING A GLUCOSE MEAL.

The initiation of these ECG changes is from the alimentary tract and, to attempt to define the mechanism, five patients were given the meal of 100 ml. 50 per cent glucose by transnasal intrajejunal tube in the recumbent position. Some changes took place in all patients but they were much less than in the sitting position (Fig. 26). It has already been shown that lying down does not abolish bowel hypermotility after taking glucose and that the other cardiovascular changes are less in this posture. The fact that the ECG changes are also less further illustrates that a reflex from the bowel cannot develop fully in recumbency. It is attractive to postulate that some /

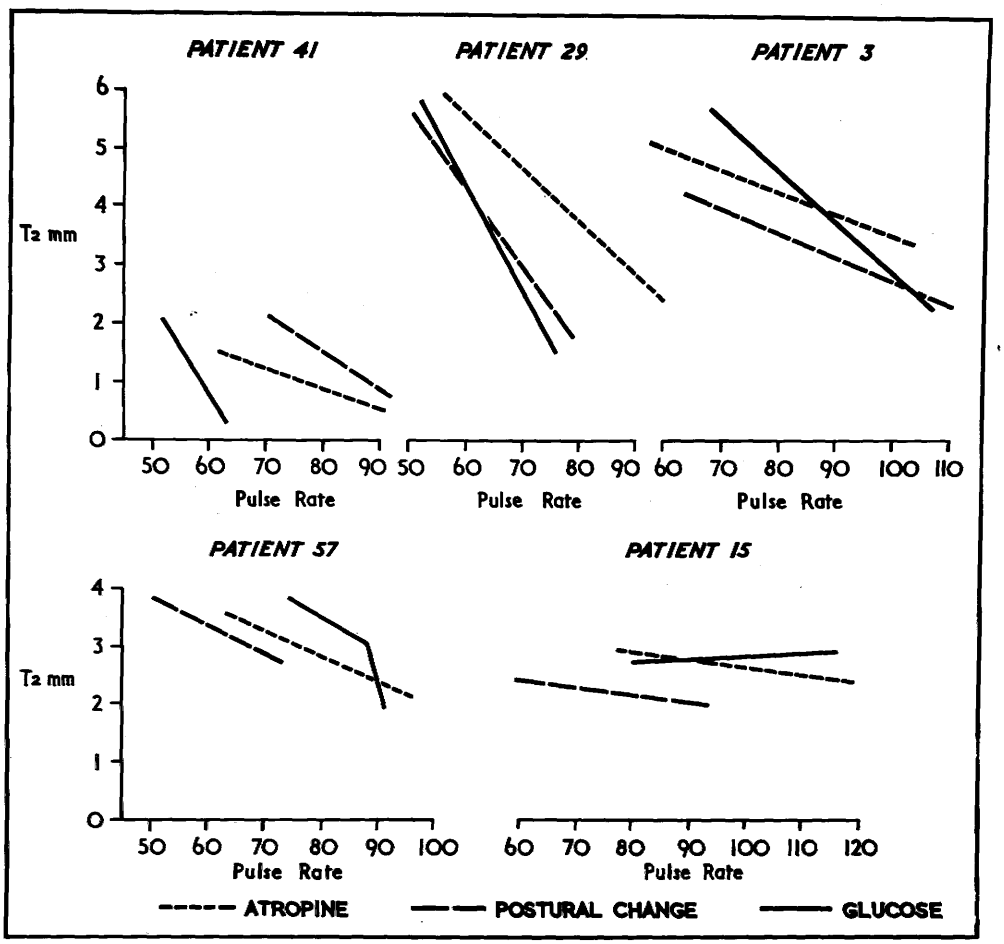


FIG. 25. COMPARISON OF RELATIONSHIP BETWEEN HEIGHT OF T WAVE IN LEAD II AND PULSE RATE IN PATIENTS AFTER POLYA GASTRECTOMY FOLLOWING ADMINISTRATION OF ORAL GLUCOSE (100 ml. 50 PER CENT), SUBCUTANEOUS ATROPINE (1/100 gr. ATROPINE SULPHATE) AND CHANGE OF POSTURE FROM HORIZONTAL TO 75 DEGREES TO THE HORIZONTAL.

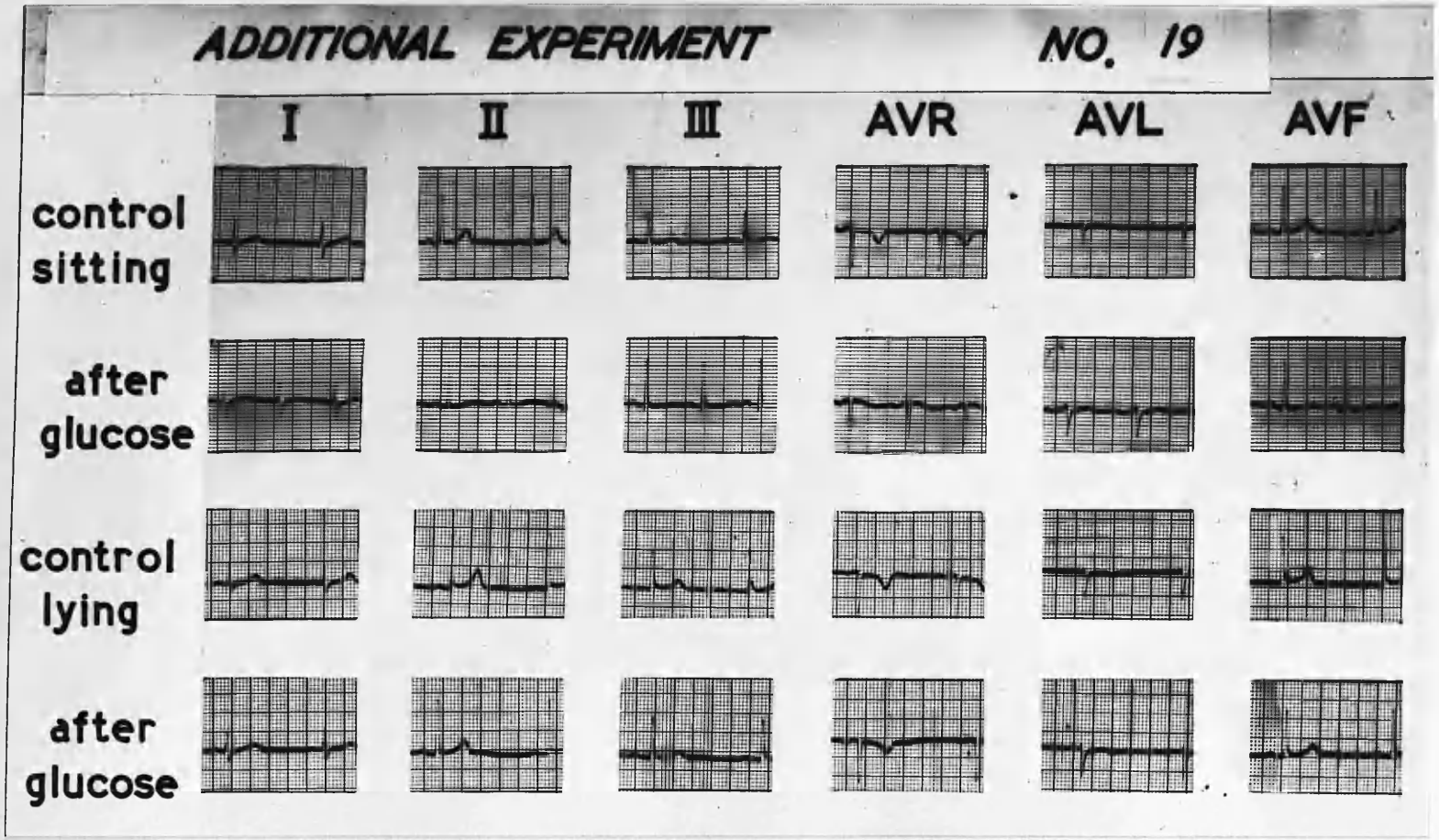

ADDITIONAL EXPERIMENT
NO. 19
I
II
III
AVR
AVL
AVF
control sitting
after glucose
control lying
after glucose

FIG. 26.

FIG. 26. ELECTROCARDIOGRAPHIC CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN THE SITTING POSITION AND THE ADMINISTRATION OF THE SAME MEAL BY INTRA-JEJUNAL TUBE IN RECUMBENCY.

PATIENT 58.

GLUCOSE SITTING Tracings - Control and 25 mins.

Heart rate increased from 60 to 100: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- flatter and broader II, III, aVF, less negative aVR and positive aVL: U:- II and aVF.

GLUCOSE LYING Tracings - Control and 25 mins.

Heart rate unchanged: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- slightly flatter II, III, aVF and slightly less negative aVF: U:- II.

/some haemodynamic change is abolished when the effect of gravity is neutralised by lying down and the same haemodynamic change plays a part in the ECG changes in the sitting position.

CONCLUSION.

The electrocardiographic changes which follow a glucose meal are mainly due to sympathetic nervous system overactivity with some additional haemodynamic factor.

BLOOD SUGAR LEVELS AND THEIR RELATIONSHIP TO OTHER FINDINGS

FOLLOWING A MEAL IN PATIENTS AFTER GASTRECTOMY.

BLOOD SUGAR LEVELS AND THEIR RELATIONSHIP TO OTHER FINDINGS FOLLOWING A MEAL IN PATIENTS AFTER GASTRECTOMY.

Opinions vary as to the existence of a relationship between the rise in blood sugar which follows a glucose meal in patients after gastrectomy and the "dumping" syndrome. In this section an investigation of this association is reported.

MATERIAL AND METHODS.

Blood sugar estimations were made simultaneously with the cardiovascular and electrocardiographic readings previously reported. Thirteen patients were studied before operation, forty after Polya gastrectomy, six before and after conversion from Polya to Billroth I gastrectomy and seven after gastrojejunostomy. The estimations were made by the method of Hagedorn and Jensen (1923) before and at three 15-minute intervals after a glucose meal. Before taking the meal, patients were fasted for 12 hours. The meal, which was swallowed in a sitting position, consisted of 100 ml. 50 per cent glucose and, on two occasions, 100 ml. of 50 per cent mannitol. In four patients, hypertonic saline of identical osmolarity to the glucose meal (16.3 g. in 100 ml.) was administered by a transnasal intrajejunal tube and in another four an intravenous infusion of glucose (0.5 g. per kg. as a 20 per cent solution) was given under similar circumstances. /

/circumstances.

The standard meal of 100 ml. of 50 per cent glucose was introduced by a transnasal intrajejunal tube in five recumbent patients.

RESULTS.

In the thirteen patients studied before operation, the rise in blood sugar following the ingestion of the glucose meal was within the normal limits defined by Peters and Van Slyke (1946) Table XXXI. Two of these patients had "dumping" symptoms after operation; their preoperative response was no different from that of the other eleven patients who were without postgastrectomy symptoms.

After Polya gastrectomy, a more rapid rise in blood sugar took place after the ingestion of glucose (Table XXXI). The mean blood sugar levels in twenty patients in whom the ingestion of glucose produced "dumping" symptoms were not different from those in twenty patients in whom no symptoms occurred (Table XXXII, Fig. 27).

The administration of glucose intravenously in four patients produced an increase in blood sugar similar to that which followed the glucose meal (Table XXX). However, in one patient who had "dumping" symptoms following oral glucose, these did not occur when the intravenous route was used for its administration despite a similar rise of blood sugar. These findings suggest that no correlation exists between the development of a hyperglycaemic state and the onset of "dumping" symptoms. The production of /

PATIENTS	NO.	BLOOD SUGAR MG. PER CENT			
		CONTROL	MIN. AFTER GLUCOSE 15	30	45
PREOP.	13	90	108	142	148
AFTER POLYA GASTRECTOMY	40	90	131	163	181
AFTER GASTRO-JEJUNOSTOMY	7	90	123	160	161

TABLE XXXI.

MEAN BLOOD SUGAR LEVELS FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 13 PATIENTS BEFORE OPERATION, 40 AFTER POLYA GASTRECTOMY AND 7 AFTER GASTROJEJUNOSTOMY WITH VAGOTOMY.

PATIENTS	NO.	BLOOD SUGAR MGM. PER CENT			
		CONTROL	MIN. AFTER GLUCOSE 15	30	45
"DUMPING" SYMPTOMS	20	89±3.85*	126±5.51*	166±7.43*	186±4.85*
NO "DUMPING" SYMPTOMS	20	91±3.22*	136±8.11*	161±7.53*	176±4.26*
t		0.38	0.99	0.46	1.51
P		>0.7	>0.3	>0.6	>0.1

* S.E.MEAN

TABLE XXXII.

MEAN BLOOD SUGAR LEVELS FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE IN 40 PATIENTS AFTER GASTRECTOMY COMPARING THOSE WITH AND WITHOUT "DUMPING" SYMPTOMS.

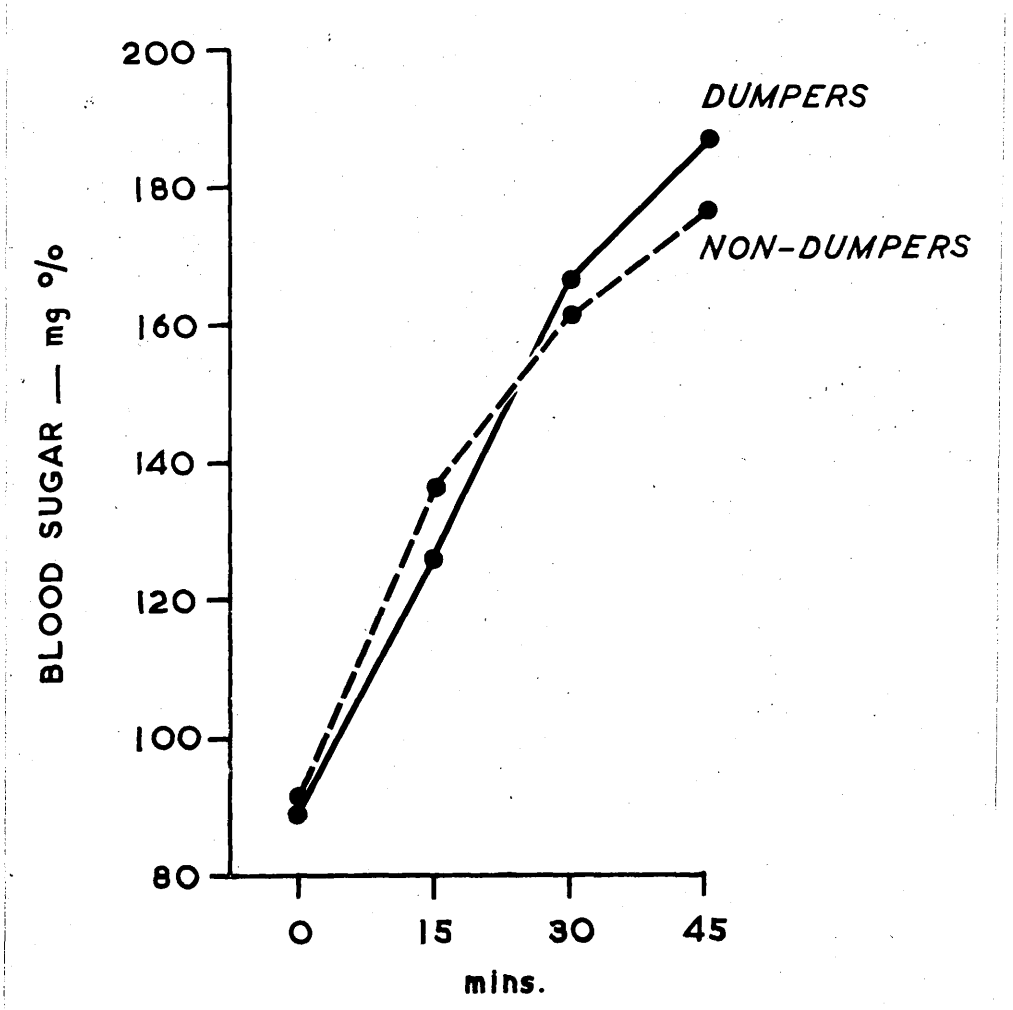


FIG. 27. COMPARISON OF MEAN BLOOD SUGAR LEVELS FOLLOWING INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 20 PATIENTS WITH "DUMPING" SYMPTOMS AND 20 WITH NO "DUMPING" SYMPTOMS AFTER POLYA GASTRECTOMY.

/of typical "dumping" attacks in two patients given a mannitol meal and in two of four patients given hypertonic saline confirms these findings.

In the five patients in whom blood sugar levels were estimated when the glucose meal was given in both sitting and recumbent positions, the changes in blood sugar were identical (Table XXXIII). One of these five patients had typical "dumping" attacks when sitting but none when lying flat.

The rate of rise of blood sugar was not related to any of the cardiovascular changes which were found after administration of glucose and similar changes followed the meal of mannitol despite no increase in blood sugar levels. (Fig. 28). In particular, there was no correlation between the rise of blood sugar and fall of plasma volume (Fig. 29). Similarly, the electrocardiographic changes were unrelated to the hyperglycaemia (see previous Section).

Following gastrojejunostomy and vagotomy in seven patients, the rise of blood sugar after ingestion of glucose was again more rapid than that in patients before operation (Table XXXI). No relationship between the blood sugar levels and symptoms or cardiovascular changes was observed.

Six patients who were studied before and after conversion of a Polya to a Billroth I type of gastrectomy had a slightly slower absorption of glucose after the operation. As had previously been stated, this operation did not result in /

FIVE PATIENTS	BLOOD SUGAR MG. PER CENT			
	CONTROL	MINS. AFTER GLUCOSE 15	30	45
LYING	82	158	177	221
SITTING	83	153	196	219

TABLE XXXIII.

MEAN BLOOD SUGAR LEVELS FOLLOWING 100 ml. 50 PER CENT GLUCOSE IN 5 PATIENTS AFTER GASTRECTOMY COMPARING INTRAJEJUNAL ADMINISTRATION WHEN LYING AND ORAL INTAKE WHEN SITTING.

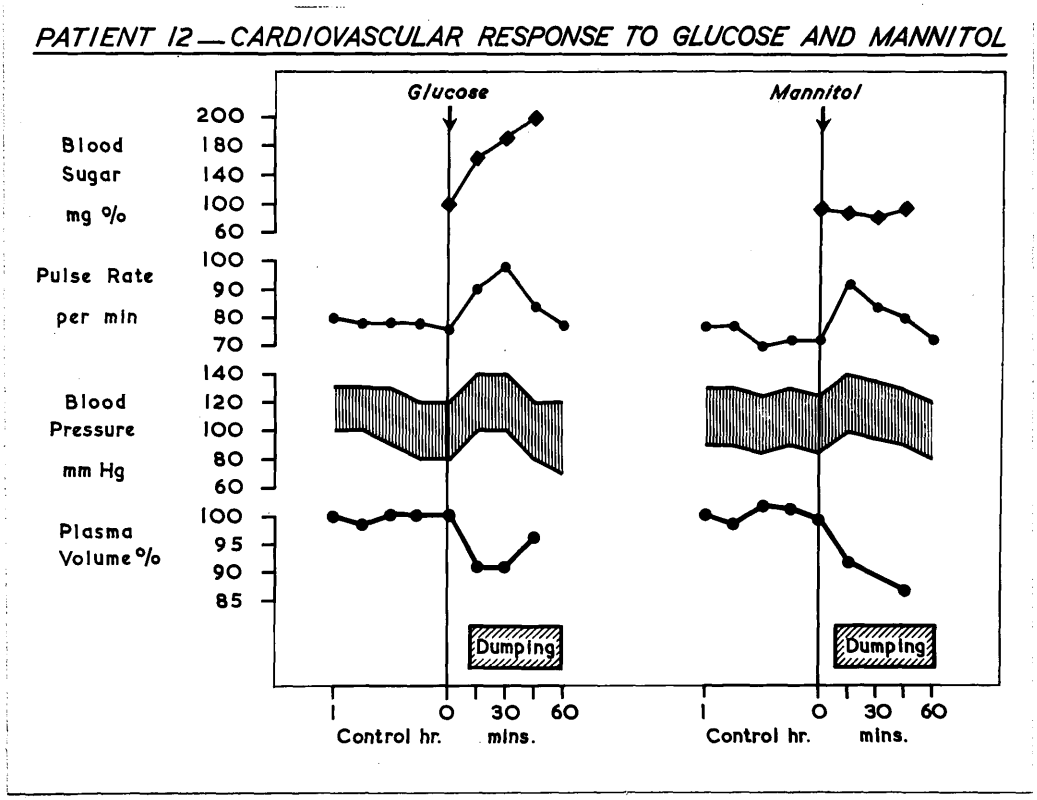


FIG. 28. COMPARISON OF CARDIOVASCULAR CHANGES FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE AND 100 ml. 50 PER CENT MANNITOL IN THE SAME PATIENT AFTER POLYA GASTRECTOMY.

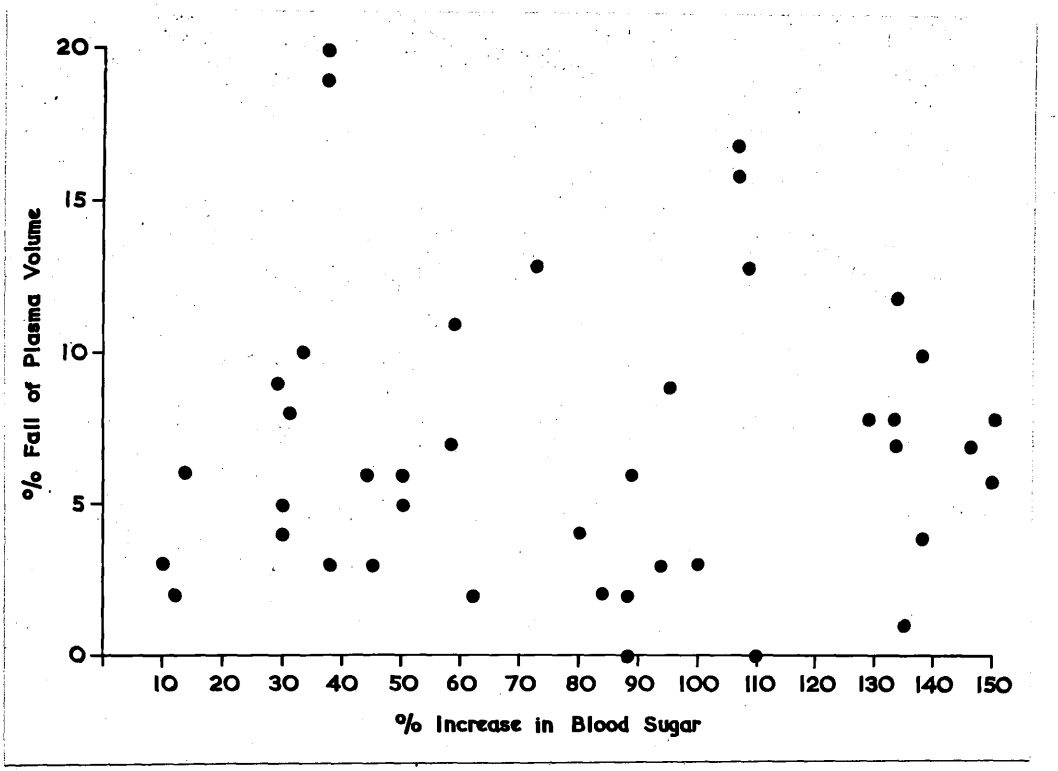


FIG. 29. SCATTERGRAM SHOWING LACK OF CORRELATION BETWEEN FALL IN PLASMA VOLUME AND RATE OF RISE IN BLOOD SUGAR FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN PATIENTS AFTER POLYA GASTRECTOMY.

/in a change in symptoms or in the cardiographic and cardiovascular findings.

DISCUSSION.

As noted in the review of the literature, opinion is divided about the significance of the changes in the blood sugar in causing "dumping" symptoms after a meal. Some believe that hyperglycaemia is primarily responsible for their occurrence (Glaessner, 1945: Winklestein, 1947); others believe that it is coincidental (Schwartz et al., 1942: Schechter and Necheles, 1949). The present study has failed to detect a relationship between the level of blood sugar and the onset of "dumping" symptoms following ingestion of glucose in postgastrectomy patients.

Changes in the level of sugar in the blood have also been held responsible for some of the cardiovascular changes which follow a glucose meal in patients after partial gastrectomy. In the previous section, evidence was presented to show that the electrocardiographic changes which occur under these circumstances were not related to hyperglycaemia. Although Amdrup and Jørgensen (1957) found that a rapid rise in blood sugar resulted in a rise in plasma volume, while slower absorption was associated with its diminution, we have been unable to confirm that the rate of rise of blood sugar is related to the fall in plasma volume.

CONCLUSION.

The level of sugar in the peripheral blood following the ingestion of a glucose meal in patients after gastrectomy rises at an increased rate compared to patients with intact stomachs. This alteration in glucose absorption bears no relationship to the occurrence of "dumping" symptoms or to cardiovascular changes in the postgastrectomy patient.

GASTROINTESTINAL ABSORPTION OF IRON AFTER PARTIAL GASTRECTOMY.

COMPARISON OF THE POLYA AND THE BILLROTH I OPERATIONS.

GASTROINTESTINAL ABSORPTION OF IRON AFTER PARTIAL GASTRECTOMY. COMPARISON OF THE POLYA AND THE BILLROTH I OPERATIONS.

Hypochromic anaemia is common after partial gastrectomy although several authors have observed that its occurrence is less frequent after the Billroth I than after the Polya operation (Morley and Roberts, 1928: Bohmansson, 1950: Henley, 1952: Wallensten, 1954). It is believed that the higher incidence of anaemia after the gastrojejunal anastomosis results from short-circuiting of the duodenum, an important area for iron absorption. A study of the role of the duodenum in this respect has been made by comparing the absorption of radio-active iron in patients in whom a conversion of a Polya to a Billroth I gastrectomy (or vice versa) has been performed.

MATERIAL AND METHODS.

Four patients were studied. Patients A and B had a Polya gastrectomy for peptic ulcer in 1952. Severe "dumping" symptoms developed and were treated by detaching the gastro-jejunal anastomosis and joining the gastric stoma to the duodenum (Billroth I operation). The other two patients (C and D) were initially treated by a Billroth I gastrectomy in 1955 and 1956 respectively. They developed recurrent ulceration at the gastro-duodenal stoma which necessitated further operation when the anastomosis was converted into one of Polya type (gastrojejunal). Care was taken to replace any blood lost at operation by whole /

/whole blood transfusion.

Studies of iron absorption were made on each patient before and 3 - 6 months after the conversion operation. No iron therapy was given between the studies. A test dose of 5 mg. ferrous iron (as ferrous sulphate) labelled with 5 microcuries of radioactive iron (^{59}Fe)* was given along with 50 mg. ascorbic acid. The patients were fasted for 12 hours before the iron was given between 9 and 10 a.m., and nothing further was taken by mouth for 2 hours. Absorption of the radioactive iron was estimated by determining the percentage of the dose excreted in the faeces. Collections were made for 8 days or until there was less than 1 per cent of the dose present. Utilization of the isotope was measured by estimating the percentage of the dose appearing in the peripheral blood.

The radioactivity of both blood and faeces was estimated in a well-type scintillation counter with an automatic scaler (Ecko, N530A). The quantities used were approximately 5 g. aliquots of homogenised faeces and 5 ml. of venous blood haemolysed with saponin. Each determination was done in triplicate. In order to calculate accurately the total radioactivity in the blood, the volume of the blood was determined by the Evans Blue technique (Bedwell et al., 1955), the packed cell volume being /

* Specific activity 55 microcuries in 30-40 micrograms of the element: obtained from Radiochemical Centre, Amersham.

/being corrected for trapped plasma (Chaplin and Mollison, 1952) and for variation from the total body haematocrit (Chaplin et al., 1953). In addition the haemoglobin (Gibson and Harrison, 1945) and serum iron (King and Wootton, 1956) were estimated. Random samples of gastric juice were tested for free hydrochloric acid using 0.5 per cent dimethylaminoazobenzene (Töpfer's Reagent) and faeces examined for occult blood in all four patients. In patients B and C biopsy of the marrow of the iliac crest was carried out at the time of the second study of iron absorption. Three patients were examined radiologically following a barium meal before and after the operation.

RESULTS.

The absorption and utilization of iron in the four gastrectomy patients was not altered by conversion of a gastro-duodenal to a gastrojejunal anastomosis or vice versa (Table XXXIV) (Fig. 30). All patients had free acid present in the gastric juice at the time that the tests were made.

Radiological examination demonstrated that, with the Billroth I type of gastrectomy, the gastric remnant emptied more slowly and that there was less hypermotility of the small bowel than following the Polya procedure. No reflux of barium into the duodenum was noted in patients with a gastrojejunal anastomosis (Polya).

Stainable iron was found in the marrow of both patients in whom iliac crest biopsy was performed. Erythropoiesis was /

PATIENT	RADIOACTIVE IRON			
	FAECAL RECOVERY		BLOOD LEVEL	
	PERCENTAGE DOSE		PERCENTAGE DOSE	
	B I	P	B I	P
A	54	56	43	44
B	78	87	7	4
C	102	82	4	14
D	79	89	17	11
MEAN	78.3	78.5	17.8	18.3

TABLE XXXIV.

COMPARISON OF IRON ABSORPTION IN 4 PATIENTS AFTER POLYA AND BILLROTH I PARTIAL GASTRECTOMY.

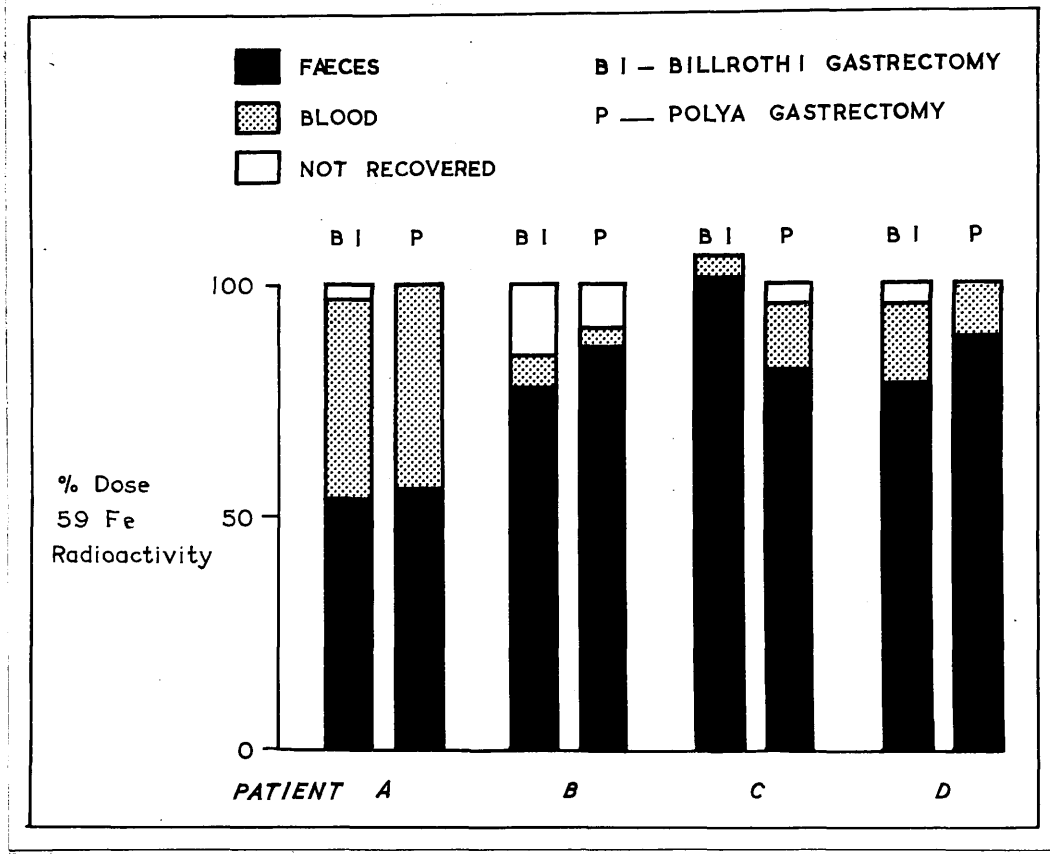


FIG. 30. COMPARISON OF ABSORPTION AND UTILISATION OF A DOSE OF 5 MG. FERROUS IRON LABELLED WITH 5 MICROCURIES RADIOACTIVE IRON (59 Fe.) TAKEN ORALLY IN 4 PATIENTS WHO HAD BOTH A POLYA AND A BILLROTH I GASTRECTOMY.

/was normoblastic.

DISCUSSION.

The mean figures for the absorption of iron in the four patients studied agree closely with those of other workers (Baird et al., 1957: Smith and Mallett, 1957).

As the order in which the operations were done was different in the two pairs of patients, this cannot have influenced the results.

A valid comparison of the results of the tests of iron absorption after each operation can be made only if there is no appreciable change in the body stores of iron. It is well known that depleted body stores stimulate absorption of iron (Moore et al., 1944: Dubach et al., 1948: Bothwell et al., 1958), an effect still found in patients after gastrectomy (Baird et al., 1957: Smith and Mallett, 1957). Replacement of blood lost at operation was thus of importance in maintaining body stores of iron.

Patients C and D developed stomal ulceration following their original gastrectomy of Billroth I type, and may have lost significant amounts of blood by chronic bleeding from the ulcerated area. One patient had a positive faecal occult blood test. Such blood loss may have led to depletion of body stores of iron and a false increase in iron absorption. In both these patients an additional part of the stomach was resected at the time of conversion of the Billroth I to a Polya gastrectomy. This measure, which was taken to prevent further /

/further ulceration by reducing the acid-producing potential of the gastric remnant would in itself tend to decrease the absorption of iron. Both these factors would give higher absorption figures during the phase of the Billroth I gastrectomy than following conversion to one of Polya type. In fact, the measured iron absorption was less during the Billroth I phase in patient D and only slightly greater in C, indicating clearly that the absorption of iron was not augmented by the contents of the gastric remnant passing through the duodenum.

Despite the known possibilities of blood loss in patients C and D, the body store of iron, as estimated by haemoglobin and serum iron levels, was the same in all patients during each phase of the experiment. In patient B, iron absorption was at the lower limits of the normal range despite a low haemoglobin of 11 g. per cent at both estimations. Although iron absorption is usually increased in anaemia (even after gastrectomy - Baird et al., 1957; Smith and Mallett, 1957), it is not directly related to the degree of anaemia but rather to the state of the body iron stores. Patient B had stainable iron in the iliac crest marrow. As gross iron depletion had not occurred, it is not unduly surprising that the absorption of iron from the intestine was not increased.

It has been reported that food may pass round into the blind duodenal loop in patients with a Polya type of /

/of gastrectomy (Kay, 1958). As radio-opaque medium did not reflux into the duodenum of the three patients studied, it is unlikely that significant amounts of iron were reaching the duodenum after Polya gastrectomy. The lack of difference between the two types of anastomosis in the amount of iron absorbed was not therefore due to failure to exclude the duodenal loop in the Polya phase.

The clinical finding that hypochromic anaemia is more common after Polya than after Billroth I gastrectomy is usually explained by the by-passing of the duodenum which occurs after the former operation, leading to diminished iron assimilation. Evidence supporting this concept was adduced by Wallensten (1958) from a study of four patients after both Billroth I and Polya type gastrectomies using serum iron concentrations over 24 hours as a measure of iron absorption. He found that three of the four patients had higher serum iron levels after the Billroth I operation but he stressed the difficulty of assessing the serum iron curves.

The present study indicates that, in the same patient, the presence or absence of the duodenum in direct continuity with the gastric remnant does not appreciably alter iron assimilation after gastrectomy. The lack of significant change in iron absorption after gastrectomy, whether of Polya or Billroth I type (Baird et al., 1957: Smith and Mallett, 1957), is further evidence against the duodenum being primarily concerned with iron /

/iron absorption. Although Baird and Wilson (1959) have shown a failure of increase of absorption of food iron in anaemic patients after gastrectomy, relative figures concerning the Polya and Billroth I procedures are not available.

CONCLUSION.

Studies of iron absorption have been made on four patients with partial gastrectomies when the gastric remnant was connected to the duodenum and when it was anastomosed to a loop of jejunum. By-passing of the duodenum did not affect the absorption of inorganic iron.

GENERAL SUMMARY.

GENERAL SUMMARY.

Like all fashions, those in surgical treatment change although their duration is longer than in some other spheres. In the operative treatment of peptic ulcer, partial gastrectomy has held the field for some years but is now beginning to lose its eminent position. This commencing decline in popularity is in no small way due to the occurrence of unpleasant sequelae to gastrectomy, of which the "dumping" syndrome and hypochromic anaemia cause greatest incapacity. This Thesis has described the work which has been undertaken to investigate the alterations in physiology which occur after gastrectomy and which may be responsible for these syndromes.

"DUMPING" SYNDROME.

The many reports on this syndrome reflect its importance to patient and surgeon. There is little agreement about the pathogenesis of these symptoms although most authors believe that the rapid passage of food into the small bowel is a primary factor involved.

The symptoms included in the definition of "dumping" have been enumerated and attention has been drawn to their high incidence (46 per cent) in a group of ninety-six patients who had partial gastrectomy for peptic ulcer in 1952. Symptoms persisted in about half of the patients for the 6 years of the follow-up /

/follow-up although during this time their severity tended to become less. In most cases symptoms began soon after operation but in about one fifth their onset was delayed from 1 to 4½ years after gastrectomy. The wide range of incidence of "dumping" in published reports has been discussed. The suggestion in the literature that iron deficiency plays a part in the aetiology of the syndrome has not been confirmed by a study of the haemoglobin levels in patients with and without "dumping" symptoms.

Gastrointestinal motility has been studied in thirty-five patients after operation. Changes consisting mainly of an increased rate of gastric emptying with hypermotility of the small bowel have been noted. No jejunal distension occurred. Gastric emptying was rapid in most cases whether "dumping" symptoms occurred or not. In three patients with initial rapid gastric emptying "dumping" symptoms developed and persisted despite slowing down of the rate of emptying with about half of the meal still in the gastric remnant. This indicated that while rapid gastric emptying may initiate the changes responsible for symptoms, it is not essential for their maintenance.

Although small bowel overactivity was consistently present in patients with "dumping" symptoms, hypermotility also occurred in several patients who were symptom-free. That hypermotility is not sufficient in itself to produce symptoms is demonstrated by the finding that patients in the recumbent position may still have increased motor activity but do not have /

/have "dumping" symptoms. In the cases studied "dumping" symptoms were only recorded when the patient was upright.

Indistinct outlining of the small bowel on radiographs, thought by other workers to be caused solely by dilution of hypertonic bowel contents, has been shown to be due in part to the hypermotility which spreads the opaque meal more diffusely through the gut. This blurred appearance of the small bowel was also found equally in patients with and without "dumping" symptoms.

The cardiovascular response to a glucose meal has been studied in eleven patients before and in fifty-one after operation. This response, consisting of a rise in pulse rate, a fall in plasma volume and sometimes a change in blood pressure (usually a slight increase), was significantly greater in patients after gastrectomy than in those with intact stomachs. No direct correlation has been found between the degree of these changes and the presence or absence of "dumping" symptoms. A subcutaneous injection of adrenaline elicited similar responses to those following a meal of glucose. Sympathetic overactivity may thus play a part in the production of the cardiovascular changes.

Although conversion from Polya to Billroth I gastrectomy appears to benefit patients with "dumping" symptoms, in this study neither subjective nor cardiovascular changes were affected, despite a somewhat slower rate of gastric emptying following conversion.

Cardiovascular changes following oral glucose also /

/also occurred after gastrojejunostomy with vagotomy. This indicated that intact vagus nerves were not required for production of the changes.

Lying down not only prevented the onset of symptoms but also diminished the extent of the cardiovascular changes which follow the ingestion of glucose in patients after gastrectomy.

The effect of a glucose meal on the electrocardiogram was studied on ninety-six occasions in fifty-two patients. Similar changes, consisting mainly of flattening of the T wave were noted before and after operation. They were more frequent and more marked in those patients treated by partial gastrectomy. In these latter patients the electrocardiographic changes were not consistently associated with either "dumping" symptoms or the degree of cardiovascular change. Further experiments which were carried out to elicit the cause of the electrocardiographic changes indicated that they were due primarily to relative overactivity of the sympathetic nervous system with an additional haemodynamic factor.

The level of sugar in the peripheral blood was estimated at 15 minute intervals following a glucose meal in forty patients after Polya gastrectomy. The rapidity with which it rose bore no relationship to the occurrence of "dumping" symptoms or to changes in the cardiovascular system.

It has been observed clinically that many of the symptoms and signs of the "dumping" syndrome are similar to the effects of stimulation of the sympathetic nervous system, for example, dizziness /

/dizziness,sweating, pallor, tachycardia and a slight rise in blood pressure. While in the studies reported in this Thesis a connection has not been demonstrated between the alterations in physiological response and the severity of the symptoms, it was noticeable that these alterations were frequently characteristic of a state of overactivity of the sympathetic nervous system. Taking these factors into account, the following hypothesis is advanced to account for the changes observed following the ingestion of a meal by a patient after gastrectomy (Fig. 31).

The small size of the gastric remnant and its direct anastomosis to the small bowel allows ingested food to enter the jejunum more rapidly than normal. This elicits intense hypermotility, especially if the meal is hypertonic. The hypermotility gives rise to exaggerated impulses which are conducted by the splanchnic nerves to the central nervous system. In addition, the increased motility leads to a relative accumulation of metabolites in the intestinal muscle causing a vasodilatation with loss of fluid into the muscles. Further fluid loss into the bowel lumen results from the osmotic pressure of the bowel contents. These factors reduce the circulating blood volume setting in motion the presso-receptor reflexes. These combine with the splanchnic afferent stimulation to induce a state of overactivity of the sympathetic system which is variable in degree.

This results in:-

(1) Cardioaccelerator response which gives rise to an /

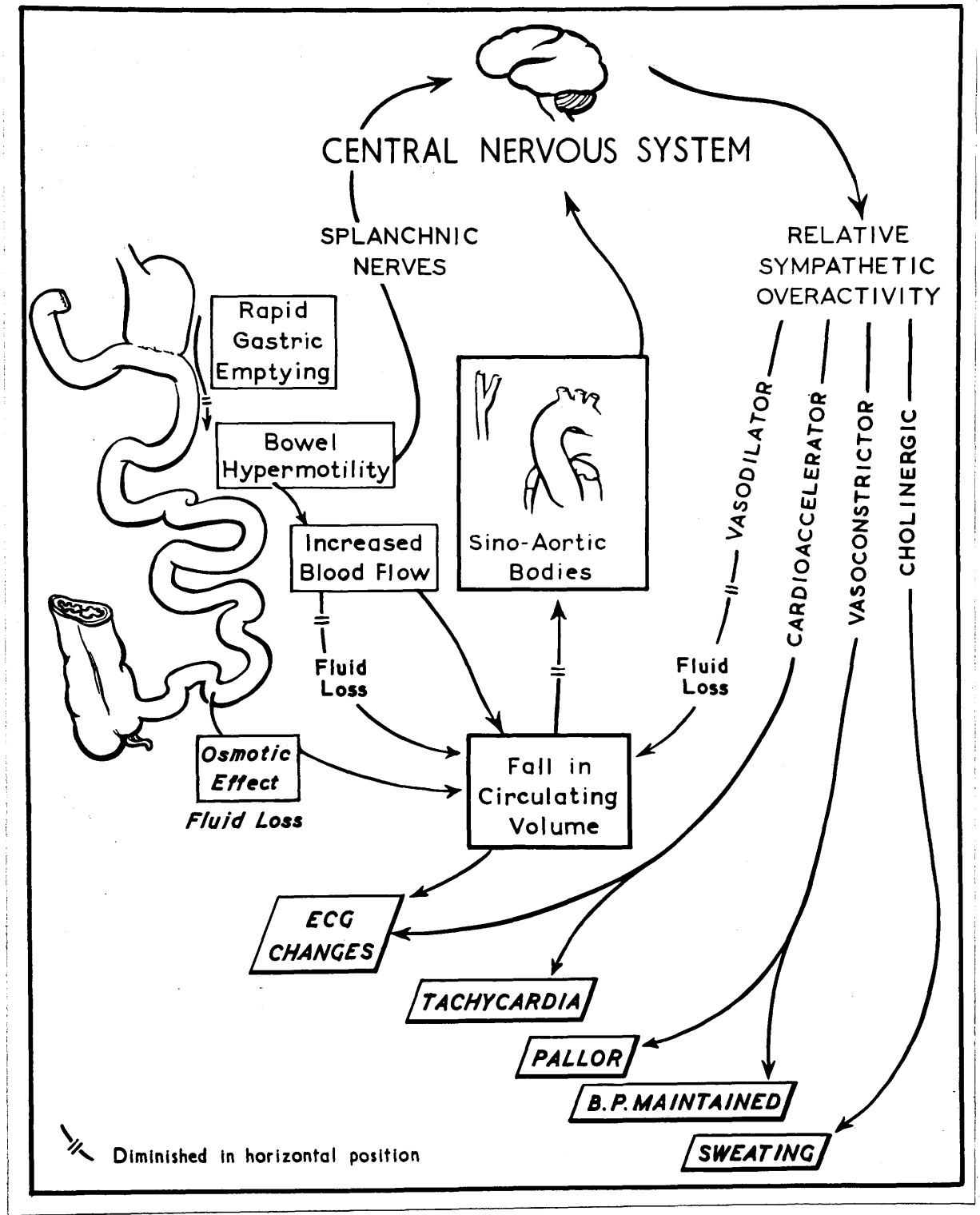


FIG. 31.

SUGGESTED MECHANISM OF CHANGES FOLLOWING INGESTION OF A MEAL IN PATIENTS AFTER GASTRECTOMY.

/an increase in pulse rate and contributes to the electrocardiographic changes. The remainder of these arise from the circulatory changes mentioned above. In susceptible patients this response causes a marked tachycardia and, not infrequently, palpitations.

2. Vasoconstrictor response. This maintains the blood pressure and when exaggerated can cause pallor. In some cases this mechanism fails and hypotension follows.

3. Sweating. In patients who have a marked overactivity of the sympathetic nervous system or in whom the end organs, namely the sweat glands, are unduly sensitive there is obvious sweating initiated by the cholinergic sympathetic fibres.

4. Muscle vasodilatation. Stimulation of the vasodilator fibres to blood vessels in muscle allows "loss" of blood in the local circulation and of fluid into the surrounding tissue spaces. This exaggerates the relative loss of circulating blood volume which is the precipitating factor of the sympathetic overactivity.

That these changes are less when the patient is horizontal is readily explained by neutralising the effect of gravity. Loss of fluid from the circulation in parts below the heart is lessened and less stimulation of presso-receptors will occur.

The sequence of events leading up to the sympathetic over-activity occurs whether the patient experiences "dumping" symptoms or not. In the patients with symptoms it must be postulated that a greater lability of the autonomic nervous /

/nervous system exists or that the "target organs" are unduly sensitive. In either case this leads not only to a response of sufficient magnitude to maintain homeostasis, as in the unaffected, but also to one which in itself causes the typical symptoms of the syndrome.

HYPOCHROMIC ANAEMIA.

Although this complication is of rarer occurrence than "dumping" symptoms in patients after gastrectomy, when it occurs it causes much lack of well-being. Its reported incidence varies but in general this appears greater after Polya than after Billroth I gastrectomy. It has been suggested that the iron deficiency is caused mainly by interference with the normal absorption of iron by the duodenum and jejunum.

This study has confirmed that the mean haemoglobin level in patients after Polya gastrectomy is lower than that of normal patients of a similar age. Anaemia was found in 32 per cent of eighty-five men followed-up 6 years after partial gastrectomy for peptic ulcer. In these patients a history of chronic blood loss was more common than in those who were anaemic.

The absorption of radioactive iron was determined in four patients who had a Polya gastrectomy converted to one of the Billroth I type or vice versa. No alteration in iron absorption followed revision of the gastrectomy, clearly indicating that the higher incidence of anaemia after the Polya operation is not due /

/due to the loss of the iron-absorbing area by-passed in the duodenum. Similar studies using organic food iron are merited.

These studies of the altered physiology of the gastrointestinal tract which results from partial gastrectomy have not produced conclusive evidence of the cause of postgastrectomy "dumping" symptoms or why they should occur in some patients and not in others. However, it is believed that they are of value in clarifying the nature of the changes which do take place as well as demonstrating the usefulness of physiological methods in clinical surgical research.

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SYNDROMES AFTER PARTIAL GASTRECTOMY.

VOLUME II.

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VOLUME II.

CONTENTS. PAGES.

PAGES.

RESULTS OF FOLLOW-UP STUDY OF PATIENTS 6 YEARS AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

AVERAGE WEIGHT IN HEALTH LBS.	WEIGHT 6 YRS. POSTOP. LBS.	DIFFERENCE LBS.	DIFFERENCE PER CENT
128	112	-16	-12.5
* 147	124	-23	-15.7
192	171	-21	-10.9
* 156	131	-24	-16.0
159	140	-19	-11.9
140	124	-16	-11.4
128	112	-16	-12.5
196	166	-30	-15.3
144	123	-21	-14.6
142	119	-23	-16.2
130	114	-16	-12.3
140	118	-22	-15.7
162	143	-19	-11.7
156	139	-17	-10.9
* 131	122	- 9	- 6.9
* 144	133	-11	- 7.7
* 124	114	-10	- 9.2
* 161	151	-10	- 6.2
136	128	- 8	- 5.9
126	115	-11	- 8.7

AVERAGE WEIGHT IN HEALTH LBS.	WEIGHT 6 YRS. POSTOP. LBS.	DIFFERENCE LBS.	DIFFERENCE PER CENT
161	151	-10	- 6.2
* 108	98	-10	- 9.3
142	130	- 8	- 5.6
108	98	-10	- 9.3
118	110	- 8	- 6.8
126	121	- 5	- 4.0
130	124	- 6	- 4.6
140	138	- 2	- 1.4
154	147	- 7	- 4.5
116	116	0	0
109	104	0	0
150	150	0	0
142	142	0	0
145	145	0	0
140	144	+ 4	+ 2.9
144	151	+ 7	+ 4.9
147	152	+ 5	+ 3.4
140	150	+10	+ 7.1
147	168	+21	+14.3
158	176	+18	+11.4
147	167	+20	+13.6

AVERAGE WEIGHT IN HEALTH BEFORE OPERATION COMPARED WITH THE WEIGHT 6 YEARS AFTER GASTRECTOMY IN 41 PATIENTS WHO HAD "DUMPING" SYMPTOMS AFTER OPERATION.

* Severe symptoms. Mean weight loss -10.1 per cent.

AVERAGE WEIGHT IN HEALTH LBS.	WEIGHT 6 YRS. POSTOP.	DIFFERENCE LBS.	DIFFERENCE PER CENT
172	154	-18	-10.5
154	131	-23	-14.9
160	134	-26	-16.3
134	106	-28	-20.9
156	126	-30	-19.2
133	112	-21	-15.8
196	136	-59	-30.0
195	133	-62	-31.6
140	125	-15	-10.7
135	119	-16	-11.9
126	114	-12	- 9.5
175	169	-11	- 6.3
158	144	-14	- 8.9
168	154	-14	- 8.3
161	148	-13	- 8.1
131	119	-12	- 9.2
127	120	- 7	- 5.5
148	137	-11	- 7.4
134	125	- 9	- 6.7
154	152	- 2	- 1.3
140	136	- 4	- 2.9
125	121	- 4	- 3.2
154	148	- 6	- 3.9
172	168	- 4	- 2.3

AVERAGE WEIGHT IN HEALTH LBS.	WEIGHT 6 YRS. POSTOP. LBS.	DIFFERENCE LBS.	DIFFERENCE PER CENT
168	161	- 7	- 4.2
138	135	- 3	- 2.2
146	145	- 2	- 1.3
133	127	- 6	- 4.5
136	131	- 5	- 3.7
132	126	- 6	- 4.5
147	144	- 3	- 2.0
126	126	0	0
126	126	0	0
161	161	0	0
164	164	0	0
145	145	0	0
126	126	0	0
168	168	0	0
140	140	0	0
163	163	0	0
129	129	0	0
134	134	0	0
140	142	+ 2	+ 1.4
134	140	+ 6	+ 4.5
140	146	+ 6	+ 4.3
128	137	+ 9	+ 7.0
118	128	+10	+ 8.5
128	142	+14	+10.9

AVERAGE WEIGHT IN HEALTH BEFORE OPERATION COMPARED WITH THE WEIGHT 6 YEARS AFTER GASTRECTOMY IN 48 PATIENTS WHO HAD NO "DUMPING" SYMPTOMS AFTER OPERATION.

MEN	AGE	Hb. g.%	PCV %	MCHC %
1	43	11.4	42.5	27
2	44	15.0	51.5	29
3	47	15.5	47.0	33
4	51	16.4	49.5	33
5	57	15.5	45.0	34
6	62	14.2	42.0	34
7	65	13.5	44.0	31
8	33	14.3	45.5	31
9	41	14.1	47.0	30
10	43	14.9	47.5	31
11	52	14.8	46.5	32
12	53	14.3	45.0	32
13	59	14.3	46.0	31
14	62	11.3	36.5	31
15	53	12.9	44.0	29
16	42	15.0	48.0	31
17	47	13.6	45.0	30
18	35	14.6	47.0	31
19	48	14.6	48.0	30
20	40	17.2	57.0	30

MEN	AGE	Hb. g.%	PCV %	MCHC %
21	47	16.4	52	32
22	38	14.6	45	32
23	40	16.2	48	34
24	52	15.3	47	33
25	55	15.8	50	32
26	58	13.6	41	33
27	60	15.3	45	34
28	49	16.2	49	33
29	51	14.5	48	30
30	40	15.8	50	32
31	50	14.0	47	30
32	52	15.5	49	32
33	60	15.1	44	34
34	45	14.9	45	33
35	45	15.0	47	32
36	52	14.7	48	31
37	56	13.2	43	31
38	48	15.4	42	37
39	37	13.1	32	41
40	45	14.3	45	32

AGE, Hb., PCV AND MCHC OF 40 "NORMAL" MEN.

MEN	AGE*	Hb. g.%	PCV %	MCHC %
1	52	14.2	47.0	30
2	60	13.3	44.0	30
3	41	13.2	45.5	29
4	49	14.2	45.0	32
5	50	13.3	41.0	32
6	42	13.8	43.0	32
7	46	14.7	48.0	31
8	42	12.8	39.0	33
9	42	13.3	43.0	31
10	47	12.2	41.0	30
11	57	9.3	36.5	26
12	31	10.9	38.0	29
13	38	12.2	44.0	28
14	41	11.3	37.0	30
15	39	12.2	41.0	30
16	43	11.8	41.0	29
17	39	11.0	38.0	29
18	32	12.3	42.0	29
19	42	13.5	46.0	29
20	47	10.5	45.0	24
21	48	12.0	43.0	29
22	43	13.3	44.0	31

MEN	AGE*	Hb. g.%	PCV %	MCHC %
23	41	15.0	47.5	32
24	27	14.2	52.0	27
25	27	14.0	44.0	32
26	42	13.2	46.0	30
27	38	14.0	44.0	32
28	35	11.9	40.0	30
29	46	12.7	42.0	30
30	47	10.9	41.5	26
31	53	10.7	42.0	25
32	25	11.5	40.0	28
33	30	11.7	40.0	29
34	40	12.7	-	-
35	41	12.4	28.0	26
36	55	13.9	45.0	31
37	30	12.8	47.0	27
38	41	12.9	43.0	30
39	37	14.0	46.0	30
40	45	13.5	44.0	31
41	37	13.3	43.0	31
42	52	12.8	45.0	28
43	45	14.2	45.5	31

AGE, Hb., PCV AND MCHC OF 43 MEN AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER WHO HAD "DUMPING" SYMPTOMS AFTER OPERATION.

* Age at operation in 1952.

MEN	AGE*	Hb. g.%	PCV %	MCHC %
1	40	13.5	44.0	31
2	43	13.7	49.0	28
3	22	12.0	44.0	27
4	25	12.0	44.0	27
5	27	13.6	-	-
6	40	12.4	44.0	28
7	19	10.0	41.0	24
8	26	14.6	46.5	30
9	42	14.6	50.0	29
10	31	11.0	-	-
11	56	13.1	45.0	30
12	53	13.1	42.0	31
13	42	14.2	47.5	30
14	39	13.9	43.0	32
15	26	14.0	45.0	31
16	41	15.4	46.0	33
17	35	12.8	42.0	30
18	31	14.2	45.0	32
19	37	12.6	41.0	31
20	53	13.5	44.0	31
21	62	8.0	31.5	25

MEN	AGE*	Hb. g.%	PCV %	MCHC %
22	52	9.7	35.0	33
23	38	12.4	44.0	28
24	48	11.1	43.0	26
25	43	12.0	44.0	27
26	42	12.2	44.0	28
27	43	12.6	43.0	29
28	44	13.5	44.0	31
29	33	13.9	46.0	30
30	31	14.7	47.5	31
31	29	12.6	42.0	30
32	44	13.8	43.0	32
33	31	14.0	44.0	32
34	45	12.2	41.0	30
35	35	14.0	45.0	31
36	46	12.8	41.0	31
37	34	12.9	41.0	31
38	34	14.1	44.0	32
39	52	13.5	43.0	31
40	27	6.7	29.0	23
41	53	12.4	46.0	27
42	48	14.0	44.0	32

AGE, Hb., PCV AND MCHC OF 42 MEN AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER WHO HAD NO "DUMPING" SYMPTOMS AFTER OPERATION.

* Age at operation in 1952.

WOMEN	AGE	Hb. g.%	PCV %	MCHC %
1	76	10.9	-	-
2	48	9.8	36.0	27
3	51	12.4	42.0	30
4	36	10.8	37.0	29
5	56	12.0	41.0	29
6	53	12.4	40.0	31
7	74	12.4	40.5	31
8	68	14.6	43.0	34
9	41	11.3	37.0	31
10*	53	10.1	41.5	24
11*	60	9.5	34.0	28

AGE, Hb., PCV AND MCHC OF 11 WOMEN AFTER PARTIAL GASTRECTOMY FOR PEPTIC ULCER.

* "Dumping" symptoms

RESULTS OF RADIOLOGICAL STUDIES.

PATIENT	GASTRIC EMPTYING MIN.	SMALL BOWEL LOSS OF DETAIL	SMALL BOWEL TIME TO CAECUM MIN.	LARGE BOWEL MOTILITY ASSESSMENT	LARGE BOWEL MOTILITY TIME (MIN.)		ONSET OF DUMPING SYNDROME
5	45	+++	15	RAPID	RECTUM	30	15
17	45	++	15	RAPID	RECTUM	90	15
22	5	+++	6	RAPID	RECTUM	60	6
23	60	++	30	RAPID	RECTUM	45	27
25	20	++	30	RAPID	DESC.COLON	60	20
29	10	++	20	MEDIUM	SPL. FLEX.	45	15
39	10	+++	14	MEDIUM	TRANS.COLON	34	20
40	10	+++	17	MEDIUM	TRANS.COLON	45	17
42	10	+++	30-45	MEDIUM	SPL. FLEX.	90	15
64	5	++	30	NORMAL	HEP. FLEX.	60	15
65	10	+++	10	RAPID	DESC.COLON	90	15
66	5	+++	12	RAPID	RECTUM	25	12
67	10	+++	20	NORMAL	HEP. FLEX.	45	15

RADIOLOGICAL FINDINGS IN 13 PATIENTS AFTER POLYA GASTRECTOMY WHO HAD SYMPTOMS FOLLOWING THE INGESTION OF 200 ml. RAYBAR/GLUCOSE MIXTURE.

PATIENT	GASTRIC EMPTYING MINS.	SMALL BOWEL		LARGE BOWEL MOTILITY	
		LOSS OF DETAIL	TIME TO CAECUM	ASSESSMENT	TIME MINS.
13	5	+++	20	MEDIUM	SPL. FLEX. 90
32	5	+++	10	MEDIUM	TRANS.COLON 90
37	5	+++	90+	-	-
41	5	++	22-45	NORMAL	HEP. FLEX. 45
43	5	+++	15	MEDIUM	TRANS.COLON 30-90
57	10	++	60	NORMAL	HEP. FLEX. 60
58	5	++	30	NORMAL	CAECUM 90
68	60	+++	20	MEDIUM	TRANS.COLON 30
69	30	+++	60+	-	-
70	5	+++	20	RAPID	DESC.COLON 90
71	5	++	30	MEDIUM	TRANS.COLON 60
72	25	+++	60+	-	-
73	60+	+++	90+	-	-
74	5	++	90+	-	-
75	10	++	90+	-	-
76	10	+++	20	NORMAL	TRANS.COLON 60

RADIOLOGICAL FINDINGS IN 16 PATIENTS AFTER POLYA GASTRECTOMY WHO HAD NO DUMPING SYMPTOMS FOLLOWING THE INGESTION OF 200 ml. RAYBAR/GLUCOSE MIXTURE.

PATIENT	GASTRIC EMPTYING	SMALL BOWEL		LARGE BOWEL MOTILITY	DUMPING SYMPTOMS
		LOSS OF DETAIL	TIME TO CAECUM		
22	60	++	.90+	-	Nil
77	90	+	90+	-	Nil

RADIOLOGICAL FINDINGS IN 2 PATIENTS AFTER BILLROTH I GASTRECTOMY FOLLOWING THE INGESTION OF 200 ML. RAYBAR/GLUCOSE MIXTURE.

PATIENT	GASTRIC EMPTYING	SMALL BOWEL		LARGE BOWEL MOTILITY			DUMPING SYMPTOMS
		LOSS OF DETAIL	TIME TO CAECUM MIN.	ASSESSMENT	TIME (MIN.)		
49	30	+++	12	MEDIUM	TRANS.COLON	60	NIL
50	5	+	90+	-	-		PRESENT
59	5	+++	20	NORMAL	ASC.COLON	60	NIL
78	5	+++	20	RAPID	DESC.COLON	40	NIL

RADIOLOGICAL FINDINGS IN 4 PATIENTS AFTER GASTROJEJUNOSTOMY WITH VAGOTOMY FOLLOWING THE INGESTION OF 200 ML. RAYBAR/GLUCOSE MIXTURE.

DETAILS OF RESPONSES TO TEST SUBSTANCES IN PATIENTS BEFORE AND AFTER OPERATION.

The results of the experiments are set out in the following pages to allow correlation of the various parameters monitored simultaneously.

EFFECT OF 100 ml. 50 PER CENT GLUCOSE.

ELEVEN PATIENTS BEFORE OPERATION.

PATIENT 1 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	122	-	70	125	85	-	-	-
15	125.8	120	-5	72	125	85	-	-	-
32	117.4	117	0	72	130	85	-	-	-
45	112.6	115	+3	68	125	85	-	-	-
60	114.1	113	-1	68	125	85	-	86	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
76	114.1	110	-4	80	125	80	+	110	-
91	111.2	108	-3	72	130	80	+	123	-
106	106.5	106	0	70	130	80	+	110	-
120	-	-	-	68	125	80	-	-	-

RHSA STANDARD (1 IN 1,000) 42.0 CPS/ML CONTROL PLASMA VOLUME 3.00 L

PATIENT 2 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC			
0	-	57.0	-	90	105	70	-	-	-
15	55.7	55.5	0	90	95	70	-	-	-
30	53.4	53.4	0	90	105	80	-	-	-
47	50.7	51.4	+1	98	105	80	-	-	-
61	49.2	50.0	+2	94	110	70	-	110	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	51.4	48.4	-6	94	105	80	+	115	-
90	46.5	46.6	0	100	100	75	++	170	-
105	44.8	45.0	0	98	105	80	+	180	-
120	-	-	-	98	105	85	-	-	-

RHSA STANDARD (1 IN 1,000) 14.2 CPS/ML CONTROL PLASMA VOLUME 2.25 L

PATIENT 3 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC			
0	-	55.0	-	70	125	95	-	-	-
15	54.5	53.2	-2	68	130	100	-	-	-
30	50.2	51.2	+2	70	135	100	-	-	-
45	49.0	49.6	+1	68	135	100	-	-	-
58	48.0	48.0	0	68	130	100	-	100	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	46.7	46.3	-1	80	135	90	+	125	-
90	44.1	44.7	+1	74	135	85	+	160	-
105	43.5	43.3	0	72	130	90	+	130	-
120	-	-	-	68	125	85	-	-	-
RHSA STANDARD (1 IN 1,000) 18.2 CPS/ML					CONTROL PLASMA VOLUME 2.98 L				

PATIENT 4 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	57.2	-	74	115	75	-	-	-
15	57.5	56.0	-1	72	105	70	-	-	-
30	54.6	54.6	0	70	110	75	-	-	-
45	52.3	53.2	+2	70	105	70	-	-	-
58	52.0	52.2	0	68	110	75	-	80	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	51.8	51.0	-2	74	105	75	-	105	-
90	50.2	49.8	-1	68	105	75	-	115	-
105	48.6	48.6	0	70	105	70	-	105	-
120	-	-	-	70	100	75	-	-	-

RHSA STANDARD (1 IN 1,000) 19.4 CPS/ML | CONTROL PLASMA VOLUME 2.88 L

PATIENT 5 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS / MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	110	-	84	120	80	-	-	-
15	109.4	109	0	84	120	80	-	-	-
30	106.2	106.5	0	82	120	80	-	-	-
45	104.5	105	0	84	120	80	-	-	-
58	104.3	104	0	82	120	80	-	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	101.4	101	0	90	115	80	-	110	-
90	99.6	99.5	0	84	115	80	-	130	-
105	96.8	97	0	86	120	85	-	125	-
120	-	-	-	82	120	80	-	-	-

RHSA STANDARD (1 IN 1,000) 37.2 CPS/ML CONTROL PLASMA VOLUME 3.04 L

PATIENT 6 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	144	-	100	115	85	-	-	-
15	143.6	142	-1	102	110	85	-	-	-
30	137.0	137	0	100	110	80	-	-	-
45	-	-	-	102	110	85	-	-	-
57	130.5	132	+1	100	115	85	-	105	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	129.7	128	+1	106	110	80	-	113	-
90	126.8	125	+1	110	100	85	-	147	-
105	122.4	122.5	0	96	95	80	-	150	-
120	-	-	-	98	100	85	-	-	-

RHSA STANDARD (1 IN 1,000) 37.9 CPS/ML

CONTROL PLASMA VOLUME 2.37 L

PATIENT 7 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	130	-	82	120	85	-	-	-
15	128.0	127	-1	80	110	80	-	-	-
30	123.2	124	+1	84	120	85	-	-	-
45	119.7	120.5	+1	82	115	85	-	-	-
58	119.2	118.5	-1	80	120	80	-	103	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	118.8	114	-4	80	115	80	-	-	-
90	115.8	111	-4	80	120	90	-	113	-
105	108.4	108	0	82	110	85	-	115	-
120	-	-	-	80	115	80	-	-	-

RHSA STANDARD (1 IN 1,000) 45.5 CPS/ML CONTROL PLASMA VOLUME 3.15 L

PATIENT 8 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC			
0	-	67.8	-	74	105	70	-	-	-
15	67.7	66.9	-1	72	105	70	-	-	-
30	65.2	65.7	+1	74	110	70	-	-	-
45	64.4	64.8	+1	72	105	70	-	-	-
60	65.1	63.8	-2	76	110	70	-	-	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	64.7	63.0	-3	80	105	70	-	-	-
90	63.9	62.3	-3	76	105	70	-	-	-
105	61.3	61.5	0	76	110	75	-	-	-
120	-	-	-	76	110	75	-	-	-
RHSA STANDARD (1 IN 1,000) 41.2 CPS/ML					CONTROL PLASMA VOLUME 3.04 L				

5 ml. of RHSA GIVEN.

PATIENT 9 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC			
0	-	137	-	68	155	95	-	-	-
15	133.9	134	0	64	150	90	-	-	-
30	128.9	132	+2	62	140	95	-	-	-
45	126.6	129	+1	62	150	90	-	-	-
60	126.7	127	0	64	140	95	-	105	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
80	123.7	124	0	62	150	90	-	105	-
93	122.0	122.5	0.	64	150	90	-	145	-
108	121.3	120	-1	64	145	90	-	143	-
120	-	-	-	66	140	90	-	-	-
RHSA STANDARD (1 IN 1,000) 49.4 CPS/ML					CONTROL PLASMA VOLUME 4.00 L				

PATIENT 10 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME EVANS BLUE		% CHANGE	PULSE BEATS / MIN	BLOOD PRESS. MM HG SYSTOLIC	DIASTOLIC	E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
0				72	120	90	-	-	-
15				70	110	85	-	-	-
30				72	120	85	-	-	-
45		2.33L	-	72	110	90	-	-	-
60				70	110	85	-	65	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75				74	105	80	-	-	-
90		2.23L	-4	76	110	80	-	103	-
105				72	115	75	-	155	-
120				80	100	75	-	-	-
RHSA STANDARD (1 IN 1,000) CPS/ML					CONTROL PLASMA VOLUME L				

PATIENT 11 BEFORE OPERATION

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME EVANS BLUE		% CHANGE	PULSE BEATS / MIN	BLOOD PRESS. MM HG SYSTOLIC	DIASTOLIC	E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
0				68	100	70	-	-	-
15				70	105	70	-	-	-
30				-	-	-	-	-	-
45		3.00L	-	68	100	75	-	-	-
60				76	100	70	-	70	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75				80	120	80	-	-	-
90		2.76L	-7	74	110	80	-	105	-
105				78	115	85	-	160	-
120				80	100	65	-	-	-
RHSA STANDARD (1 IN 1,000) CPS/ML					CONTROL PLASMA VOLUME L				

EFFECT OF 100 ml. 50 PER CENT GLUCOSE

TWENTY-THREE PATIENTS AFTER POLYA PARTIAL GASTRECTOMY WITH "DUMPING" SYMPTOMS.

(20 with RHSA for plasma volume and 3 with EB).

PATIENT 5 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	37.3	-	64	120	80	-	-	-
15	37.5	36.9	-2	62	120	80	-	-	-
30	35.2	36.5	+2	64	120	80	-	-	-
45	35.7	36.0	+1	62	120	80	-	-	-
58	36.1	35.6	-1	62	115	80	-	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	37.6	35.2	-7	80	130	85	+	140	Moderate
90	36.6	34.8	-6	78	135	85	++	142	Moderate
106	34.2	34.3	0	72	130	85	+	130	-
120	-	-	-	68	120	80	±	-	-

RHSA STANDARD (1 IN 1,000) 13.8 CPS/ML CONTROL PLASMA VOLUME 3.32 L

PATIENT 10 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	278	-	64	120	90	-	-	-
15	271.5	270	0	68	120	90	-	-	-
30	275.0	263	-4	68	130	95	-	-	-
45	256.3	256	0	68	120	90	-	-	-
60	247.4	250	+1	68	120	90	-	60	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
80	259.7	243	-7	88	110	75	+	100	-
95	258.6	236	-10	88	80	60	+	140	Moderate
110	243.5	232	-4	82	100	75	+	145	Moderate
120	-	-	-	84	110	80	-	-	-

RHSA STANDARD (1 IN 1,000) 93.4 CPS/ML CONTROL PLASMA VOLUME 3.02 L

PATIENT 12 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	65.0	-	78	130	90	-	-	-
15	64.8	63.0	-3	78	130	90	-	-	-
30	61.0	61.0	0	78	130	80	-	-	-
45	59.6	59.6	0	76	120	80	-	-	-
58	57.9	57.8	0	76	120	80	-	100	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	60.7	56.0	-8	98	140	100	+	168	Severe
90	58.6	54.4	-8	90	140	100	+	195	Severe
105	54.7	52.9	-4	84	120	90	-	223	Moderate
120	-	-	-	76	120	90	-	-	Moderate

RHSA STANDARD (1 IN 1,000) 19.6 CPS/ML CONTROL PLASMA VOLUME 2.71 L

PATIENT 13 - TEST 1 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	69.5	-	62	130	90	-	-	-
15	70.9	69.0	-3	64	125	90	-	-	-
28	67.1	68.1	+1	64	130	90	-	-	-
45	67.3	67.3	0	62	125	85	-	-	-
58	66.7	66.7	0	64	120	85	-	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	74.0	66.0	-12	80	135	90	++	140	Moderate
90	69.5	65.3	-6	86	130	85	++	210	Severe
105	66.7	64.8	-4	82	135	90	+	220	Moderate
120	-	-	-	84	130	90	-	-	-
RHSA STANDARD (1 IN 1,000) 24.2 CPS/ML					CONTROL PLASMA VOLUME 3.13 L				

PATIENT 13 - TEST 2 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	43.5	-	60	130	90	-	-	-
16	44.3	43.0	-3	60	135	90	-	-	-
29	42.8	42.5	-1	60	135	90	-	-	-
46	41.8	41.9	0	58	130	100	-	-	-
58	41.3	41.3	0	62	130	90	-	-	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
79	45.8	40.8	-12	80	155	105	++	-	Moderate
91	43.2	40.4	-7	82	150	105	++	-	Severe
109	42.9	39.9	-7	86	145	105	+	-	Moderate
120	-	-	-	80	140	95	+	-	-

RHSA STANDARD (1 IN 1,000) 17.6 CPS/ML CONTROL PLASMA VOLUME 3.17 L

PATIENT 14 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	115	-	80	95	65	-	-	-
15	112.1	112	0	72	95	65	-	-	-
30	110.2	110	0	72	90	60	-	-	-
45	108.0	108	0	70	85	60	-	-	-
60	111.8	107	-2	72	90	60	-	65	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	115.0	106	-8	80	110	75	+	-	Severe
85	115.8	104	-11	112	110	75	++	135	Severe
100	110.4	103	-7	84	90	65	+	112	Moderate
120	-	-	-	76	90	65	-	-	Moderate
RHSA STANDARD (1 IN 1,000) 38.4 CPS/ML					CONTROL PLASMA VOLUME 3.06 L				

PATIENT 15 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	122	-	84	110	80	-	-	-
15	120.0	118	-2	88	105	75	-	-	-
30	-	-	-	82	105	80	-	-	-
45	113.3	115	+2	84	105	75	-	-	-
60	113.4	113	0	82	110	75	-	65	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
79	117.6	111	-6	116	110	80	-	150	Severe
94	116.0	108	-8	122	120	80	+	165	Severe
109	108.9	105	-4	112	105	75	+	160	Moderate
120	-	-	-	110	120	80	-	-	-

RHSA STANDARD (1 IN 1,000) 39.2 CPS/ML CONTROL PLASMA VOLUME 2.89 L

PATIENT 16 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	41.0	-	62	125	80	-	-	-
15	40.4	39.8	-1	62	120	80	-	-	-
30	37.8	38.5	+2	64	130	85	-	-	-
45	36.5	37.3	+2	62	120	80	-	-	-
60	36.9	36.2	-2	64	125	80	-	75	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	37.9	35.2	-8	96	115	80	-	135	Severe
90	35.9	34.1	-5	84	120	75	-	225	Severe
105	33.6	33.1	-1	76	115	75	-	214	Moderate
120	-	-	-	64	120	80	-	-	-

RHSA STANDARD (1 IN 1,000) 9.8 CPS/ML CONTROL PLASMA VOLUME 2.15 L

PATIENT 17 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	46.6	-	62	120	80	-	-	-
15	46.4	45.6	-2	62	115	75	-	-	-
30	43.8	44.5	+2	60	115	75	-	-	-
46	42.4	43.7	+3	62	115	70	-	-	-
61	43.4	42.8	-1	60	105	70	-	120	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
78	42.0	41.7	-1	70	90	60	-	130	Severe
90	41.9	40.8	-3	72	100	65	-	175	Severe
105	39.3	39.9	+1	64	95	60	-	240	Severe
120	-	-	-	62	105	70	-	-	Moderate
RHSA STANDARD (1 IN 1,000) 7.6 CPS/ML					CONTROL PLASMA VOLUME 2.45 L				

15 ml. RHSA GIVEN.

PATIENT 18 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	135	-	72	120	90	-	-	-
18	131.0	128	-3	72	115	85	-	-	-
30	125.5	125	0	72	110	80	-	-	-
45	115.4	123	+6	70	115	85	-	-	-
60	121.0	120	0	72	110	80	-	78	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	123.8	117	-6	86	115	90	+	110	Moderate
90	126.1	114	-12	108	115	85	+	180	Moderate
105	123.9	111	-12	96	110	80	±	190	-
120	-	-	-	88	115	80	-	-	-

RHSA STANDARD (1 IN 1,000) 54.5 CPS/ML CONTROL PLASMA VOLUME 3.45 L

PATIENT 19 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	90.0	-	84	80	60	No Record	-	-
13	86.9	87.0	0	78	85	60	"	-	-
29	80.5	84.0	+4	74	80	60	"	-	-
45	85.6	81.8	-5	78	90	65	"	-	-
60	79.5	79.6	0	78	90	60	"	93	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	79.1	76.6	-3	78	90	65	No Record	120	Moderate
92	78.1	74.0	-6	80	85	65	"	120	Moderate
105	69.9	72.0	+3	78	90	60	"	235	-
120	-	-	-	80	90	65	"	-	-

RHSA STANDARD (1 IN 1,000) 31.7 CPS/ML CONTROL PLASMA VOLUME 3.17 L

PATIENT 20 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	176	-	60	100	60	No Record	-	-
15	172.4	172	0	60	100	60	"	-	-
30	172.2	168	-2	62	105	65	"	-	-
45	165.1	166	+1	64	105	60	"	-	-
60	163.4	164	0	64	100	60	"	85	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	171.9	161	-7	80	105	65	No Record	125	Moderate
90	176.5	158	-5	96	100	60	"	175	Moderate
105	158.4	156	-2	92	100	65	"	200	-
120	-	-	-	72	110	65	"	-	-

RHSA STANDARD (1 IN 1,000) 49.4 CPS/ML CONTROL PLASMA VOLUME 2.52 L

PATIENT 21 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	202	-	-	-	-	No Record	-	-
16	198.8	197	-1	66	115	75	"	-	-
30	185.5	192	+4	64	115	75	"	-	-
45	188.0	188	0	-	-	-	"	-	-
60	183.4	183	0	62	115	70	"	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
77	182.2	179	-2	64	115	75	No Record	130	-
93	180.5	175	-3	60	115	75	"	190	Moderate
105	171.5	171	0	60	115	70	"	205	Moderate
120	-	-	-	64	120	75	"	-	-

RHSA STANDARD (1 IN 1,000) 54.7 CPS/ML CONTROL PLASMA VOLUME 2.40 L

PATIENT 22 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	222	-	88	150	100	No Record	-	-
15	215.3	215	0	80	150	95	"	-	-
30	209.3	209	0	80	150	95	"	-	-
43	208.8	202	-3	70	140	100	"	-	-
65	191.1	196	+2	64	135	100	"	120	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
82	208.0	187	-11	96	160	105	No Record	120	Moderate
95	214.1	183	-17	100	155	100	"	160	Moderate
110	202.9	179	-13	96	150	100	"	200	Moderate
120	-	-	-	80	145	95	"	-	Moderate

RHSA STANDARD (1 IN 1,000) 54.7 CPS/ML CONTROL PLASMA VOLUME 2.22 L

PATIENT 23 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	52.6	-	52	125	70	-	-	-
16	51.6	51.6	0	50	125	70	-	-	-
31	50.9	50.9	0	50	120	70	-	-	-
45	48.2	49.8	+3	50	120	70	-	-	-
65	48.9	48.9	0	52	120	70	-	85	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
80	49.8	47.8	-4	60	140	75	+	75	-
93	52.0	47.2	-10	66	115	65	++	100	Moderate
109	50.1	46.4	-7	72	120	70	++	110	Moderate
120	-	-	-	62	120	70	+	-	Moderate

RHSA STANDARD (1 IN 1,000) 14.2 CPS/ML CONTROL PLASMA VOLUME 2.43 L

PATIENT 24 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	53.6	-	72	155	100	-	-	-
17	53.0	52.9	0	70	145	100	-	-	-
31	52.3	52.1	0	68	155	100	-	-	-
46	51.4	51.4	0	70	140	100	-	-	-
61	50.8	50.8	0	72	145	100	-	65	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
77	50.2	50.0	0	80	135	95	+	105	-
93	47.1	49.4	+5	80	140	95	+	150	Moderate
109	47.0	48.8	+4	74	140	90	+	185	Moderate
120	-	-	-	54	135	95	±	-	-

RHSA STANDARD (1 IN 1,000) 14.3 CPS/ML CONTROL PLASMA VOLUME 2.39 L

PATIENT 25 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML. PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	63.8	-	60	125	80	-	-	-
15	63.0	62.7	0	58	120	80	-	-	-
30	61.6	61.2	-1	60	120	80	-	-	-
45	59.8	60.0	0	62	125	85	-	-	-
60	58.7	58.9	0	64	125	85	-	93	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	62.3	57.7	-8	76	120	80	++	160	Moderate
90	62.2	56.5	-10	80	120	75	++	230	Moderate
105	55.2	55.3	0	68	125	80	+	250	Moderate
120	-	-	-	60	125	80	±	-	-

RHSA STANDARD (1 IN 1,000) 16.6 CPS/ML CONTROL PLASMA VOLUME 2.35 L

PATIENT 26 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	46.5	-	58	150	95	-	-	-
15	45.9	45.0	-2	56	140	85	-	-	-
28	43.5	43.5	0	56	135	85	-	-	-
45	40.9	41.8	+2	54	140	90	-	-	-
60	40.3	40.3	0	56	130	90	-	103	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	41.9	39.0	-7	68	140	95	++	113	Moderate
90	40.4	37.8	-7	64	140	85	++	175	Moderate
105	38.2	36.4	-5	58	140	90	+	195	Moderate
120	-	-	-	54	130	85	±	-	-

RHSA STANDARD (1 IN 1,000) 13.0 CPS/ML CONTROL PLASMA VOLUME 2.40 L

PATIENT 27 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS / MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	55.1	-	66	90	60	-	-	-
15	55.1	53.8	-2	68	90	60	-	-	-
29	52.1	52.3	0	66	80	55	-	-	-
45	50.5	51.0	+1	68	90	60	-	-	-
60	49.9	49.8	0	66	85	60	-	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	51.8	48.3	-7	94	105	75	++	125	Moderate
90	51.5	47.1	-9	84	100	70	++	145	Moderate
105	47.5	46.0	-3	80	90	60	+	180	-
120	-	-	-	70	95	65	-	-	-
RHSA STANDARD (1 IN 1,000) 19.2 CPS/ML					CONTROL PLASMA VOLUME 3.12 L				

PATIENT 28 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS / MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	190	-	76	115	80	No Record	-	-
15	188.3	183	-3	68	120	80	"	-	-
30	175.0	181	+3	66	110	80	"	-	-
45	179.0	179	0	68	110	75	"	-	-
60	177.0	177	0	74	110	75	"	113	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
78	191.3	174	-10	100	125	80	No Record	150	Moderate
93	179.6	172	-4	96	125	85	"	170	Moderate
108	170.1	170	0	88	120	80	"	160	-
120	-	-	-	72	120	80	"	-	-

RHSA STANDARD (1 IN 1,000) 62.0 CPS/ML CONTROL PLASMA VOLUME 2.84 L

PATIENT 29 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	44.5	-	62	120	90	-	-	-
17	44.1	44.2	0	60	120	90	-	-	-
30	43.8	43.9	0	56	120	85	-	-	-
46	43.3	43.5	0	56	120	80	-	-	-
61	43.2	43.2	0	56	115	80	-	85	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
78	44.3	42.9	-3	62	130	90	++	105	Moderate
93	45.7	42.5	-8	72	115	85	+	140	Moderate
108	44.4	42.1	-6	70	110	80	+	160	-
120	-	-	-	68	115	80	±	-	-

RHSA STANDARD (1 IN 1,000) 14.4 CPS/ML CONTROL PLASMA VOLUME 2.91 L

PATIENT 44 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME EVANS BLUE		% CHANGE	PULSE BEATS / MIN	BLOOD PRESS. MM HG SYSTOLIC	DIASTOLIC	E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
0				80	110	80	-	-	-
15				76	120	90	-	-	-
30				78	115	90	-	-	-
45		2.24L	-	78	115	85	-	-	-
60				80	110	80	-	88	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75				88	125	90	-	100	Moderate
90		2.09L	-7	92	120	80	-	155	Moderate
105				92	120	85	-	185	-
RHSA STANDARD (1 IN 1,000) CPS/ML					CONTROL PLASMA VOLUME L				

PATIENT 45 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME EVANS BLUE		% CHANGE	PULSE BEATS / MIN	BLOOD PRESS. MM HG SYSTOLIC	DIASTOLIC	E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
0				80	100	70	-	No Record	-
15				80	95	70	-	"	-
30				80	85	60	-	"	-
45		2.10L	-	78	85	60	-	"	-
60				78	90	60	-	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75				80	90	60	+	No Record	Severe
90		2.18L	+4	84	90	60	+	"	Severe
105		1.90L	-12	86	90	60	+	"	Moderate
RHSA STANDARD (1 IN 1,000) CPS/ML					CONTROL PLASMA VOLUME L				

PATIENT 51 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME EVANS BLUE		% CHANGE	PULSE BEATS / MIN	BLOOD PRESS. MM HG SYSTOLIC	BLOOD PRESS. MM HG DIASTOLIC	E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
0				62	110	80	-	No Record	-
15				64	115	85	-	"	-
30				68	110	80	-	"	-
45		3.05 L	-	66	110	85	-	"	-
59				64	115	85	-	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75		2.99 L	-2	90	90	70	+	No Record	Severe
90		2.68 L	-12	72	90	70	+	"	Severe
105		3.02 L	-1	68	105	80	+	"	Moderate

RHSA STANDARD (1 IN 1,000) CPS/ML CONTROL PLASMA VOLUME L

EFFECT OF 100 ml. 50 PER CENT GLUCOSE

TWENTY PATIENTS AFTER POLYA PARTIAL

GASTRECTOMY WITHOUT "DUMPING"

SYMPTOMS.

PATIENT 2 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	40.3	-	74	105	75	-	-	-
15	40.5	39.2	-3	76	100	70	-	-	-
30	36.3	36.7	+1	74	100	65	-	-	-
45	34.8	34.8	0	76	95	65	-	-	-
63	32.5	33.3	+2	74	95	65	-	110	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
74	31.5	31.5	0	78	105	75	+	140	-
93	32.0	29.9	-7	96	115	75	++	160	-
111	28.7	28.4	-1	88	95	70	++	180	-
120	-	-	-	86	95	65	+	-	-
RHSA STANDARD (1 IN 1,000) 10.3 CPS/ML					CONTROL PLASMA VOLUME 2.30 L				

PATIENT 3 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	46.5	-	64	120	80	-	-	-
15	46.8	45.6	-3	66	120	80	-	-	-
30	44.4	44.4	0	62	125	85	-	-	-
45	43.2	43.8	+1	64	115	85	-	-	-
58	42.6	43.2	+1	68	110	75	-	85	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	46.2	42.4	-10	112	120	80	++	158	-
90	44.4	41.4	-7	110	120	80	++	197	-
105	40.8	40.5	-1	92	120	80	+	165	-
120	-	-	-	78	115	75	-	-	-
RHSA STANDARD (1 IN 1,000) 16.7 CPS/ML					CONTROL PLASMA VOLUME 3.28 L				

PATIENT 4 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	41.9	-	62	110	70	-	-	-
15	42.0	41.2	-2	60	105	70	-	-	-
30	40.2	40.6	+1	64	105	70	-	-	-
45	40.4	40.1	-1	62	105	70	-	-	-
58	39.6	39.8	+1	62	110	70	-	100	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
74	41.3	39.2	-5	72	110	70	-	130	-
89	40.8	38.8	-5	72	120	80	+	150	-
104	38.2	38.2	0	68	120	90	+	130	-
120	-	-	-	66	105	70	-	-	-
RHSA STANDARD (1 IN 1,000) 12.8 CPS/ML					CONTROL PLASMA VOLUME 2.74 L				

PATIENT 6 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	44.2	-	86	120	85	-	-	-
15	44.7	43.5	-3	84	115	85	-	-	-
30	41.9	42.6	+2	82	120	80	-	-	-
45	41.6	41.9	0	80	110	85	-	-	-
58	41.3	41.3	0	80	120	85	-	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	42.2	40.5	-4	88	125	85	+	120	-
90	39.8	39.8	0	86	120	85	++	90	-
105	39.5	39.1	-1	88	120	90	++	90	-
120	-	-	-	82	115	75	+	-	-
RHSA STANDARD (1 IN 1,000) 15.3 CPS/ML					CONTROL PLASMA VOLUME 3.30 L				

PATIENT 7 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	60.7	-	80	115	85	-	-	-
15	59.6	59.2	-1	82	110	80	-	-	-
30	57.6	57.9	+1	80	115	80	-	-	-
45	56.3	56.6	+1	80	110	80	-	-	-
60	55.6	55.2	-1	78	105	75	-	105	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	56.5	54.0	-5	88	110	80	-	137	-
90	55.5	52.8	-5	90	110	70	+	120	-
105	52.7	51.5	-2	84	110	70	+	120	-
120	-	-	-	80	105	65	-	-	-

RHSA STANDARD (1 IN 1,000) 20.3 CPS/ML CONTROL PLASMA VOLUME 3.01 L

PATIENT 11 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	78.5	-	54	105	70	-	-	-
15	76.8	76.5	0	52	110	70	-	-	-
30	74.7	74.7	0	50	100	70	-	-	-
45	73.5	73.3	0	54	105	70	-	-	-
60	71.6	71.3	0	50	105	70	-	105	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
81	74.6	70.0	-7	54	105	70	-	120	-
93	74.5	68.5	-9	52	110	70	-	135	-
108	72.4	67.0	-8	52	105	75	-	180	-
120	-	-	-	56	105	70	-	-	-
RHSA STANDARD (1 IN 1,000) 28.0 CPS/ML					CONTROL PLASMA VOLUME 3.22 L				

PATIENT 30 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	48.9	-	64	150	90	-	-	-
18	49.0	47.2	-5	64	150	90	-	-	-
25	46.0	46.5	+1	62	150	85	-	-	-
41	44.4	45.1	+1	62	150	90	-	-	-
56	44.2	44.2	0	66	150	90	-	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
70	45.6	42.8	-7	82	145	85	++	130	-
83	42.0	41.8	0	84	145	85	++	223	-
100	40.4	40.4	0	80	145	85	+	240	-
120	-	-	-	74	140	85	-	-	-
RHSA STANDARD (1 IN 1,000) 19.7 CPS/ML					CONTROL PLASMA VOLUME 3.62 L				

PATIENT 31 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	67.5	-	76	120	90	-	-	-
17	65.5	65.5	0	72	115	85	-	-	-
35	61.1	63.3	+3	74	110	75	-	-	-
45	62.5	62.5	0	72	110	75	-	-	-
58	61.0	61.0	0	70	115	75	-	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
74	63.4	59.2	-7	94	115	80	++	150	-
89	58.2	57.8	-1	92	110	75	++	148	-
104	58.5	56.2	-4	86	120	80	+	140	-
120	-	-	-	86	115	75	+	-	-
RHSA STANDARD (1 IN 1,000) 18.9 CPS/ML					CONTROL PLASMA VOLUME 2.53 L				

PATIENT 32 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	57.5	-	80	140	95	-	-	-
20	55.2	55.0	0	84	130	80	-	-	-
32	53.7	53.5	0	88	130	85	-	-	-
45	51.8	52.0	0	84	125	75	-	-	-
58	50.8	50.5	-1	86	125	75	-	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
73	51.3	49.0	-5	90	130	85	+	130	-
88	49.0	47.5	-3	94	125	85	++	185	-
102	46.2	46.0	0	96	130	90	+	215	-
120	-	-	-	88	130	85	±	-	-

RHSA STANDARD (1 IN 1,000) 24.2 CPS/ML CONTROL PLASMA VOLUME 3.79 L

PATIENT 33 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	66.0	-	80	105	70	No Record	-	-
15	68.4	65.8	-2	80	115	75	"	-	-
33	64.4	65.1	+1	80	110	70	"	-	-
49	64.7	64.9	0	78	115	70	"	-	-
60	64.6	64.5	0	80	115	70	"	105	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	64.8	64.0	-1	96	110	70	No Record	140	-
90	63.0	63.8	+1	96	115	75	"	170	-
105	64.4	63.5	-3	94	110	70	"	195	-
120	-	-	-	98	110	70	"	-	-

RHSA STANDARD (1 IN 1,000) 20.5 CPS/ML CONTROL PLASMA VOLUME 2.93 L

PATIENT 34 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS / MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	26.8	-	88	105	75	-	-	-
15	26.3	26.2	0	86	105	70	-	-	-
30	25.7	25.7	0	80	100	70	-	-	-
45	24.3	25.1	+3	80	110	70	-	-	-
60	24.1	24.5	+1	84	105	70	-	75	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
78	26.5	23.9	-11	108	110	70	++	270	-
93	23.9	23.4	-2	120	120	75	++	235	-
108	23.1	23.0	0	106	110	65	++	230	-
120	-	-	-	96	110	65	+	-	-

RHSA STANDARD (1 IN 1,000) 7.6 CPS/ML CONTROL PLASMA VOLUME 2.55 L

PATIENT 35 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	71.6	-	62	160	100	-	No Record	-
15	73.6	70.2	-5	60	160	95	-	"	-
30	68.6	68.8	0	62	150	90	-	"	-
45	66.9	67.0	0	62	150	90	-	"	-
60	65.4	65.6	0	58	150	90	-	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
78	67.6	64.2	-5	74	160	100	-	No Record	-
93	66.1	63.0	-4	72	160	100	-	"	-
108	65.3	61.8	-5	72	145	90	-	"	-
120	-	-	-	74	145	85	-	"	-
RHSA STANDARD (1 IN 1,000) 26.4 CPS/ML					CONTROL PLASMA VOLUME 3.32 L				

PATIENT 36 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	197	-	68	100	60	No Record	No Record	-
15	196	192	-2	70	110	70	"	"	-
30	189	189	0	-	-	-	"	"	-
45	185	187	+1	76	110	65	"	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
60	212	185	-14	108	130	75	No Record	No Record	-
75	200	182	-10	120	115	70	"	"	-
90	180	180	0	108	120	75	"	"	-
105	-	-	-	84	110	65	"	"	-

RHSA STANDARD (1 IN 1,000) 63.0 CPS/ML CONTROL PLASMA VOLUME 2.83 L

PATIENT 37 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	55.4	-	64	100	65	-	-	-
15	52.3	52.0	0	62	100	60	-	-	-
32	47.3	48.0	+1	60	100	60	-	-	-
46	44.1	45.0	+2	62	100	60	-	-	-
60	42.5	42.2	-1	60	90	60	-	65	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
79	42.5	39.0	-8	80	85	60	++	100	-
95	39.2	36.6	-7	100	100	70	+	150	-
111	30.8	33.4	+7	88	110	70	+	150	-
120	-	-	-	92	100	70	-	-	-
RHSA STANDARD (1 IN 1,000) 18.6 CPS/ML					CONTROL PLASMA VOLUME 3.03 L				

PATIENT 38 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	64.2	-	64	130	85	-	-	-
15	63.4	63.0	-1	68	135	90	-	-	-
30	61.7	61.8	0	68	125	85	-	-	-
45	-	-	-	64	130	80	-	-	-
60	59.1	59.2	0	64	130	80	-	75	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	62.5	58.5	-7	84	135	90	++	95	-
90	60.8	57.3	-6	90	130	80	++	140	-
105	57.3	56.2	-2	86	130	80	++	150	-
120	-	-	-	92	130	80	+	-	-
RHSA STANDARD (1 IN 1,000) 22.9 CPS/ML					CONTROL PLASMA VOLUME 3.21 L				

PATIENT 39 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	37.5	-	70	120	80	-	-	-
15	37.1	37.1	0	70	115	80	-	-	-
33	36.9	36.9	0	74	105	75	-	-	-
46	36.5	36.7	0	72	105	80	-	-	-
55	36.4	36.5	0	72	105	80	-	100	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
73	38.5	36.2	-6	82	125	75	+	140	-
90	37.1	35.8	-4	90	130	75	+	180	-
105	34.7	35.4	+2	86	130	75	+	200	-
120	-	-	-	88	125	80	±	-	-

RHSA STANDARD (1 IN 1,000) 17.3 CPS/ML CONTROL PLASMA VOLUME 4.15 L

PATIENT 40 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	PULSE BEATS/MIN	BLOOD PRESS. MM HG SYSTOLIC	DIASTOLIC	E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
0	-	85.0	-	84	110	70	-	-	-
15	82.7	81.5	-1	74	105	60	-	-	-
30	77.5	77.5	0	76	100	60	-	-	-
45	71.3	74.0	+4	74	100	60	-	-	-
58	72.0	71.4	-1	78	100	60	-	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	73.0	67.8	-8	92	110	60	++	125	-
90	71.7	64.8	-11	94	120	70	++	150	-
105	62.1	61.7	-1	102	120	70	+	200	-
120	-	-	-	88	115	70	+	-	-
RHSA STANDARD (1 IN 1,000) 11.5 CPS/ML					CONTROL PLASMA VOLUME 2.70 L				

20 ml. RHSA GIVEN

PATIENT 41 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS / MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	75.1	-	60	105	60	-	-	-
15	74.1	74.2	0	60	100	60	-	-	-
30	73.6	73.5	0	56	100	60	-	-	-
42	72.0	72.7	+1	58	105	65	-	-	-
60	72.1	71.9	0	60	110	65	-	70	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
80	75.1	70.9	-6	64	110	75	+	140	-
90	74.9	70.0	-7	68	135	80	+	180	-
105	70.0	69.2	-1	64	135	70	+	175	-
120	-	-	-	60	115	70	-	-	-

RHSA STANDARD (1 IN 1,000) 23.4 CPS/ML CONTROL PLASMA VOLUME 2.81 L

PATIENT 42 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS / MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	85.0	-	62	110	75	-	-	-
15	83.2	83.1	0	60	110	70	-	-	-
31	81.0	81.0	0	62	115	75	-	-	-
45	78.1	79.2	+1	58	120	80	-	-	-
60	78.1	77.3	-1	60	110	75	-	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
76	-	-	-	68	120	80	+	150	-
92	75.5	73.8	-2	68	130	75	++	175	-
108	73.2	72.0	-2	64	120	70	++	175	-
120	-	-	-	58	110	70	+	-	-

RHSA STANDARD (1 IN 1,000) 30.0 CPS/ML CONTROL PLASMA VOLUME 3.18 L

PATIENT 43 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS / MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	63.0	-	70	120	80	-	-	-
15	60.6	60.2	-1	68	120	80	-	-	-
30	57.9	58.0	0	68	120	80	-	-	-
45	54.8	55.8	+2	66	115	80	-	-	-
64	55.6	54.2	-2	68	120	80	-	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
79	56.5	53.3	-6	74	130	85	+	105	-
95	56.6	52.8	-7	88	125	85	+	150	-
111	55.4	52.1	-6	82	120	75	+	205	-
120	-	-	-	80	120	80	±	-	-
RHSA STANDARD (1 IN 1,000) 20.1 CPS/ML					CONTROL PLASMA VOLUME 2.89 L				

EFFECT OF 100 ml. 50 PER CENT GLUCOSE

SEVEN PATIENTS AFTER BILLROTH I

PARTIAL GASTRECTOMY.

PATIENT 14 AFTER BILLROTH I GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	62.2	-	68	100	70	No Record	-	-
15	60.5	60.5	0	-	-	-	"	-	-
30	59.8	59.8	0	68	90	60	"	-	-
45	58.7	58.7	0	68	95	60	"	-	-
60	53.8	57.6	+7	68	90	60	"	138	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
77	63.1	54.3	-16	84	95	65	No Record	145	Severe
90	62.8	53.0	-19	140	100	65	"	190	Severe
105	61.4	52.1	-14	120	110	70	"	196	Moderate
120	-	-	-	96	100	70	"	-	-

RHSA STANDARD (1 IN 1,000) 22.7 CPS/ML CONTROL PLASMA VOLUME 3.30 L

PATIENT 15

AFTER BILLROTH I GASTRECTOMY

TEST SUBSTANCE

100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	101.7	-	74	120	80	-	-	-
15	100.8	100.8	0	78	125	85	-	-	-
30	96.7	99.6	+3	80	120	80	-	-	-
45	98.2	97.6	-1	80	130	90	-	-	-
60	95.8	95.8	0	82	115	85	-	105	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	98.5	94.0	-5	96	135	85	++	145	Severe
90	94.1	92.2	-2	126	145	90	++	195	Severe
105	90.0	90.4	0	126	125	85	+	140	Moderate
120	-	-	-	100	120	80	-	-	-
RHSA STANDARD (1 IN 1,000) 36.3 CPS/ML					CONTROL PLASMA VOLUME 3.20 L				

PATIENT 22 AFTER BILLROTH I GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	66.0	-	72	115	80	-	-	-
16	63.9	64.1	0	72	110	75	-	-	-
30	63.4	62.2	-1	68	115	80	-	-	-
45	60.5	60.3	0	70	115	75	-	-	-
59	58.8	58.8	0	68	110	75	-	83	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	61.7	57.0	-8	76	130	85	+	113	Moderate
90	59.3	55.3	-7	70	120	80	+	165	Moderate
105	61.6	53.8	-15	76	125	85	+	170	-
120	-	-	-	68	125	85	±	-	-
RHSA STANDARD (1 IN 1,000) 13.9 CPS/ML					CONTROL PLASMA VOLUME 1.90 L				

PATIENT 34 AFTER BILLROTH I GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	167.0	-	86	95	70	-	-	-
15	160.6	161.0	0	92	105	80	-	-	-
30	155.6	156.0	0	-	-	-	-	-	-
45	150.5	150.0	0	96	100	80	-	-	-
60	142.1	145.5	+2	96	90	70	-	100	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	143.8	141.0	-2	92	110	80	+	140	-
90	143.0	136.0	-5	110	125	85	++	195	-
105	139.7	132.0	-6	130	145	90	++	230	-
120	-	-	-	104	115	80	+	-	-

RHSA STANDARD (1 IN 1,000) 54.1 CPS/ML CONTROL PLASMA VOLUME 2.91 L

PATIENT 41 AFTER BILLROTH I GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	57.0	-	62	105	60	-	-	-
17	56.4	55.2	-2	62	100	60	-	-	-
33	52.8	53.8	+2	60	100	60	-	-	-
45	51.2	52.4	+2	60	105	60	-	-	-
58	51.8	51.2	-1	60	110	65	-	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
74	50.9	50.0	-2	72	125	75	+	120	-
88	51.5	48.8	-6	78	125	80	+	170	-
105	47.4	47.4	0	70	115	70	+	155	-
120	-	-	-	62	105	60	-	-	-
RHSA STANDARD (1 IN 1,000) 17.5 CPS/ML					CONTROL PLASMA VOLUME 2.61 L				

8.5 ml. RHSA GIVEN

PATIENT 44 AFTER BILLROTH I GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	197	-	68	120	90	-	-	-
15	193.6	194	0	70	125	80	-	-	-
30	190.0	190	0	76	120	90	-	-	-
44	186.0	187	0	78	115	80	-	-	-
58	184.0	184	0	72	115	80	-	80	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
70	192.4	182	-6	100	125	85	++	103	Moderate
80	198.6	179	-11	80	90	70	++	130	Moderate
95	186.5	176	-6	88	85	65	+	160	Moderate
120	-	-	-	80	110	75	-	-	-

RHSA STANDARD (1 IN 1,000) 55.8 CPS/ML CONTROL PLASMA VOLUME 2.55 L

PATIENT 45 AFTER BILLROTH I GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS / MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	75.0	-	82	100	70	No Record	-	-
15	72.6	73.1	+1	-	-	-	"	-	-
31	72.0	71.6	0	-	-	-	"	-	-
44	70.1	70.0	0	68	95	65	"	-	-
60	67.6	68.2	+1	76	95	70	"	80	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
76	67.2	66.8	-1	80	95	60	No Record	105	-
91	68.5	65.0	-5	78	95	60	"	105	Severe
106	70.0	63.7	-10	90	95	70	"	103	Moderate
120	-	-	-	68	105	75	"	-	-

RHSA STANDARD (1 IN 1,000) 20.5 CPS/ML CONTROL PLASMA VOLUME 2.44 L

EFFECT OF 100 ml. 50 PER CENT GLUCOSE

EIGHT PATIENTS AFTER GASTRO-

JEJUNOSTOMY WITH VAGOTOMY.

PATIENT 1 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	58.0	-	60	110	65	-	-	-
15	57.5	56.7	-1	60	115	70	-	-	-
30	55.4	55.6	0	60	120	75	-	-	-
45	54.6	54.7	0	58	110	60	-	-	-
60	54.5	53.7	-2	58	105	65	-	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	55.8	52.6	-6	74	115	75	++	150	-
90	54.1	51.5	-5	80	120	70	++	137	-
105	50.7	50.7	0	78	125	70	+	125	-
120	-	-	-	70	110	70	+	-	-
RHSA STANDARD (1 IN 1,000) 18.5 CPS/ML					CONTROL PLASMA VOLUME 3.19 L.				

10 ml. RHSA GIVEN

PATIENT 8 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	83.3	-	64	105	75	-	-	-
15	85.7	82.0	-4	66	95	65	-	-	-
30	79.0	81.0	+2	64	90	65	-	-	-
45	78.8	80.0	+2	62	95	65	-	-	-
60	78.8	79.0	-2	68	95	65	-	80	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	80.1	78.0	-3	80	105	75	-	100	-
90	76.2	76.0	0	78	100	65	-	155	-
105	72.7	75.0	+3	64	100	65	-	150	-
120	-	-	-	62	95	65	-	-	-

RHSA STANDARD (1 IN 1,000) 30.8 CPS/ML CONTROL PLASMA VOLUME 3.33 L

PATIENT 9 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	76.6	-	68	105	70	No Record	-	-
17	74.5	74.5	0	64	105	70	"	-	-
30	73.9	73.3	-1	-	-	-	"	-	-
45	70.8	71.8	+1	-	-	-	"	-	-
60	71.6	70.2	-2	64	110	75	"	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
78	74.1	68.5	-8	60	110	75	No Record	130	-
94	70.4	66.9	-5	80	110	70	"	210	-
103	65.9	65.9	0	80	110	75	"	190	-

RHSA STANDARD (1 IN 1,000) 20.5 CPS/ML CONTROL PLASMA VOLUME 2.41 L

PATIENT 46 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	62.0	-	82	120	80	-	No Record	-
15	60.6	60.8	0	80	115	75	-	"	-
30	60.4	59.8	-1	76	115	70	-	"	-
45	58.1	58.2	0	76	110	75	-	"	-
56	57.4	57.4	0	76	115	70	-	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
73	55.7	56.1	+1	80	120	75	++	No Record	-
90	54.6	54.7	0	86	120	80	++	"	-
105	53.7	53.6	0	82	115	75	+	"	-
120	-	-	-	80	110	70	±	"	-

RHSA STANDARD (1 IN 1,000) 15.2 CPS/ML CONTROL PLASMA VOLUME 2.21 L

PATIENT 47 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	15.5	-	64	110	70	-	-	-
15	15.5	15.3	-1	68	115	80	-	-	-
30	14.6	15.2	+4	66	110	80	-	-	-
45	15.3	15.1	-1	64	110	80	-	-	-
60	14.9	14.9	0	64	110	75	-	88	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
77	15.5	14.7	-5	68	115	80	+	90	Moderate
90	17.2	14.5	-18	72	110	80	+	105	Moderate
105	15.2	14.3	-5	68	115	80	+	125	Moderate
120	-	-	-	70	110	80	-	-	-

RHSA STANDARD (1 IN 1,000) 5.30 CPS/ML CONTROL PLASMA VOLUME 3.07 L

PATIENT 48 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	89.0	-	72	100	80	No Record	-	-
15	86.7	86.2	-1	-	-	-	"	-	-
30	84.1	84.3	0	66	100	75	"	-	-
45	81.9	81.5	0	68	90	65	"	-	-
60	77.6	78.9	+2	68	90	65	"	85	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	81.9	77.0	-6	80	90	65	No Record	105	-
90	74.7	74.8	0	76	70	?	"	160	Moderate
105	72.4	72.5	0	76	75	?	"	198	Moderate
120	-	-	-	72	100	70	"	-	-

RHSA STANDARD (1 IN 1,000) 28.8 CPS/ML CONTROL PLASMA VOLUME 2.91 L

PATIENT 49 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	80.8	-	82	115	80	-	-	-
15	78.5	78.1	0	80	120	80	-	-	-
30	75.3	75.6	0	84	120	85	-	-	-
45	74.0	73.2	-1	82	120	80	-	-	-
60	70.0	71.1	+1	88	120	90	-	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	70.1	69.0	-2	96	125	85	-	138	-
90	66.1	66.9	+1	104	135	75	-	190	Moderate
105	65.2	64.7	-1	100	130	80	-	194	Moderate
RHSA STANDARD (1 IN 1,000) 25.1 CPS/ML					CONTROL PLASMA VOLUME 2.79 L				

PATIENT 50 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	36.9	-	60	100	60	-	-	-
15	36.8	36.3	-1	58	105	70	-	-	-
30	35.9	35.9	0	58	100	70	-	-	-
45	35.2	35.4	+1	56	105	65	-	-	-
58	34.8	35.0	+1	58	100	65	-	105	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	37.2	34.5	-8	68	115	65	++	145	Moderate
90	37.0	34.0	-9	70	120	70	++	165	Moderate
105	33.5	33.5	0	68	115	70	+	145	-
120	-	-	-	60	110	65	±	-	-

RHSA STANDARD (1 IN 1,000) 12.0 CPS/ML CONTROL PLASMA VOLUME 2.93 L

EFFECT OF 100 ml. 50 PER CENT FRUCTOSE

THREE PATIENTS AFTER POLYA

PARTIAL GASTRECTOMY.

PATIENT 10 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent FRUCTOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC			
0	-	84.0	-	56	100	70	No Record	-	-
17	81.7	82.6	+1	-	-	-	"	-	-
34	81.2	81.0	0	-	-	-	"	-	-
46	79.9	80.0	0	58	100	70	"	-	-
64	78.1	78.3	0	60	100	70	"	85	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
87	84.1	76.1	-11	70	70	?	No Record	112	Severe
107	78.0	74.5	-5	74	90	60	"	165	Severe
120	-	-	-	60	90	60	"	-	Moderate

RHSA STANDARD (1 IN 1,000) 28.2 CPS/ML CONTROL PLASMA VOLUME 3.02 L

PATIENT 18 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent FRUCTOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	160	-	76	130	90	No Record	-	-
15	152.9	152	0	74	120	90	"	-	-
30	154.9	148	-5	76	120	90	"	-	-
47	135.6	142	+5	76	120	90	"	-	-
60	136.7	137	+2	74	120	90	"	63	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	135.1	134	-1	72	120	90	No Record	85	-
90	132.8	130	-2	80	120	90	"	103	Moderate
105	126.1	126	0	82	110	85	"	105	Moderate
120	-	-	-	72	110	80	"	-	-

RHSA STANDARD (1 IN 1,000) 63.4 CPS/ML CONTROL PLASMA VOLUME 3.50 L

PATIENT 51 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent FRUCTOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	70.0	-	74	120	80	No Record	-	-
15	70.5	69.0	-2	72	110	80	"	-	-
34	66.9	68.0	+2	-	-	-	"	-	-
46	68.1	67.5	-1	-	-	-	"	-	-
65	65.9	66.5	+1	72	110	80	"	100	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
80	66.6	65.6	-2	96	130	90	No Record	120	-
100	69.0	64.7	-7	98	125	90	"	137	-
118	64.3	63.8	-1	100	120	80	"	145	-

RHSA STANDARD (1 IN 1,000) 26.3 CPS/ML CONTROL PLASMA VOLUME 3.38 L

EFFECT OF 100 ml. 50 PER CENT MANNITOL

TWO PATIENTS AFTER POLYA PARTIAL

GASTRECTOMY.

PATIENT 12 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent MANNITOL

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	73.0	-	76	130	90	No Record	-	-
15	72.6	71.3	-2	70	130	90	"	-	-
28	68.1	70.0	+3	72	125	85	"	-	-
45	66.7	68.0	-2	72	130	90	"	-	-
60	67.1	66.5	-1	70	135	85	"	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
77	70.5	64.7	-8	92	140	100	No Record	90	Moderate
92	-	-	-	84	135	90	"	85	Severe
107	71.4	61.5	-14	80	130	90	"	95	Moderate
120	-	-	-	72	120	80	"	-	-
RHSA STANDARD (1 IN 1,000) 14.4 CPS/ML					CONTROL PLASMA VOLUME 2.76 L				

14 ml. RHSA GIVEN

PATIENT 17 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 100 ml. 50 per cent MANNITOL

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	71.0	-	62	110	65	No Record	-	-
15	70.3	69.8	-1	66	105	65	"	-	-
39	67.4	67.7	0	66	105	65	"	-	-
50	65.9	66.6	+1	66	105	65	"	-	-
60	65.8	65.8	0	64	110	70	"	95	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
78	66.7	64.5	-4	76	90	55	No Record	80	Moderate
93	68.5	63.4	-9	80	85	55	"	105	Moderate
108	70.9	62.2	-12	72	100	65	"	105	-
120	-	-	-	68	105	70	"	-	-

RHSA STANDARD (1 IN 1,000) 17.4 CPS/ML CONTROL PLASMA VOLUME 2.20 L

EFFECT OF 120 g. PROTEIN

ONE PATIENT AFTER

POLYA PARTIAL

GASTRECTOMY.

PATIENT 35 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 120 g. PROTEIN

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	40.5	-	68	135	90	No Record	No Record	-
15	39.6	39.5	0	70	130	90	"	"	-
30	40.0	38.5	-4	72	140	90	"	"	-
45	36.5	37.8	+3	70	135	85	"	"	-
60	-	-	-	72	135	90	"	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
73	35.4	37.0	+4	90	140	90	No Record	No Record	-
88	36.1	36.1	0	94	135	90	"	"	Moderate
103	36.4	35.4	-3	80	130	85	"	"	Moderate
120	-	-	-	70	140	90	"	"	Moderate

RHSA STANDARD (1 IN 1,000) 14.7 CPS/ML CONTROL PLASMA VOLUME 3.25 L

EFFECT OF 1 MG. ADRENALINE SUBCUTANEOUSLY

FOUR PATIENTS BEFORE OPERATION.

PATIENT 52 BEFORE OPERATION

TEST SUBSTANCE 1 mg. ADRENALINE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	56.1	-	82	120	90	-	-	-
15	54.8	54.9	0	82	115	85	-	-	-
30	53.4	53.7	+1	78	125	90	-	-	-
45	52.5	52.5	0	84	115	90	-	-	-
60	51.5	51.4	0	88	115	90	-	92	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	51.0	50.2	-2	110	110	75	-	90	-
90	48.8	49.1	+1	108	120	90	+	105	Moderate
105	48.2	48.1	0	116	125	85	+	110	Moderate
120	-		-	100	125	80	-	-	-

RHSA STANDARD (1 IN 1,000) 24.2 CPS/ML CONTROL PLASMA VOLUME 3.89 L

PATIENT 53 BEFORE OPERATION

TEST SUBSTANCE 1 mg. ADRENALINE SUBCUT.

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	59.8	-	82	125	80	-	-	-
15	60.2	58.8	-1	84	120	85	-	-	-
30	58.1	57.8	+1	86	125	80	-	-	-
45	56.5	56.8	+1	80	115	75	-	-	-
63	56.0	55.5	-1	80	120	80	-	110	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
76	56.5	54.9	-3	88	130	75	++	115	Moderate
90	55.6	54.0	-3	94	130	70	+	120	Moderate
105	55.2	53.0	-4	88	130	70	+	105	-
120	-	-	-	80	115	75	-	-	-

RHSA STANDARD (1 IN 1,000) 27.6 CPS/ML CONTROL PLASMA VOLUME 4.01 L

PATIENT 54 BEFORE OPERATION

TEST SUBSTANCE 1 mg. ADRENALINE SUBCUT.

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	99.5	-	60	120	80	-	-	-
15	92.4	91.5	-1	58	125	80	-	-	-
30	89.0	89.0	0	60	120	80	-	-	-
45	84.5	86.5	+2	58	120	80	-	-	-
60	83.9	84.0	0	56	125	80	-	100	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	84.5	81.5	-4	76	125	80	++	105	-
90	83.7	79.0	-6	78	130	75	++	150	Moderate
105	80.3	76.5	-5	76	125	80	+	145	-
120	-	-	-	72	120	75	-	-	-

RHSA STANDARD (1 IN 1,000) 41.6 CPS/ML CONTROL PLASMA VOLUME 3.77 L

EFFECT OF 1 MG. ADRENALINE SUBCUTANEOUSLY

FOUR PATIENTS AFTER OPERATION.

(Polya partial gastrectomy in 3, gastro-jejunostomy with vagotomy in 1).

PATIENT 55 BEFORE OPERATION

TEST SUBSTANCE 1 mg. ADRENALINE SUBCUT.

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	55.7	-	68	120	90	-	-	-
15	54.9	54.8	0	70	125	90	-	-	-
30	54.0	54.0	0	64	125	90	-	-	-
46	53.0	53.2	0	64	125	90	-	-	-
60	53.0	52.5	-1	68	120	90	-	105	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	52.1	51.8	-1	74	145	85	+	100	Moderate
90	51.8	51.0	-2	72	140	80	+	115	-
105	50.8	50.2	-1	76	140	80	+	120	-
120	-	-	-	68	130	85	-	-	-
RHSA STANDARD (1 IN 1,000) 20.3 CPS/ML					CONTROL PLASMA VOLUME 3.28 L				

PATIENT 23 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 1 mg. ADRENALINE SUBCUT.

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	35.7	-	68	125	70	-	No Record	-
15	35.1	34.9	-1	68	120	70	-	"	-
30	34.0	34.1	0	66	125	75	-	"	-
47	33.5	33.4	0	68	120	75	-	"	-
63	32.4	32.6	+1	66	120	70	-	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	33.5	32.0	-4	90	170	90	+	No Record	Moderate
90	34.5	31.5	-10	88	160	80	+	"	Moderate
106	32.4	30.8	-5	84	150	75	+	"	-
120	-	-	-	76	140	70	±	"	-
RHSA STANDARD (1 IN 1,000) 9.52 CPS/ML					CONTROL PLASMA VOLUME 2.40 L				

PATIENT 29 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 1 mg. ADRENALINE SUBCUT.

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	31.8	-	52	110	70	-	No Record	-
15	32.3	31.3	-3	50	105	70	-	"	-
30	31.1	31.0	0	52	110	75	-	"	-
45	30.7	30.7	0	50	110	75	-	"	-
60	30.3	30.3	0	52	110	70	-	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
73	31.4	30.0	-5	72	140	80	+	No Record	Moderate
89	30.4	29.7	-2	68	140	75	++	"	Moderate
103	29.2	29.4	0	70	120	70	+	"	-
120	-	-	-	66	115	70	+	"	-

RHSA STANDARD (1 IN 1,000) 10.8 CPS/ML CONTROL PLASMA VOLUME 3.06 L

PATIENT 40 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE 1 mg. ADRENALINE SUBCUT.

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	60.0	-	80	115	70	-	No Record	-
15	58.3	58.0	0	82	110	70	-	"	-
30	54.9	55.0	0	80	105	60	-	"	-
47	51.0	51.8	+2	78	105	65	-	"	-
60	49.8	49.8	0	76	100	60	-	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
77	51.9	46.8	-11	80	115	65	++	No Record	Moderate
90	51.7	45.0	-15	86	120	60	++	"	Moderate
105	45.1	42.6	-6	90	110	70	+	"	-
120	-	-	-	86	105	60	±	"	-

RHSA STANDARD (1 IN 1,000) 18.3 CPS/ML CONTROL PLASMA VOLUME 2.71 L

PATIENT 46 AFTER GASTROJEJUNOSTOMY

TEST SUBSTANCE 1 mg. ADRENALINE SUBCUT.

TIME MINS	PLASMA VOLUME: COUNTS/SEC./ML PLASMA	PLASMA VOLUME: EXPECTED CTS/SEC/ML	PLASMA VOLUME: % CHANGE	PULSE BEATS/MIN	BLOOD PRESS. MM HG: SYSTOLIC	BLOOD PRESS. MM HG: DIASTOLIC	E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
0	-	51.0	-	62	115	65	-	No Record	-
16	50.5	50.5	0	60	110	65	-	"	-
30	49.5	49.8	+1	60	110	70	-	"	-
45	49.3	49.2	0	60	110	65	-	"	-
60	49.0	48.7	-1	58	105	65	-	"	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
78	48.3	48.0	-1	72	150	80	+	No Record	Moderate
92	47.9	47.5	-1	72	160	70	+	"	Moderate
107	46.9	47.0	0	72	150	70	+	"	-
120	-	-	-	66	125	70	-	"	-

RHSA STANDARD (1 IN 1,000) 11.5 CPS/ML CONTROL PLASMA VOLUME 2.22 L

EFFECT OF 100 ml. 50 PER CENT GLUCOSE IN RECUMBENCY

FIVE PATIENTS AFTER PARTIAL GASTRECTOMY.

(Polya in 4: Billroth I in 1).

PATIENT 16 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE GIVEN RECUMBENT 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	39.2	-	64	125	85	-	-	-
15	39.2	38.4	0	66	120	80	-	-	-
30	37.3	37.6	+1	64	115	75	-	-	-
45	36.6	36.9	+1	64	115	75	-	-	-
60	36.0	36.1	0	64	120	80	-	83	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	35.7	35.3	-1	70	120	80	-	180	-
90	35.0	34.7	-1	76	120	75	-	195	-
102	33.9	34.0	0	70	120	80	-	200	-
120	-	-	-	68	120	75	-	-	-

RHSA STANDARD (1 IN 1,000) 10.6 CPS/ML CONTROL PLASMA VOLUME 2.43 L

PATIENT 25 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE GIVEN RECUMBENT 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	55.0	-	60	130	85	-	-	-
15	55.0	53.9	-2	60	125	80	-	-	-
30	52.1	52.5	+1	60	130	85	-	-	-
45	51.5	51.4	0	58	120	80	-	-	-
60	50.3	50.3	0	56	120	80	-	80	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	49.7	49.1	-1	72	120	80	+	148	-
90	50.0	48.0	-4	66	125	75	+	158	-
105	48.0	47.0	-2	66	120	80	+	288	-
120	-	-	0	60	120	80	-	-	-

RHSA STANDARD (1 IN 1,000) 14.6 CPS/ML CONTROL PLASMA VOLUME 2.39 L

PATIENT 34 AFTER BILLROTH I GASTRECTOMY

TEST SUBSTANCE GIVEN RECUMBENT 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR MGM %	DUMPING SYMPTOMS
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC			
0	-	17.4	-	80	110	70	-	-	-
17	16.8	16.7	-1	78	105	70	-	-	-
32	16.2	16.2	0	80	110	70	-	-	-
42	15.7	15.8	+1	78	110	70	-	-	-
60	15.4	15.2	-1	78	110	70	-	90	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
77	15.0	14.7	-2	88	115	70	++	170	-
90	14.1	14.3	+1	90	110	65	++	180	-
105	14.0	13.8	-1	86	110	65	+	210	-
120	-	-	-	82	115	70	+	-	-
RHSA STANDARD (1 IN 1,000) 6.6 CPS/ML					CONTROL PLASMA VOLUME 3.14 L				

PATIENT 38 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE GIVEN RECUMBENT 100 ml. 50 per cent GLUCOSE

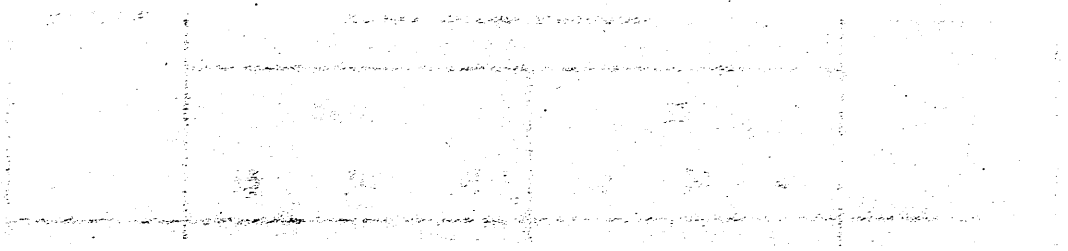
TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G CHANGE	BLOOD SUGAR	DUMPING SYMPTOMS
	COUNTS/SEC. /ML. PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC		MGM %	
0	-	43.0	-	60	135	85	-	-	-
15	42.2	42.2	0	58	140	85	-	-	-
30	40.9	41.3	+1	60	140	85	-	-	-
45	40.5	40.5	0	60	135	90	-	-	-
60	40.0	39.8	-1	56	135	85	-	80	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	40.0	39.0	-3	66	135	85	-	130	-
95	39.2	38.3	-2	66	135	85	+	180	-
105	37.6	37.6	0	66	130	80	-	205	-
120	-	-	-	60	135	85	-	-	-
RHSA STANDARD (1 IN 1,000) 15.3 CPS/ML					CONTROL PLASMA VOLUME 3.20 L				

PATIENT 43 AFTER POLYA GASTRECTOMY

TEST SUBSTANCE GIVEN RECUMBENT 100 ml. 50 per cent GLUCOSE

TIME MINS	PLASMA VOLUME			PULSE	BLOOD PRESS. MM HG		E C G	BLOOD	DUMPING
	COUNTS/SEC. /ML PLASMA	EXPECTED CTS/SEC/ML	% CHANGE	BEATS/MIN	SYSTOLIC	DIASTOLIC	CHANGE	SUGAR MGM %	SYMPTOMS
0	-	60.3	-	60	130	75	No Record	-	-
17	59.3	59.3	0	62	135	85	"	-	-
32	58.5	58.5	0	60	130	80	"	-	-
45	56.7	57.8	-2	60	130	80	"	-	-
59	56.4	56.9	+1	60	130	80	"	75	-
TEST SUBSTANCE GIVEN AT 60 MIN.									
75	55.2	56.0	+1	64	135	80	No Record	160	-
90	55.0	55.0	0	62	130	80	"	170	-
105	54.2	54.1	0	60	130	75	"	200	-
120	-	-	-	62	130	80	"	-	-

RHSA STANDARD (1 IN 1,000) 19.8 CPS/ML CONTROL PLASMA VOLUME 2.95 L



RESULTS OF COMPARISON OF TWO METHODS OF PLASMA VOLUME ESTIMATION AND OF ESTIMATED AND CALCULATED PCV FINDINGS AFTER A GLUCOSE MEAL.

PATIENT	CHANGE IN PLASMA VOLUME PER CENT MINS. AFTER GLUCOSE						"DUMPING" SYMPTOMS
	RHSA			EB			
	15	30	45	15	30	45	
2	0	- 7	- 1	- 8	- 7	- 1	NIL
15*	- 6	- 8	- 4	0	-19	+ 4	SEVERE
22*	-11	-17	-13	-10	-20	- 9	MODERATE
24	0	+ 5	+ 4	- 2	+ 3	+ 2	MODERATE
25	- 8	-10	0	-20	+ 1	-	MODERATE
26	- 7	- 7	- 5	- 7	- 8	- 5	MODERATE
37	- 8	- 7	+ 7	- 6	- 4	+ 2	NIL
42	-	- 2	- 2	0	0	+ 3	NIL

COMPARISON OF THE CHANGES IN PLASMA VOLUME AS MEASURED BY RADIOACTIVE HUMAN SERUM ALBUMEN (RHSA) AND EVANS BLUE (EB) GIVEN SIMULTANEOUSLY FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 8 PATIENTS AFTER GASTRECTOMY.

* Separate experiments with the two methods.

PATIENT	CHANGE IN PLASMA VOLUME PER CENT MINS. AFTER GLUCOSE						"DUMPING" SYMPTOMS
	RBSA			EB			
	15	30	45	15	30	45	
2	0	- 7	- 1	- 8	- 7	- 1	NIL
15*	- 6	- 8	- 4	0	-19	+ 4	SEVERE
22*	-11	-17	-13	-10	-20	- 9	MODERATE
24	0	+ 5	+ 4	- 2	+ 3	+ 2	MODERATE
25	- 8	-10	0	-20	+ 1	-	MODERATE
26	- 7	- 7	- 5	- 7	- 8	- 5	MODERATE
37	- 8	- 7	+ 7	- 6	- 4	+ 2	NIL
42	-	- 2	- 2	0	0	+ 3	NIL

COMPARISON OF THE CHANGES IN PLASMA VOLUME AS MEASURED BY RADIOACTIVE HUMAN SERUM ALBUMEN (RHSA) AND EVANS BLUE (EB) GIVEN SIMULTANEOUSLY FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 8 PATIENTS AFTER GASTRECTOMY.

* Separate experiments with the two methods.

$$\text{RED CELL VOLUME} = \frac{V \quad PCV}{(100 - PCV)}$$

V = PLASMA VOLUME

PCV = PACKED CELL VOLUME

ASSUMING THE RED CELL VOLUME DOES NOT CHANGE DURING THE PERIOD OF THE EXPERIMENT.

$$(1) \quad \frac{V_c PCV_c}{(100 - PCV_c)} = \frac{V_x PCV_x}{(100 - PCV_x)}$$

V_c & PCV_c = CONTROL VALUES

V_x & PCV_x = VALUES AFTER GLUCOSE

THE PLASMA VOLUME V_x IS ESTIMATED AND EXPRESSED AS A PROPORTION (K) OF THE CONTROL VALUE (V_c)

SUBSTITUTING $V_x = K.V_c$ IN EQUATION (1)

$$\frac{V_c PCV_c}{(100 - PCV_c)} = \frac{K.V_c PCV_x}{(100 - PCV_x)}$$

$$\frac{PCV_c}{(100 - PCV_c)} = \frac{K.PCV_x}{(100 - PCV_x)}$$

$$100\ PCV_c - PCV_c.PCV_x = 100\ K.PCV_x - KPCV_x.PCV_c$$

$$100\ PCV_c = 100\ K.\ PCV_x - KPCV_x\ PCV_c + PCV_c.PCV_x$$

$$PCV_x = \frac{100\ PCV_c}{K(100 - PCV_c) + PCV_c}$$

DERIVATION OF FORMULA FOR CALCULATING PCV FROM CONTROL PCV AND CHANGE IN PLASMA VOLUME.

PATIENT	PACKED CELL VOLUME									
	CONTROL	MINUTES AFTER GLUCOSE								
		15			30			45		
		ESTIM.	CALC.	DIFF.	ESTIM.	CALC.	DIFF.	ESTIM.	CALC.	DIFF.
1	46	47	47	0	46	47	-1.0	46	46	0
2	51	52	52.5	-0.5	-	-	-	50	51	-1.0
3	50	49	50	-1.0	49	49.5	-0.5	49	50	-1.0
4	43.5	45	44	+1.0	46	43.5	+2.5	44	43.5	+0.5
5	48	48	48	0	48	48	0	48	48	0
6	53.5	52	53	-1.0	52.5	53	-0.5	-	-	-
7	43	45	44	+1.0	44.5	44	+0.5	43	43	0
8	50	49	50.5	-1.5	49	50.5	-1.5	-	-	-
9	38	39	38	+1.0	39	38	+1.0	39	38.5	+0.5
10	48	48	49	-1.0	50	49	+1.0	-	-	-
11	41	40	41	-1.0	-	-	-	-	-	-
MEAN				-0.27			+0.17			-0.14
S.E.MEAN				±0.27			±0.42			±0.20

COMPARISON OF ESTIMATED AND CALCULATED PACKED CELL VOLUME READINGS FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 11 PATIENTS BEFORE OPERATION.

PATIENT	PACKED CELL VOLUME									
	CONTROL	MINUTES AFTER GLUCOSE								
		15			30			45		
		ESTIM.	CALC.	DIFF.	ESTIM.	CALC.	DIFF.	ESTIM.	CALC.	DIFF.
5	48	48	49.5	-1.5	46	49.5	-3.5	47	48	-1.0
10	44	45.5	46	-0.5	46	46.5	-0.5	44	45	-1.0
12	41	42	43	-1.0	42	43	-1.0	42	42	0
13	40	42.5	43	-0.5	-	-	-	44.5	42	+2.5
14	44.5	45	46.5	-1.5	45	47.5	-2.5	44	46.5	-2.5
15	42.5	43.5	44	-0.5	43	44.5	-1.5	43	43.5	-0.5
17	48	-	-	-	49	49	0	49	48	+1.0
18	50	51	51.5	-0.5	51	53.0	-2.0	51	53.0	-2.0
20	44	46	46	0	46	45.5	+0.5	44	44.5	-0.5
21	47	47	47.5	-0.5	-	-	-	45	47	-2.0
22	56	59	59	0	62	61	+1.0	59	59	0
23	41	43	42.5	+0.5	44	44	0	44	43	+1.0
24	45.5	43	45.5	-2.5	43	43.5	-0.5	43	44.5	-1.5
25	46	47.5	48	-0.5	49.5	50	-0.5	48	46	+2.0
27	43.5	44	45	-1.0	44	46.5	-2.5	43.5	44	-0.5
28	42	43.5	44.5	-1.0	43	43	0	43	42	+1.0
29	35	38.5	36	+1.5	38	37.5	+0.5	38	37	+1.0
MEAN				-0.65			-0.72			-0.32
S.E.MEAN				±0.23			±0.32			±0.30

COMPARISON OF ESTIMATED AND CALCULATED PACKED CELL VOLUME READINGS FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 17 PATIENTS AFTER GASTRECTOMY WHO HAD DUMPING SYMPTOMS AFTER THE GLUCOSE.

PATIENT	PACKED CELL VOLUME									
	CONTROL	MINUTES AFTER GLUCOSE								
		15			30			45		
		ESTIM.	CALC.	DIFF.	ESTIM.	CALC.	DIFF.	ESTIM.	CALC.	DIFF.
2	44.5	45.5	45.5	0	48	46.5	+1.5	47	44.5	+2.5
3	46	49.5	48.5	+1.0	48	48	0	46	46	0
4	42	43	43	0	-	-	-	42	42	0
6	42	41	43	-2.0	41.5	42	-0.5	41	42	-1.0
7	40	42	41.5	+0.5	43	41.5	+1.5	41	41	0
11	40	42	42	0	43.5	42.5	+1.0	44	42	+2.0
30	44	48	46	+2.0	45	44	+1.0	45	44	+1.0
31	49	51	51	0	50	50	0	-	-	-
32	41.5	42	42	0	-	-	-	-	-	-
33	43	43	43.5	-0.5	44	43	+1.0	44	43.5	+0.5
34	36	39	40.5	-1.5	37.5	38.5	-1.0	36	36	0
35	43	43	43.5	-0.5	43.5	43.5	0	45	44	+1.0
36	38	41	41.5	-0.5	40	40.5	-0.5	38	38	0
37	41	43.5	43	+0.5	44	43	+1.0	45	39	+2.5
39	32	33	33	0	32	33	-1.0	30	32	-2.0
40	50	51	52	-1.0	51	53	-2.0	51	50	+1.0
42	45	43	45	-2.0	45	45.5	-0.5	45	45.5	-0.5
43	36	37	38	-1.0	39	38	+1.0	38	38	0
MEAN				-0.19			-0.06			+0.53
S.E.MEAN				±0.26			±0.25			±0.31

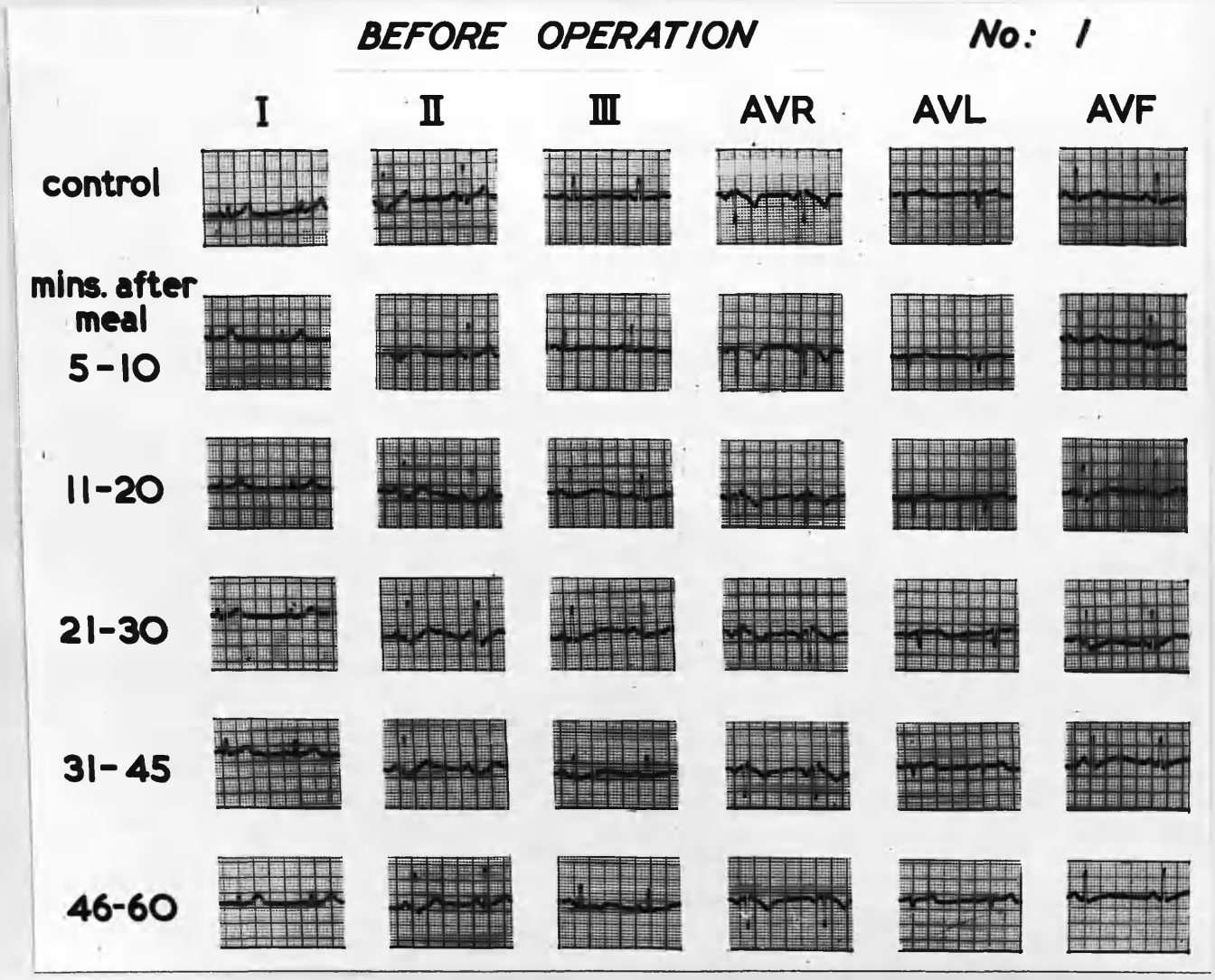
COMPARISON OF ESTIMATED AND CALCULATED PACKED CELL VOLUME READINGS FOLLOWING THE INGESTION OF 100 ml. 50 PER CENT GLUCOSE IN 18 PATIENTS AFTER GASTRECTOMY WHO HAD NO DUMPING SYMPTOMS AFTER THE GLUCOSE.

ELECTROCARDIOGRAPHIC RESULTS.

ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE.

The tracings which follow are those on which the description of the form of changes in Vol. I is based.

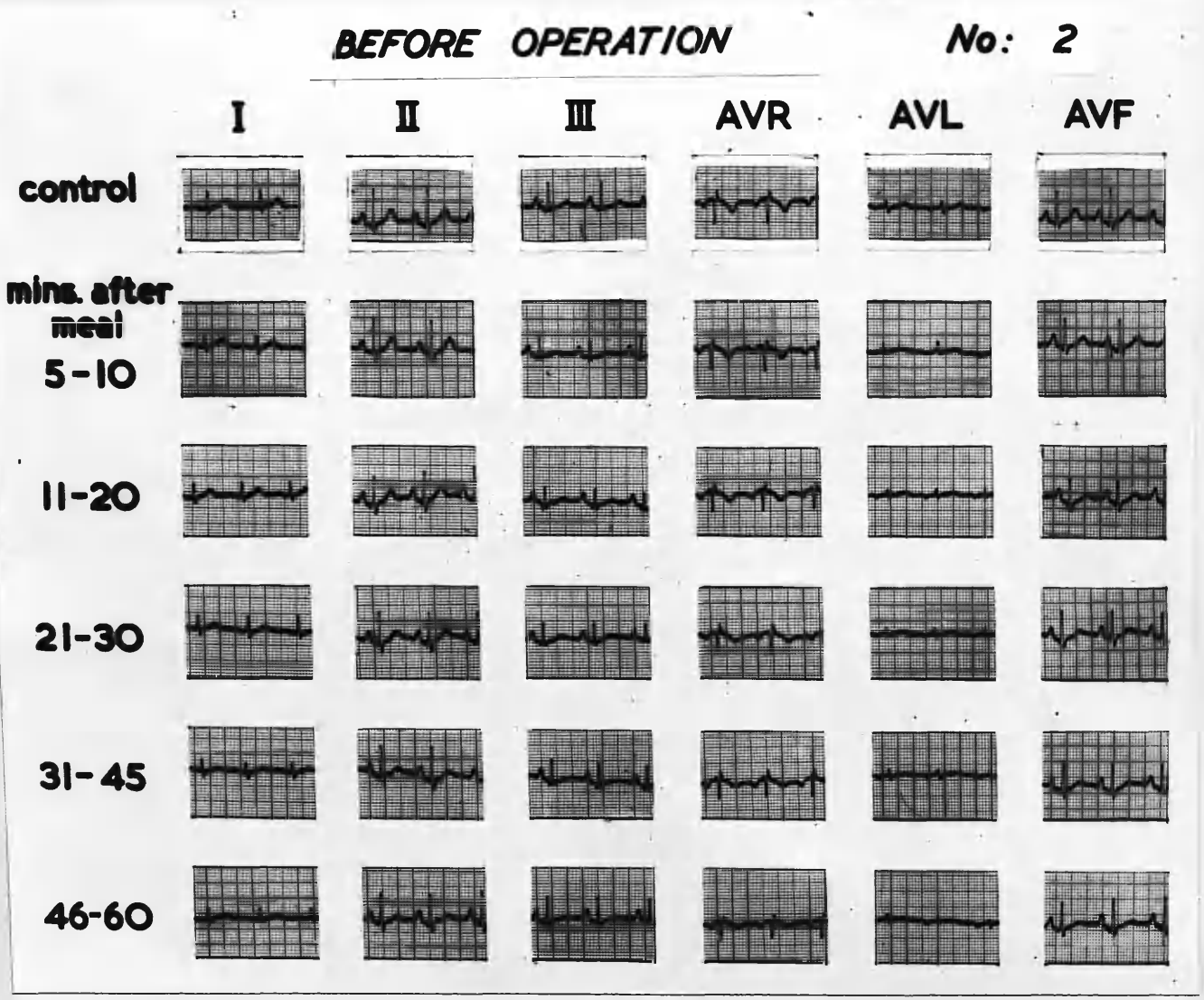
ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE IN THREE PATIENTS BEFORE OPERATION.


BEFORE OPERATION
No: 1
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

BEFORE OPERATION - EXAMPLE NO. 1.

PATIENT 1.

TRACINGS	-	Control and at 6, 11, 26, 36 and 52 mins.
HEART RATE	-	Increased from 64 to 68
P	-	No change from the control
QRS	-	Increased R II and III and decreased S aVR at 26 and 36 mins.
RT	-	ST low take off II and aVF at 11 - 36 mins.
T	-	Flattened and broadened II, aVF, biphasic III from 11 - 52 mins. Slightly flatter I from 11 - 26 mins. Less negative aVR 11, 26, and 36 mins.
U	-	Increased in II from 11 - 52 mins. Maximal changes at 36 mins.

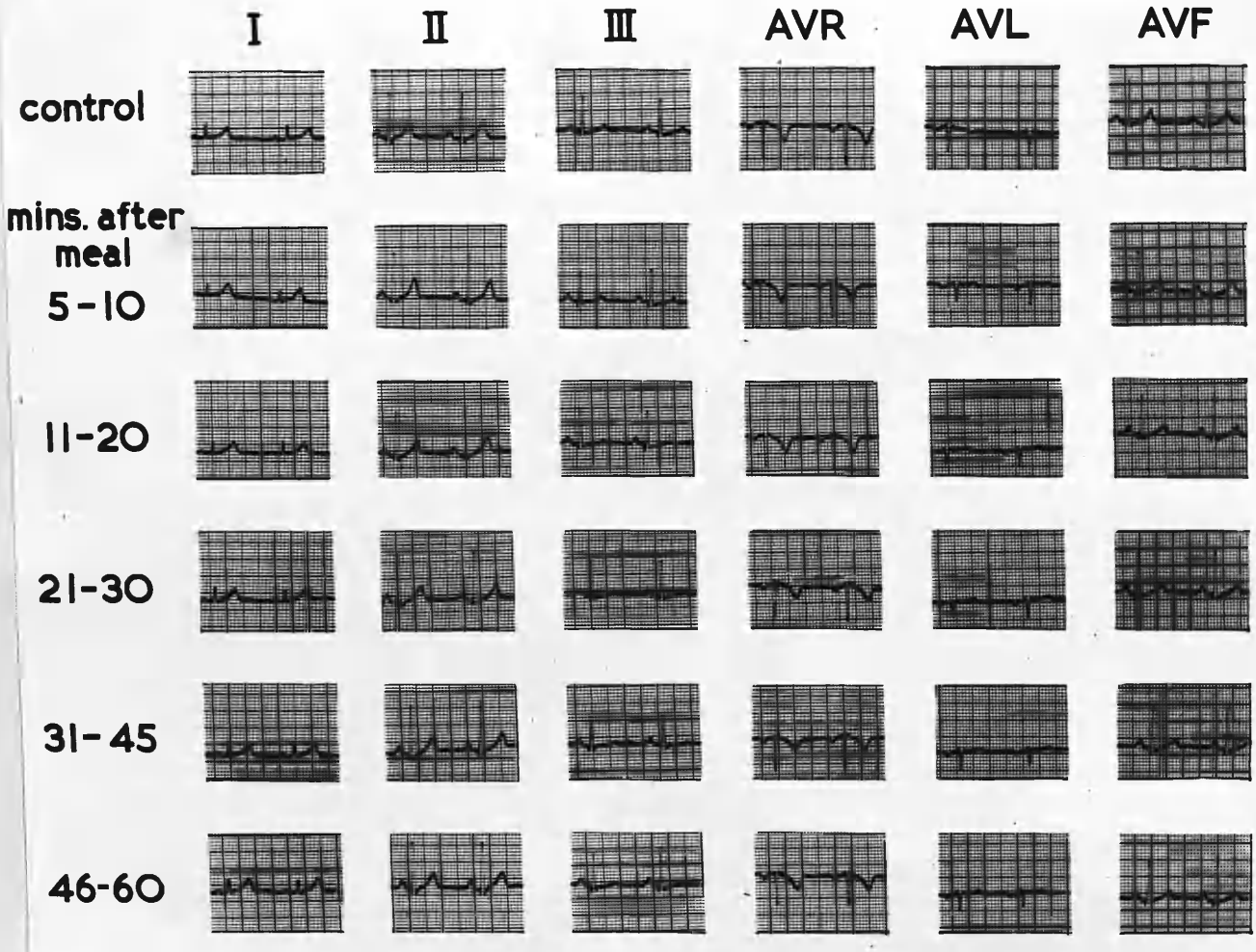

BEFORE OPERATION
No: 2
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

BEFORE OPERATION - EXAMPLE NO. 2.

PATIENT 2.

TRACINGS	-	Control and at 5, 11, 25, 33 and 60 mins.
HEART RATE	-	Increased from 90 to 100
P	-	Peaking in II and aVF from 11 - 33 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flattened and broadened in II, III and aVF from 11 - 60 mins., and in I from 25 - 60 mins. aVR less negative from 26 - 60 mins.
U	-	Increased in II and aVF from 25 - 60 mins. Maximal changes at 60 mins.

BEFORE OPERATION No: 3



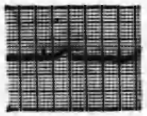
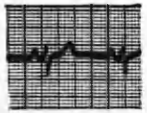
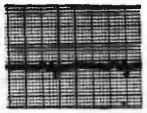
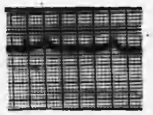
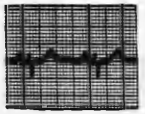
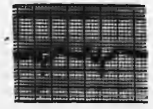
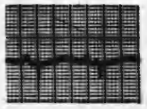
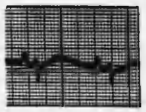
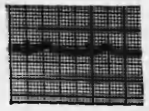
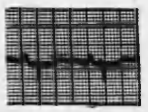
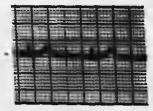
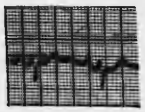
BEFORE OPERATION - EXAMPLE NO. 3.

PATIENT 3.

TRACINGS	-	Control and at 5, 17, 30, 40 and 60 mins.
HEART RATE	-	Increased from 68 to 78
P	-	No change from the control
QRS	-	No change from the control
RT	-	No change from the control
T	-	Smaller in II, III and aVF at 17 and 30 mins., and still slightly smaller aVF at 60 mins.
U	-	No change from the control
		Maximal changes at 17 mins.

ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE IN THIRTY-ONE PATIENTS AFTER A POLYA GASTRECTOMY.

AFTER POLYA GASTRECTOMY No: 1

	I	II	III	AVR	AVL	AVF
control						
mins. after meal 5-10						
11-20				NO RECORD		
21-30						
31-45						
46-60						

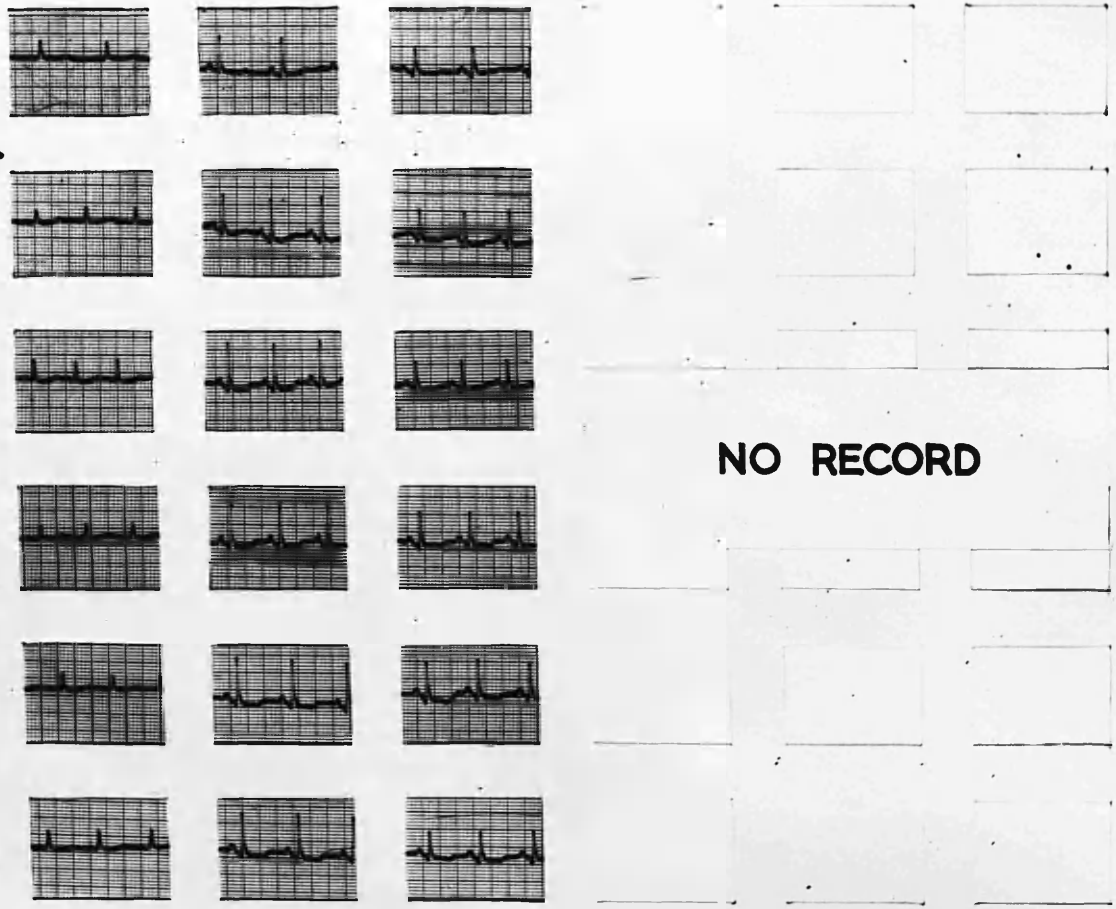
AFTER POLYA GASTRECTOMY - EXAMPLE NO. 1.

PATIENT 10.

TRACINGS	-	Control and at 5, 11, 21, 35 and 47 mins.
HEART RATE	-	Increased from 66 to 84 mins.
P	-	Peaked II and III at 21 and 35 mins.
QRS	-	Increase S III from 5 - 47 mins.
RT	-	No change from the control
T	-	Broader and flatter in II at 5 and 11 mins. Slightly flat in I at 21 mins.
U	-	Increased in II from 11 - 47 mins., and in III at 21 and 35 mins. Maximal changes at 11 mins.

AFTER POLYA GASTRECTOMY

No: 2

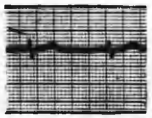
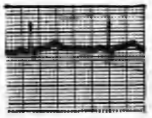
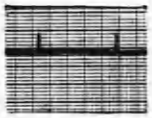
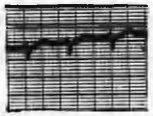
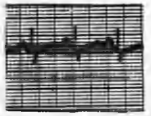
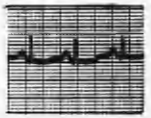
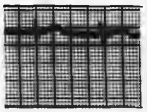
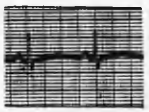
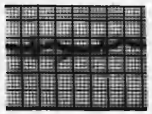
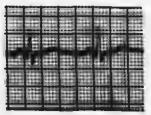
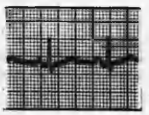
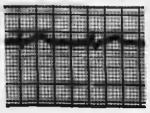
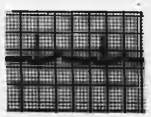
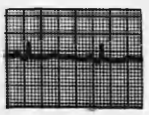
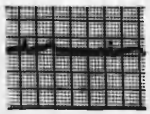
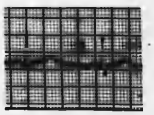
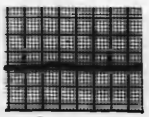


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 2.

PATIENT 34.

TRACINGS	-	Control and at 10, 20, 25, 35 and 48 mins.
HEART RATE	-	Increased from 80 to 116
P	-	Peaked in II at 20 to 60 mins.
QRS	-	R in II and III increased at 10 - 48 mins.
RT	-	ST below isoelectric line in II at 20 and 35 mins., and in III at 35 mins.
T	-	Becomes biphasic in II and inverted in III at 20 - 48 mins.
U	-	No change from the control Maximal changes at 35 mins.

AFTER POLYA GASTRECTOMY No: 3

	I	II	III	AVR	AVL	AVF
control						
mins. after meal 5-10						
11-20				NO RECORD		
21-30						
31-45						
46-60						

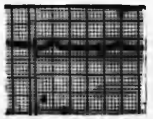
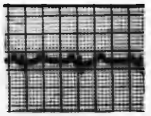
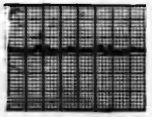
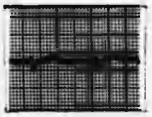
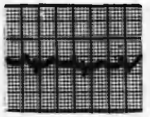
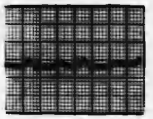
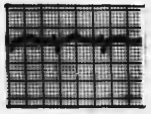
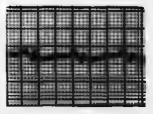
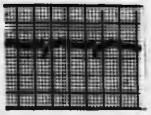
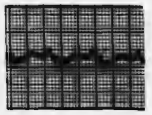
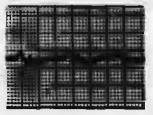
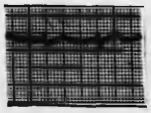
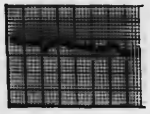
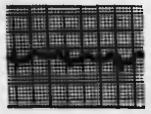
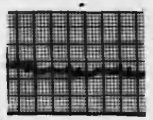
AFTER POLYA GASTRECTOMY - EXAMPLE NO. 3.

PATIENT 51.

TRACINGS	-	Control and at 7, 18, 23, 31 and 49 mins.
HEART RATE	-	Increased from 68 to 120
P	-	Peaked in II and III at 7 - 31 mins.
QRS	-	Slightly increased R in III at 7 and at 23 mins.
RT	-	ST low take off in II at 7 mins.
T	-	Broader and flatter in II at 7 - 35 mins., and biphasic in III at 7 and 18 mins.
U	-	Increased in II at 23 - 49 mins.
		Maximal changes at 7 mins.

AFTER POLYA GASTRECTOMY

No: 4

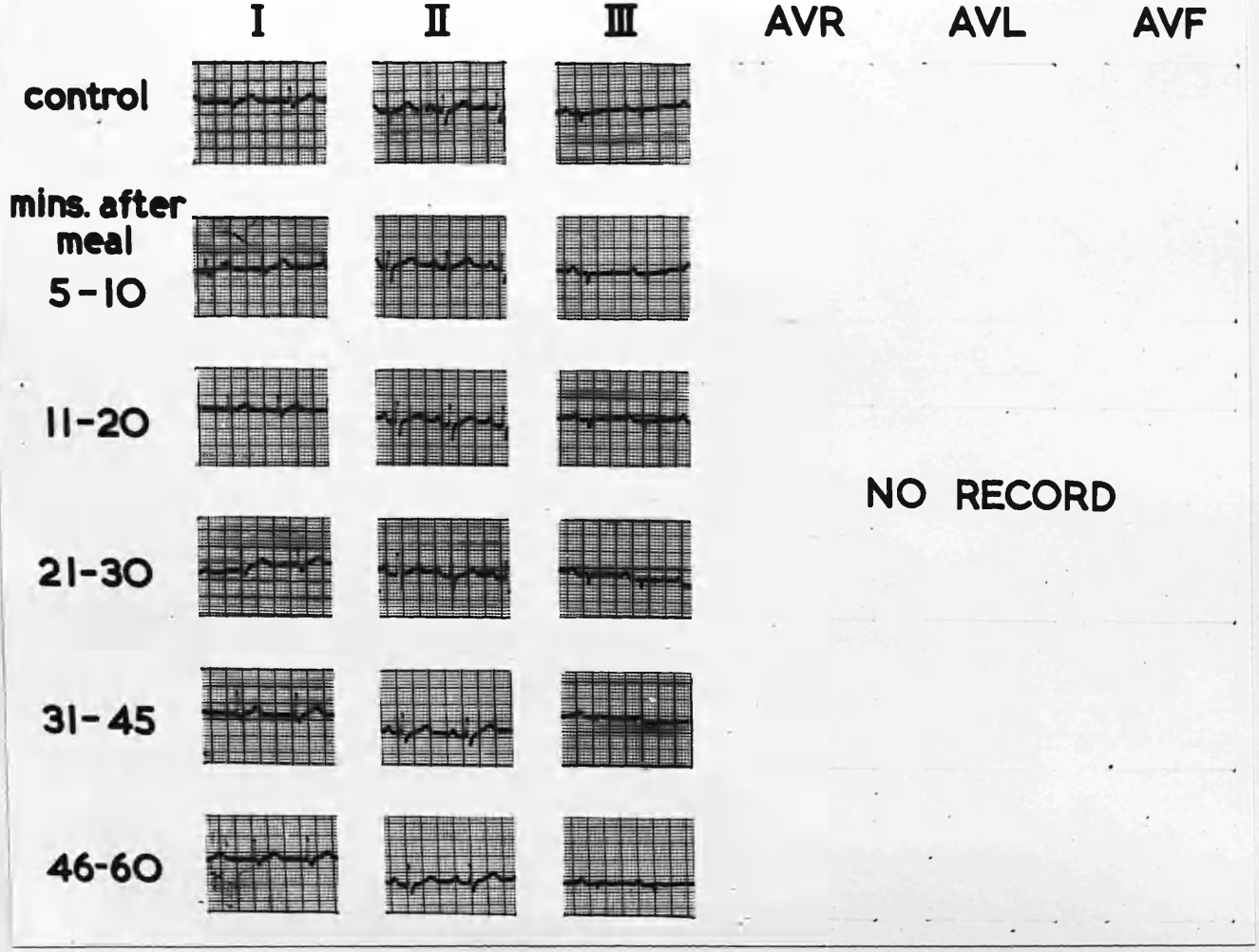
	I	II	III	AVR	AVL	AVF
control						
mins. after meal 5-10						
11-20						
21-30				NO RECORD		
31-45						
46-60						

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 4.

PATIENT 45.

TRACINGS	-	Control and at 9, 12, 20, 31 and 46 mins.
HEART RATE	-	Increased from 90 to 116
P	-	Peaked in II and III at 9 - 31 mins.
QRS	-	No change from the control
RT	-	ST sagging in III at 9 - 31 mins.
T	-	Flatter in II and III at 12 and 20 mins.
U	-	No change from the control
		Maximal changes at 20 mins.

AFTER POLYA GASTRECTOMY *No: 5*

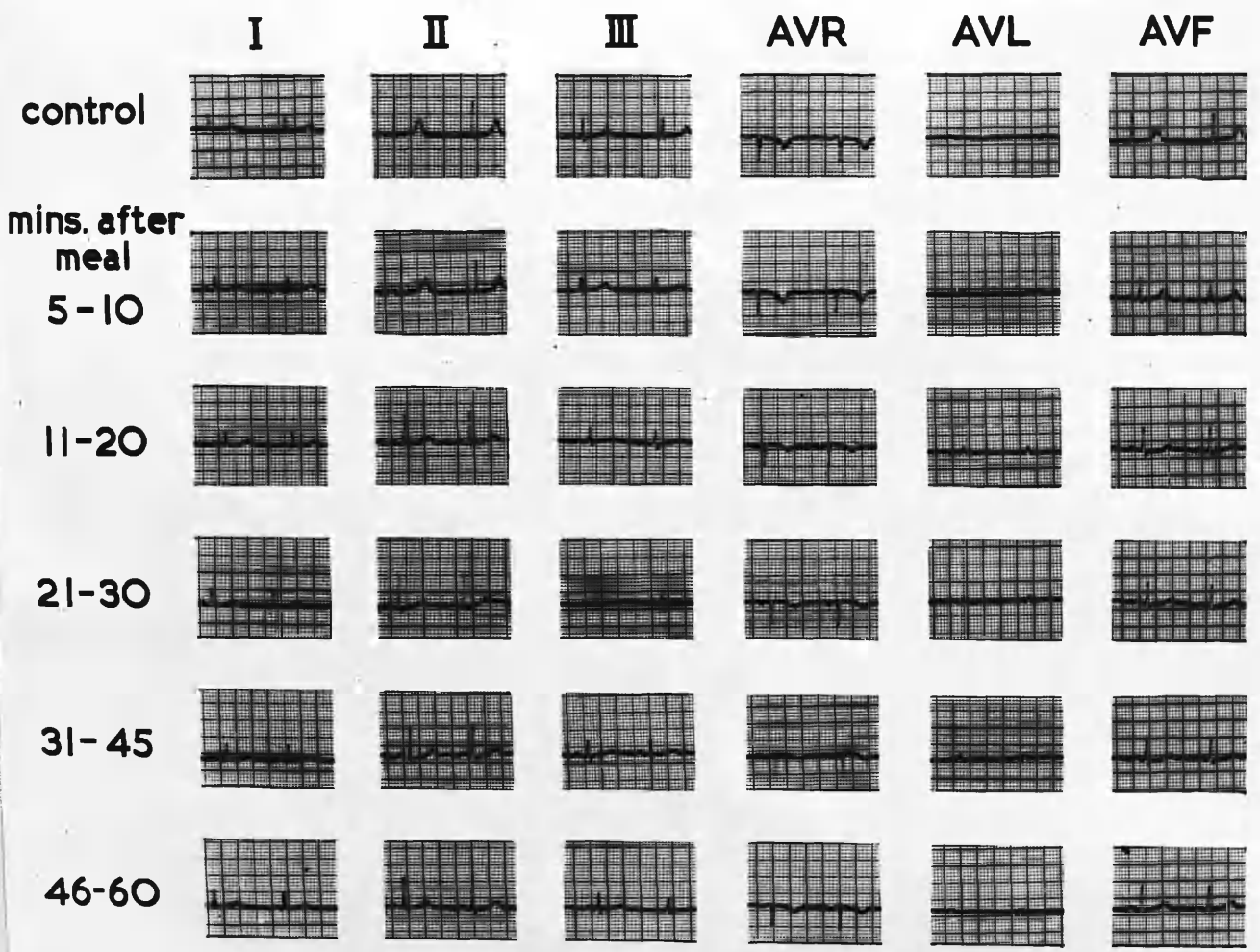


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 5.

PATIENT 15.

TRACINGS	-	Control and at 10, 16, 26, 36 and 46 mins.
HEART RATE	-	Increased from 88 to 94
P	-	No change from the control
QRS	-	No change from the control
RT	-	No change from the control
T	-	Lower in II at 16 and 26 mins.
U	-	No change from the control
		Maximal changes at 26 mins.

AFTER POLYA GASTRECTOMY *No: 7*

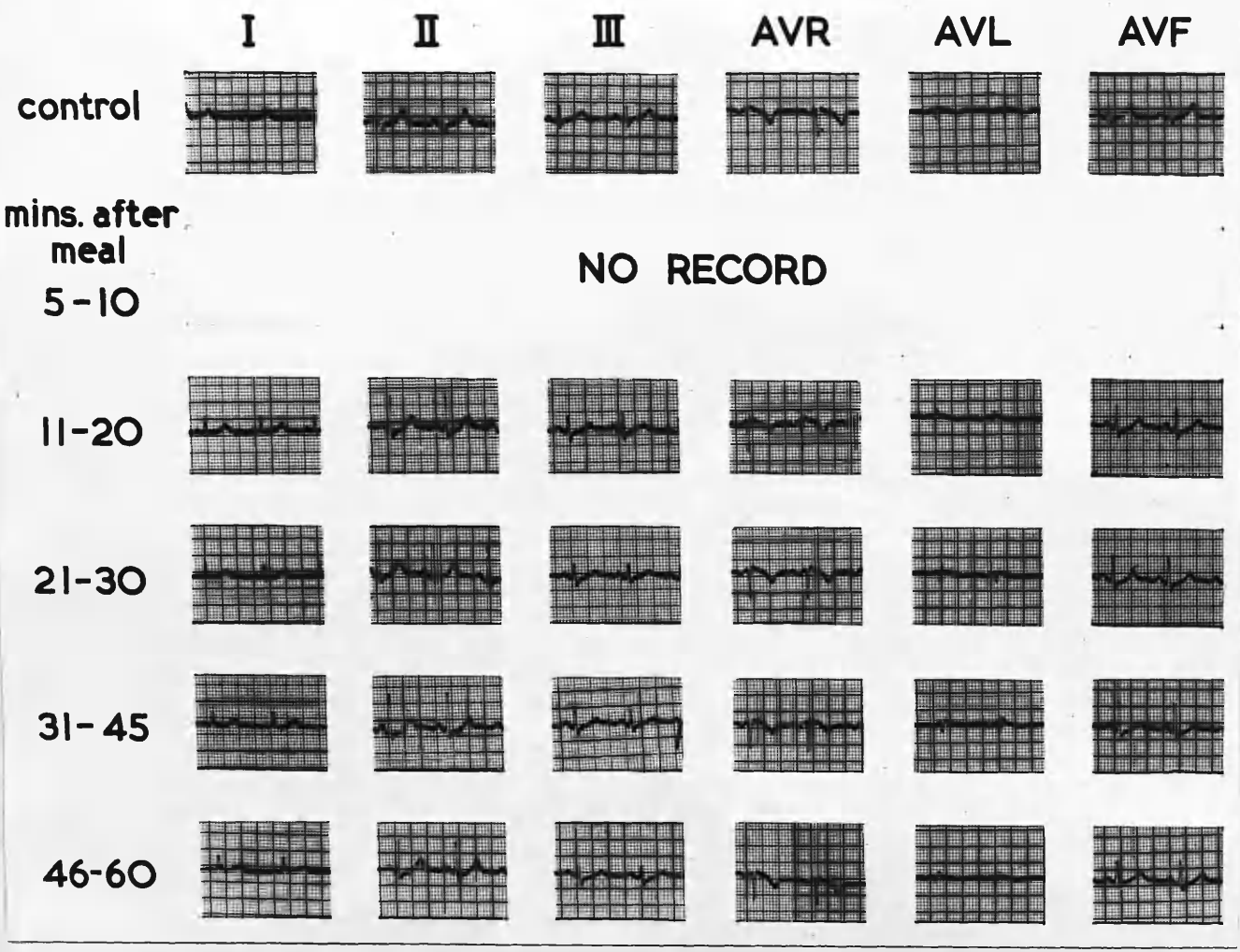


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 7.

PATIENT 43.

TRACINGS	-	Control and at 7, 18, 23, 40 and 60 mins.
HEART RATE	-	Increased from 62 to 76
P	-	Increased in II and aVF at 7 - 60 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter and broader in II, III and aVF and less negative aVR at 18 - 60 mins.
U	-	Increased in II and aVF at 18 - 60 mins., and in aVR at 23 mins. Maximal changes at 23 mins.

AFTER POLYA GASTRECTOMY *No: 8*

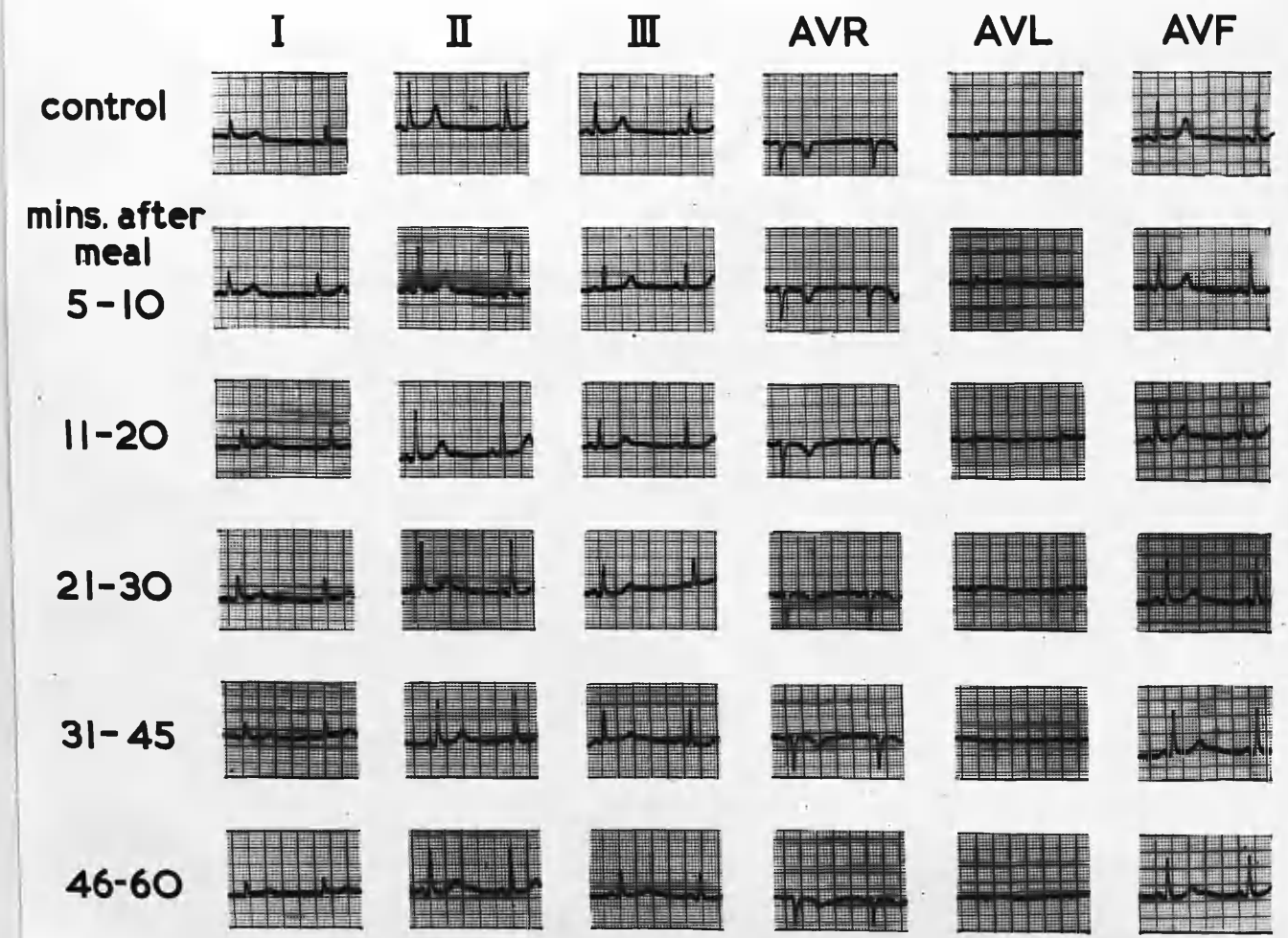


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 8.

PATIENT 39.

TRACINGS	-	Control and at 20, 30, 40 and 60 mins.
HEART RATE	-	Increased from 72 to 94
P	-	No change from the control
QRS	-	Increased R II at 20 - 60 mins.
RT	-	Low take off ST in II and aVF at 20 - 40 mins.
T	-	Flatter and broader in III and aVF at 20 - 60 mins., and slightly lower in II at 40 mins.
U	-	Increased in aVF at 20 - 40 mins. Maximal changes at 30 mins.

AFTER POLYA GASTRECTOMY No: 9

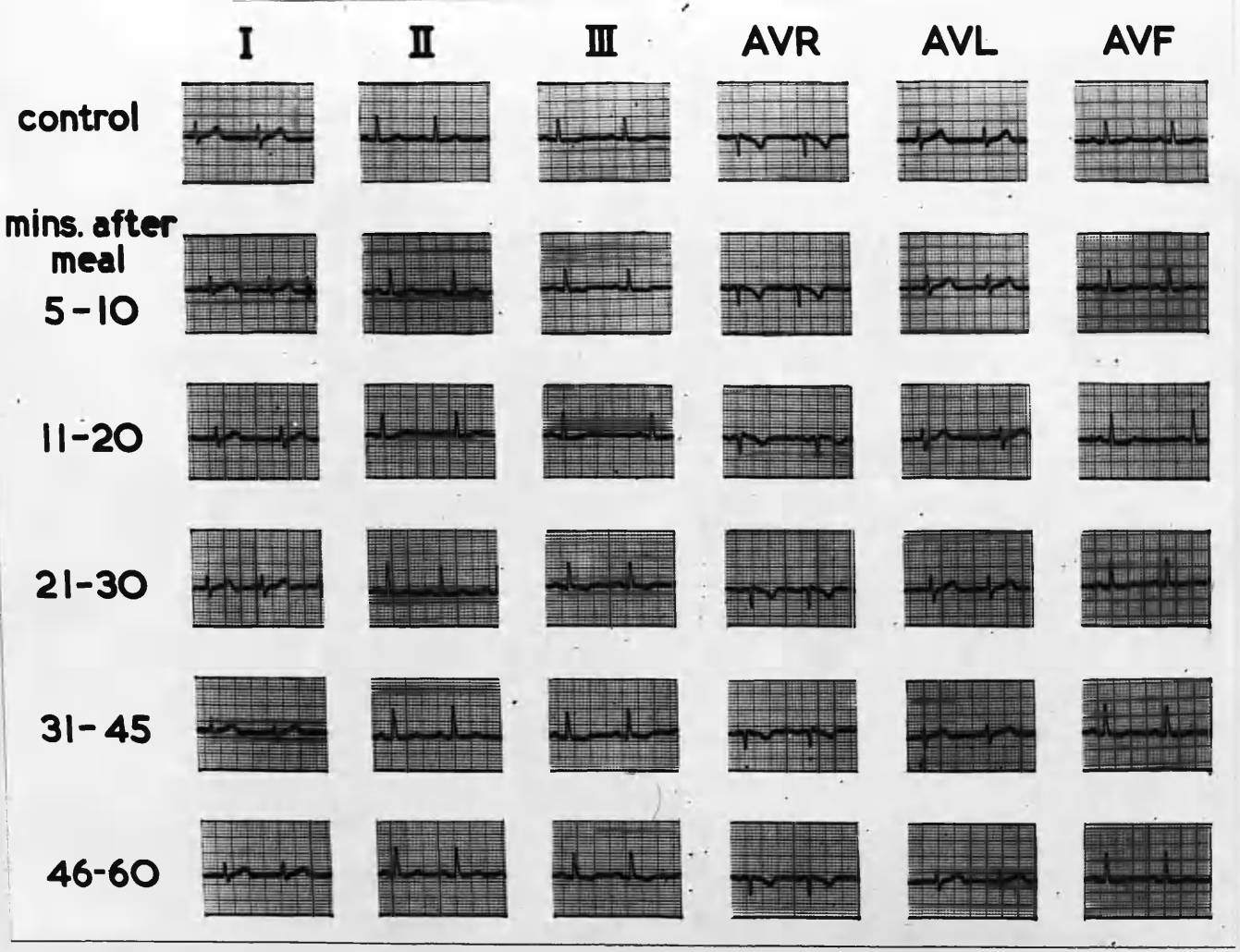


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 9.

PATIENT 23.

TRACINGS	-	Control and at 5, 19, 25, 44 and 52 mins.
HEART RATE	-	Increased from 54 to 58
P	-	No change from the control
QRS	-	Increase in R II and III at 25 and 44 mins.
RT	-	No change from the control
T	-	Smaller in II, III and aVF at 19 - 52 mins., and less negative in aVR at 25 - 52 mins.
U	-	Increased in II, III and aVF at 19 - 52 mins. Maximal changes at 44 mins.

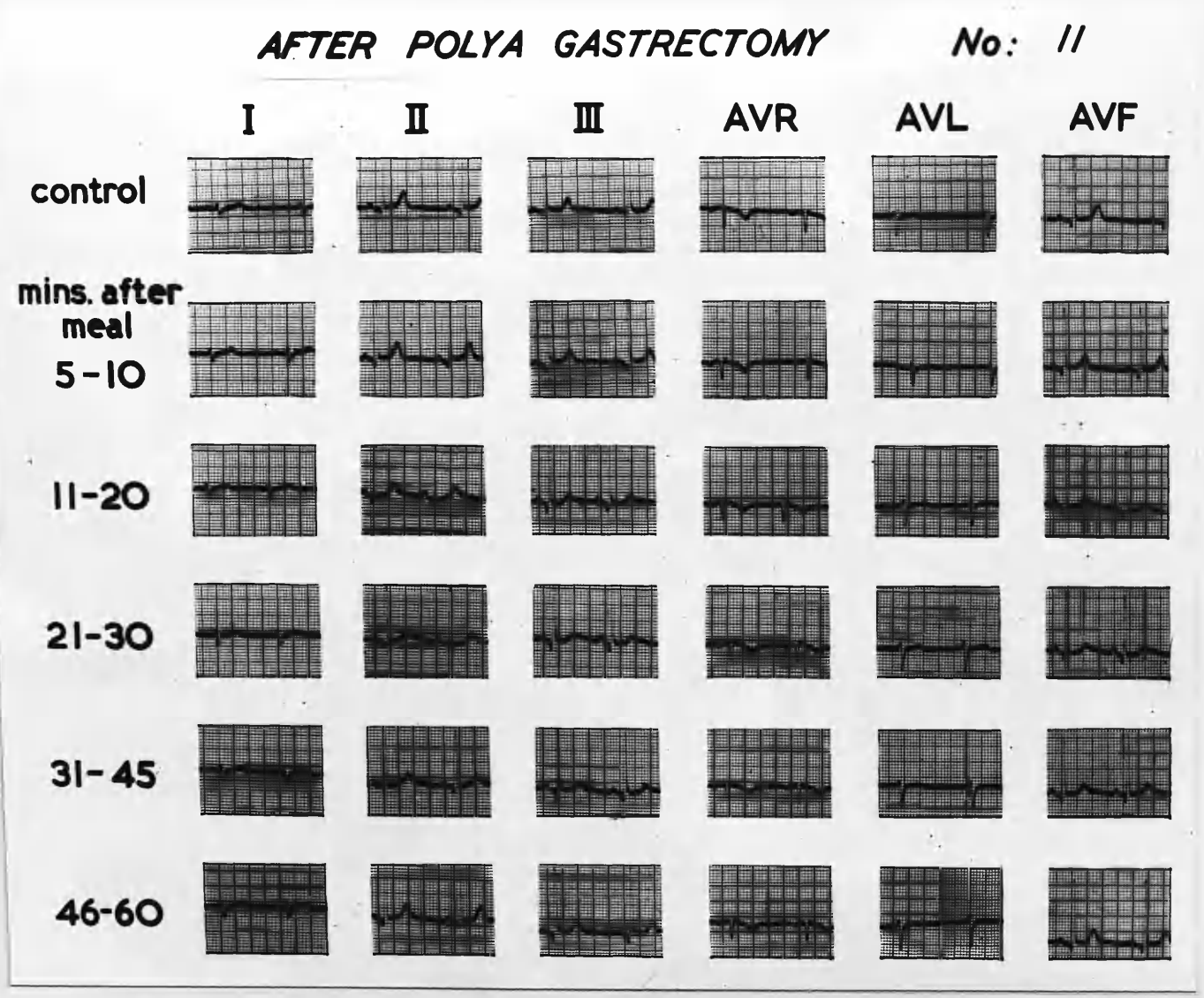
AFTER POLYA GASTRECTOMY No: 10



AFTER POLYA GASTRECTOMY - EXAMPLE NO. 10.

PATIENT 12.

TRACINGS	-	Control and at 5, 17, 24, 40 and 52 mins.
HEART RATE	-	Increased from 75 to 88
P	-	No change from the control
QRS	-	No change from the control
RT	-	ST just below isoelectric line in aVF at 17 mins.
T	-	Inversion in III at 20 - 52 mins. Less negative in aVR at 40 mins.
U	-	No change from the control
		Maximal changes at 24 mins.

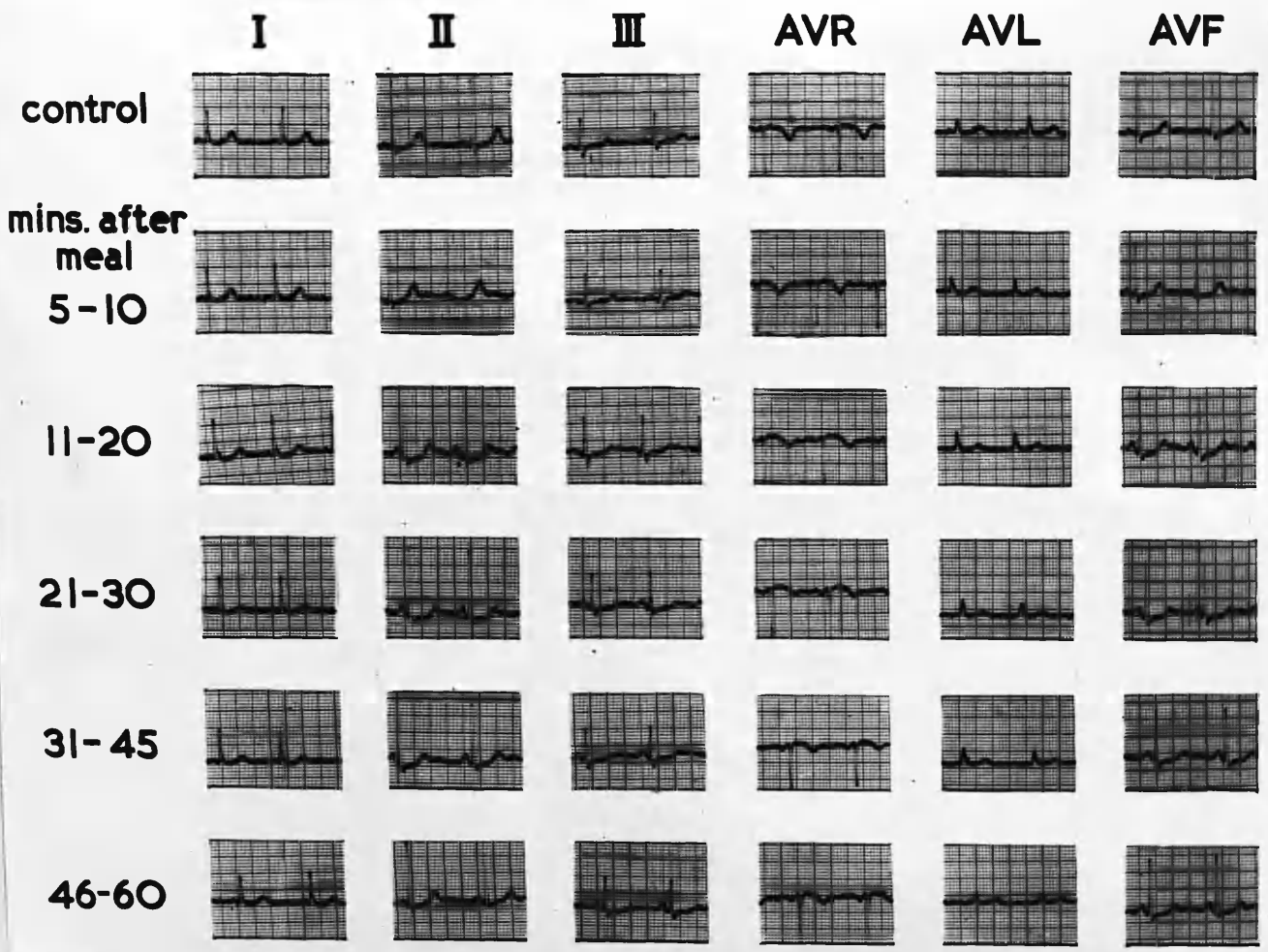

AFTER POLYA GASTRECTOMY
No: 11
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 11.

PATIENT 56.

TRACINGS	-	Control and at 5, 17, 25, 40 and 60 mins.
HEART RATE	-	Increased from 58 to 72
P	-	Peaking in II, III and aVF at 17 and 25 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter and broader in II, III and aVF at 17 to 52 mins., and less negative in aVR at 25 mins.
U	-	Increased in II, III and aVF at 17 - 60 mins. Maximal changes at 25 mins.

AFTER POLYA GASTRECTOMY No: 12

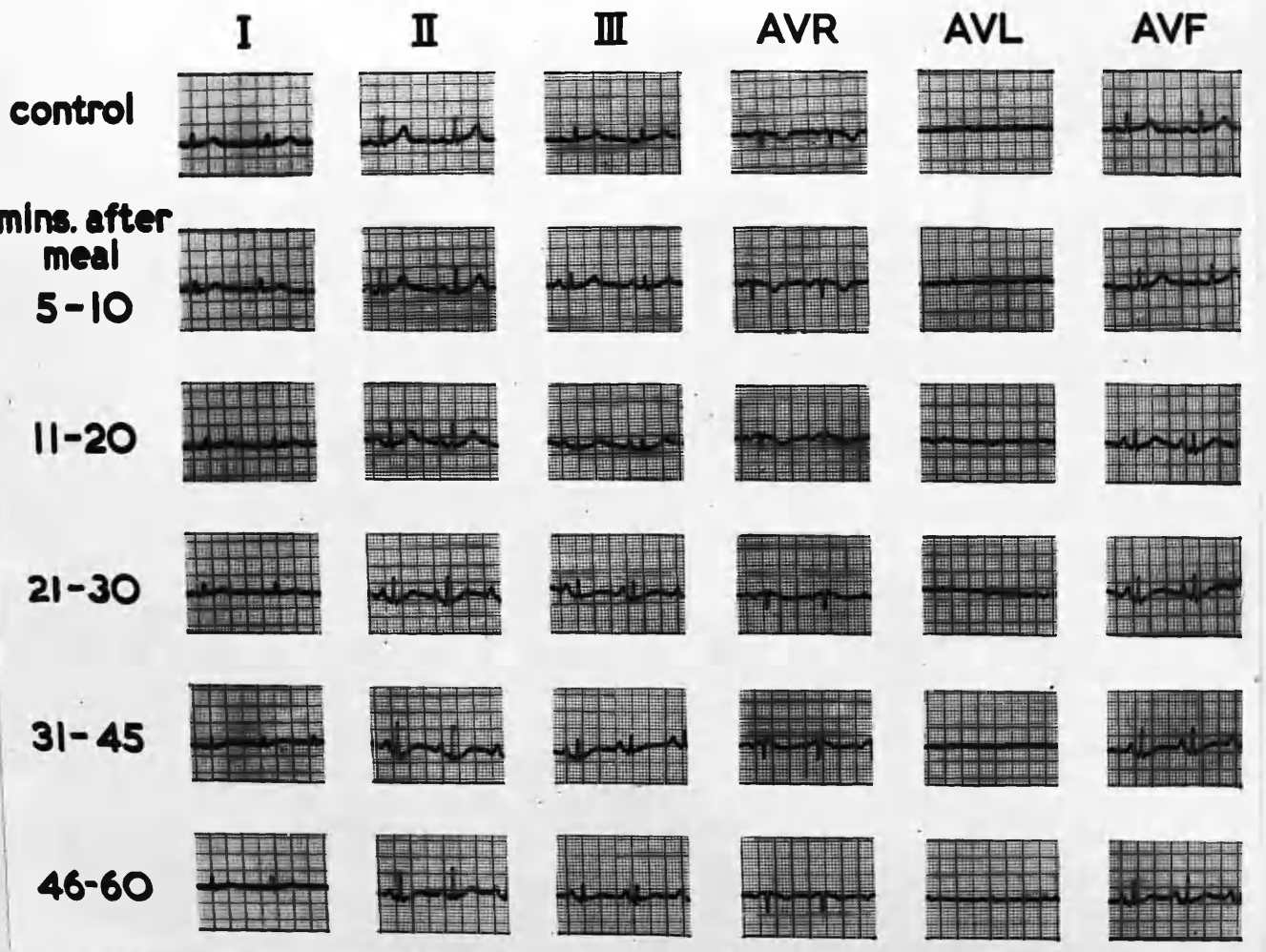


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 12.

PATIENT 30.

TRACINGS	-	Control and at 5, 12, 22, 31 and 46 mins.
HEART RATE	-	Increased from 66 to 88
P	-	Peaked in II, III and aVF at 12 to 46 mins.
QRS	-	No change from the control
RT	-	ST segment below isoelectric line in II, III and aVF at 12 - 46 mins.
T	-	Flatter and broader in II, III and aVF at 12 - 46 mins. Flatter in I at 12 - 31 mins. Less negative in aVR at 12 - 31 mins.
U	-	Increased in II and aVF at 5 - 46 mins. Maximal changes at 22 mins.

AFTER POLYA GASTRECTOMY No: 13



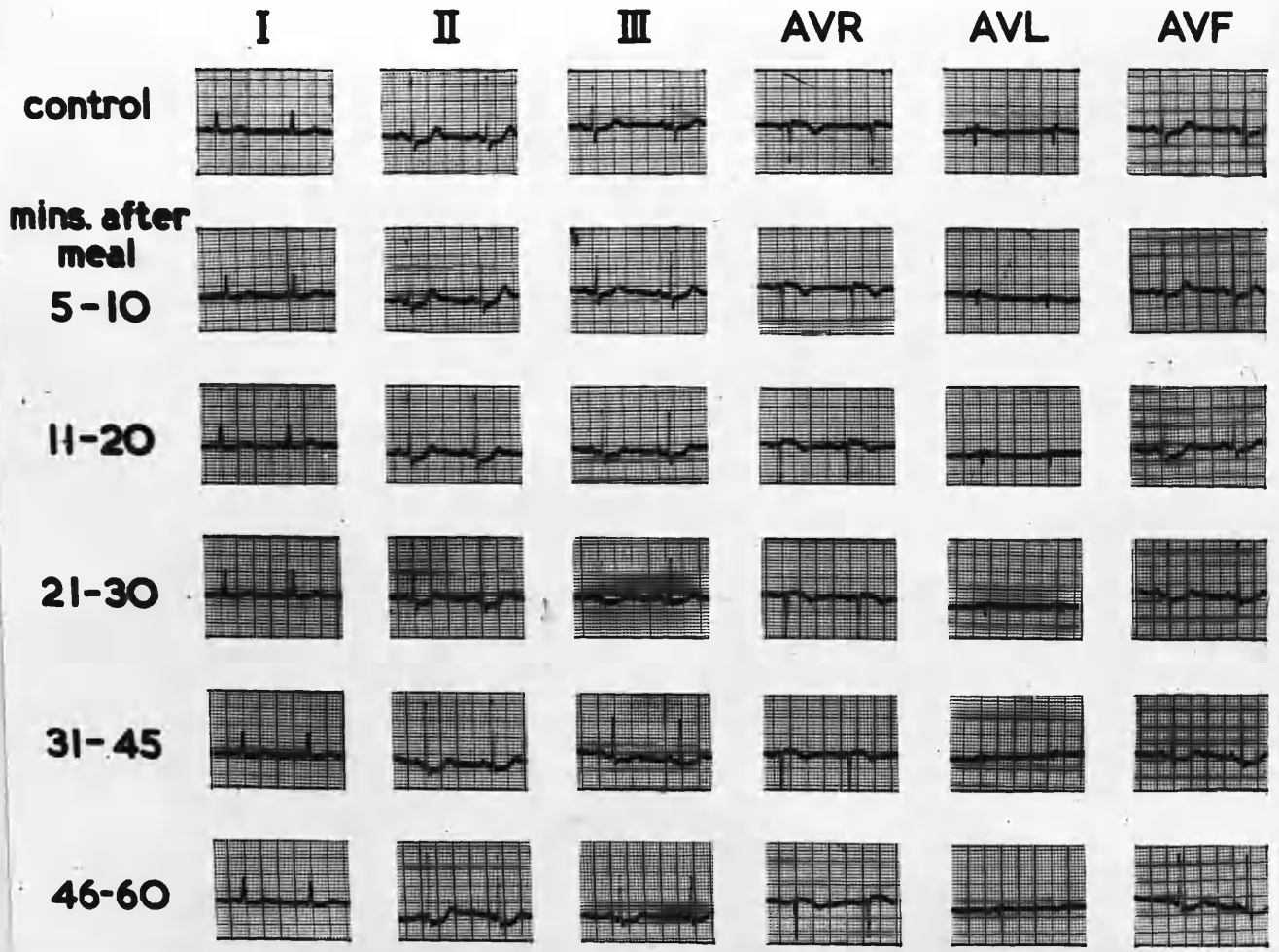
AFTER POLYA GASTRECTOMY - EXAMPLE NO. 13.

PATIENT 2.

TRACINGS	-	Control and at 10, 20, 25, 36 and 60 mins.
HEART RATE	-	Increased from 72 to 84
P	-	Peaked in II and III at 10 - 60 mins., and in aVF at 20 - 60 mins.
QRS	-	R in II and aVF increased at 36 mins.
RT	-	No change from the control
T	-	Flatter and broader in II, III and aVF at 10 - 60 mins., and less negative in aVR at 20 - 60 mins.
U	-	Increased in II, III and aVF at 20 mins., and in II up to 60 mins. Maximal changes at 36 mins.

(Compare before operation Example No. 2),

AFTER POLYA GASTRECTOMY No: 14

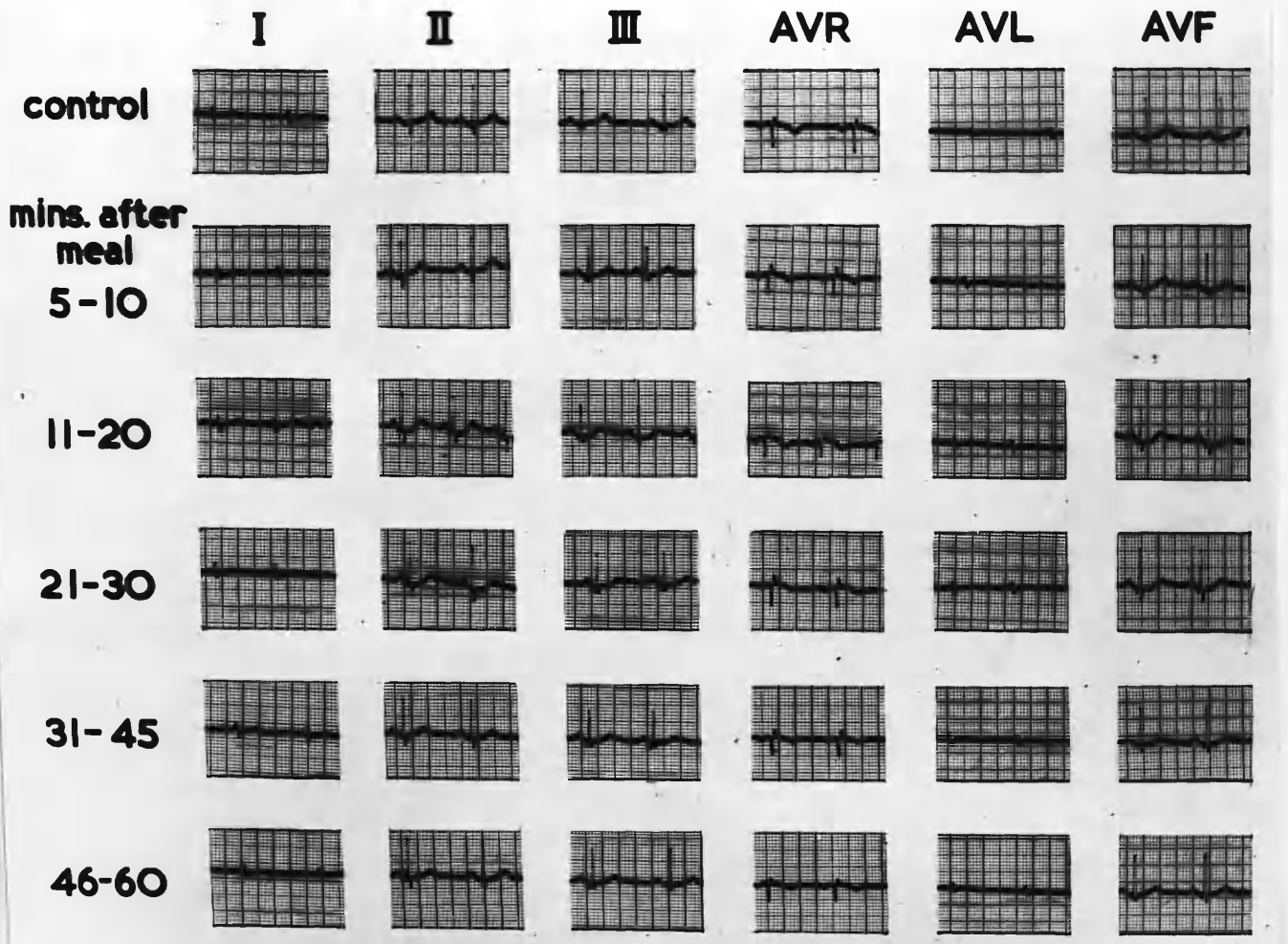


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 14.

PATIENT 24.

TRACINGS	-	Control and at 8, 17, 25, 40 and 50 mins.
HEART RATE	-	Increased from 64 to 75
P	-	No change from the control
QRS	-	No change from the control
RT	-	ST segment below isoelectric line in II (8 - 50 mins.), III (40 - 50 mins.) and aVF (17 - 50 mins.)
T	-	Flatter in II, III and aVF and less negative in aVR at 17 - 50 mins.
U	-	No change from the control
		Maximal changes at 17 mins.

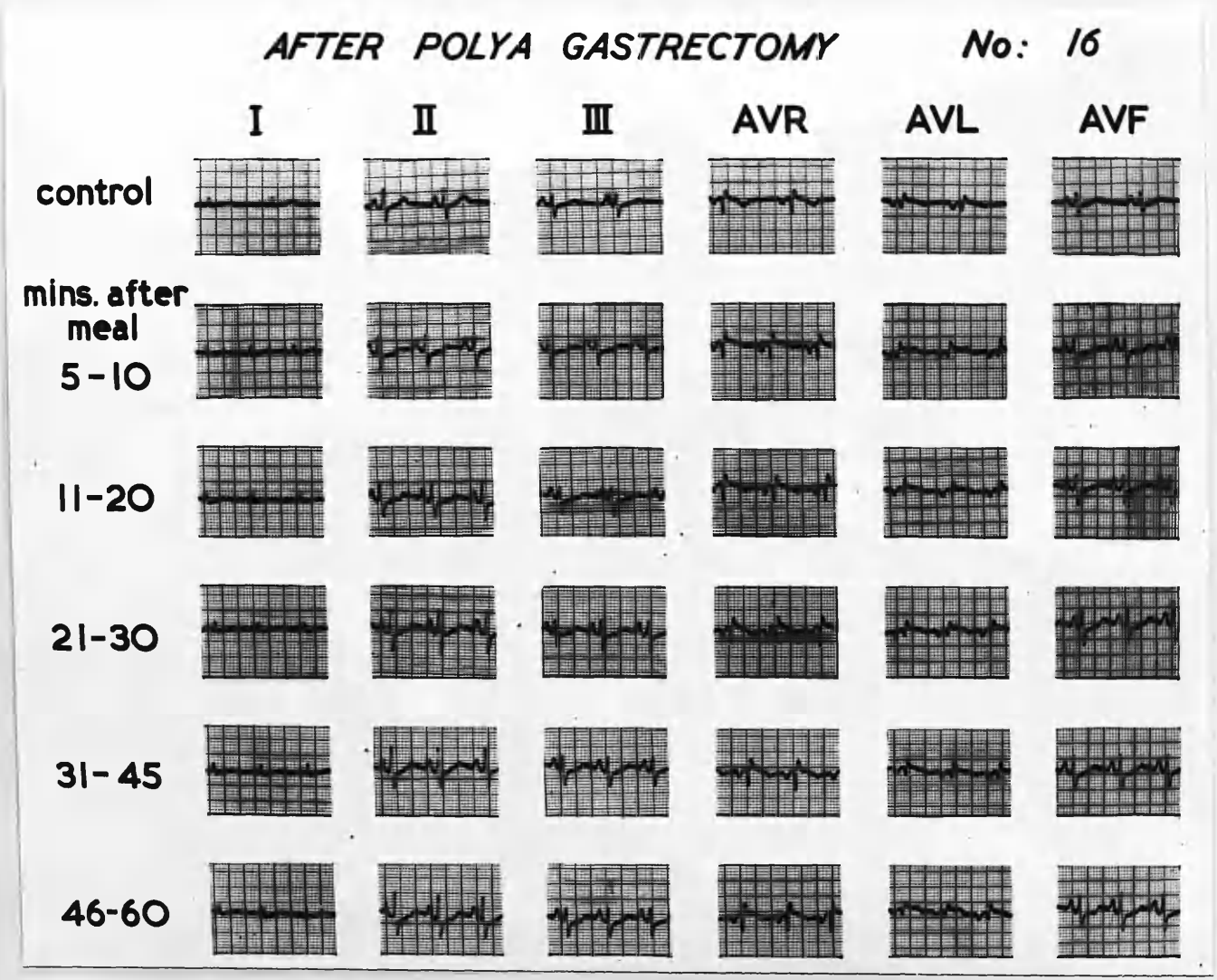
AFTER POLYA GASTRECTOMY No: 15



AFTER POLYA GASTRECTOMY - EXAMPLE NO. 15.

PATIENT 27.

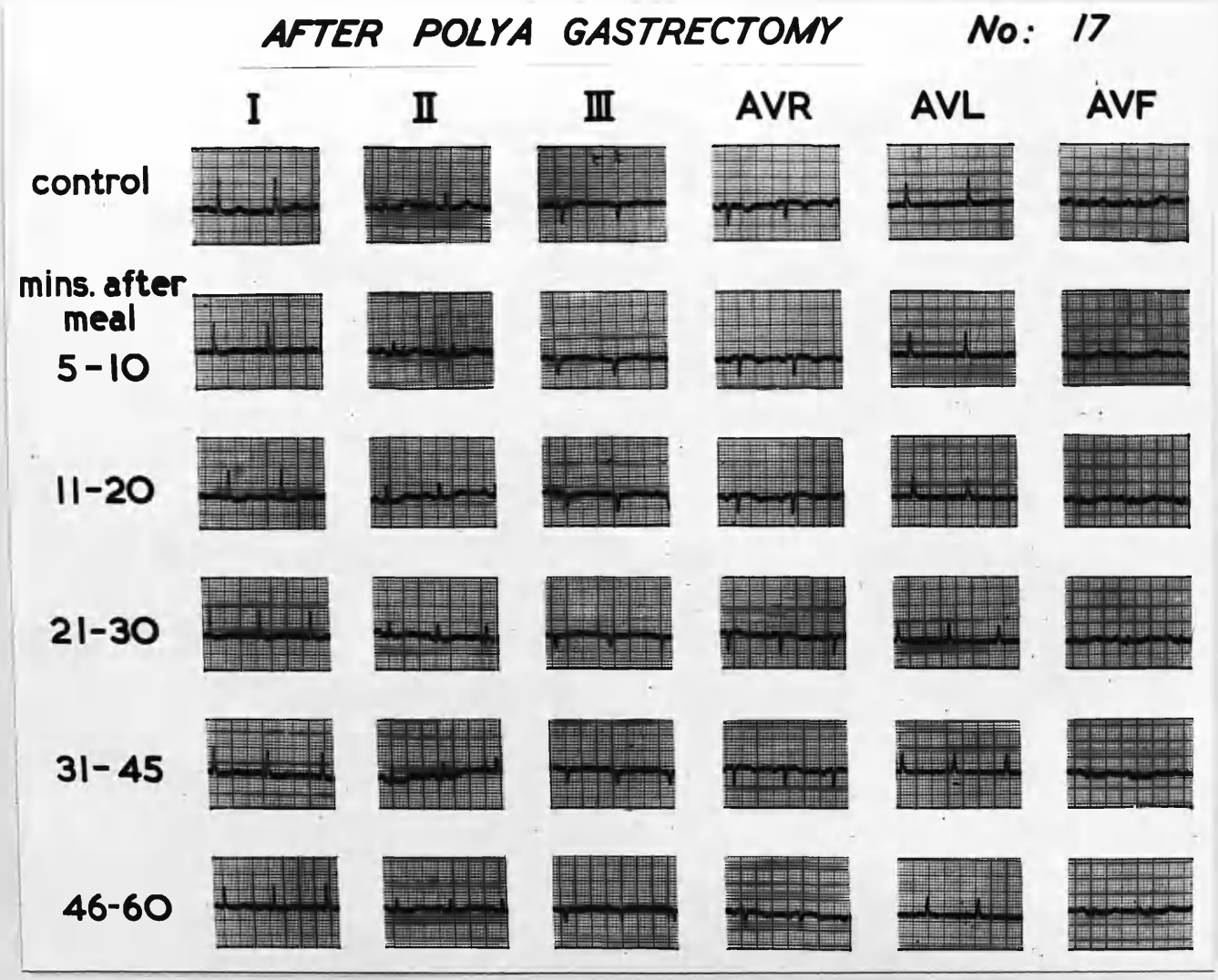
TRACINGS	-	Control and at 5, 12, 23, 40 and 60 mins.
HEART RATE	-	Increased from 66 to 94
P	-	Peaked in II, III and aVF at 12 and 23 mins.
QRS	-	No change from the control
RT	-	ST segment sags below isoelectric line in II and aVF at 12 and 23 mins.
T	-	Flatter in II, III and aVF and less negative in aVR 5 - 40 mins.
U	-	Increased in II, III and aVF at 12 and 23 mins. Maximal changes at 23 mins.


AFTER POLYA GASTRECTOMY
No: 16
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 16.

PATIENT 40.

TRACINGS	-	Control and at 10, 20, 30, 43 and 60 mins.
HEART RATE	-	Increased from 72 to 100
P	-	Peaked in II, III and aVF at 10 - 60 mins.
QRS	-	Increased in II and aVF at 30 - 60 mins.
RT	-	ST below isoelectric line in II, III and aVF and above in aVR and aVL at 10 - 60 mins.
T	-	Flatter and broader in II, III and aVF at 10 - 60 mins. and biphasic in II (10 - 20 mins.), III (10 - 30 mins.) and aVF (60 mins.). Less negative in aVR (10 - 60 mins.), biphasic aVL (10 - 60 mins.).
U	-	No change from the control Maximal changes at 10 - 60 mins.

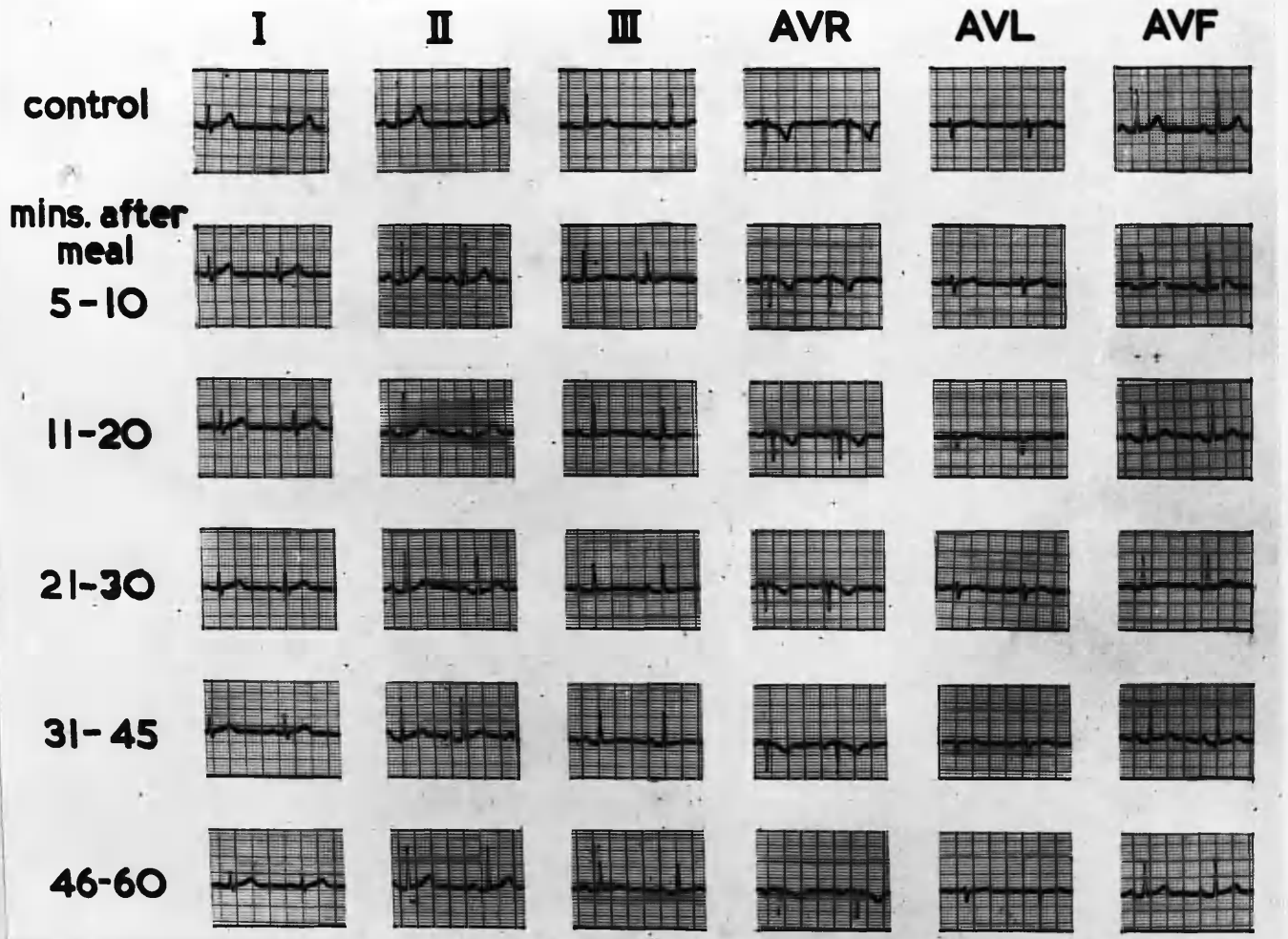

AFTER POLYA GASTRECTOMY
No: 17
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 17.

PATIENT 32.

TRACINGS	-	Control and at 5, 18, 26, 37 and 56 mins.
HEART RATE	-	Increased from 80 to 88
P	-	No change from the control
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter in I, II, III and aVF at 5 - 37 mins. Less negative in aVR at 18 - 50 mins.
U	-	Increased in II at 18 - 37 mins., and in aVF at 37 mins. Maximal changes at 26 mins.

AFTER POLYA GASTRECTOMY No: 18

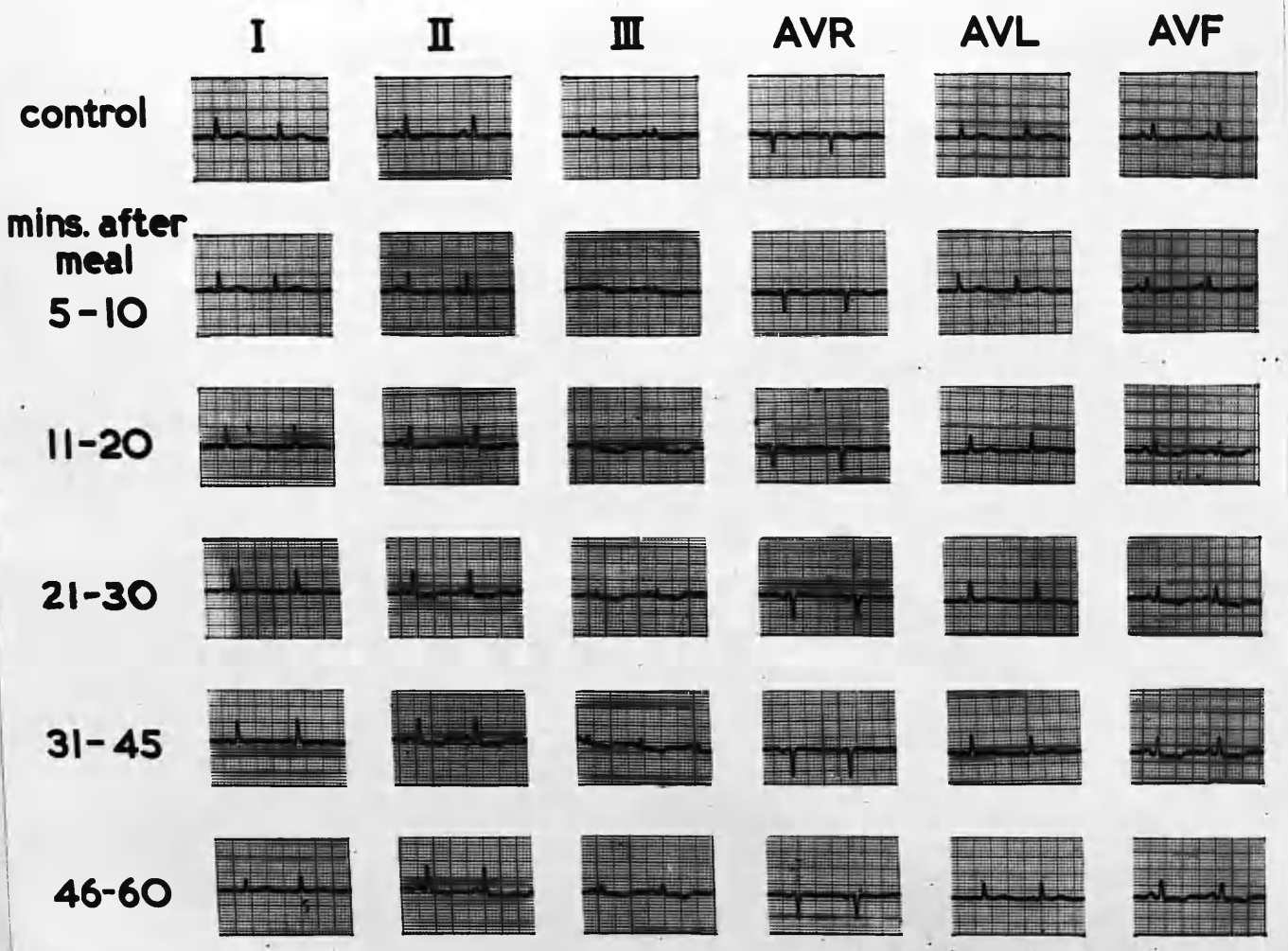


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 18.

PATIENT 5.

TRACINGS	-	Control and at 10, 17, 27, 35 and 60 mins.
HEART RATE	-	Increased from 60 to 84
P	-	Peaked in II at 27 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter in II, III and aVF at 10 - 60 mins., in I at 17 - 60 mins., and less negative in aVR at 27 and 35 mins.
U	-	Increased in II, III and aVF at 17 - 60 mins. Maximal changes at 27 mins.

AFTER POLYA GASTRECTOMY No: 19

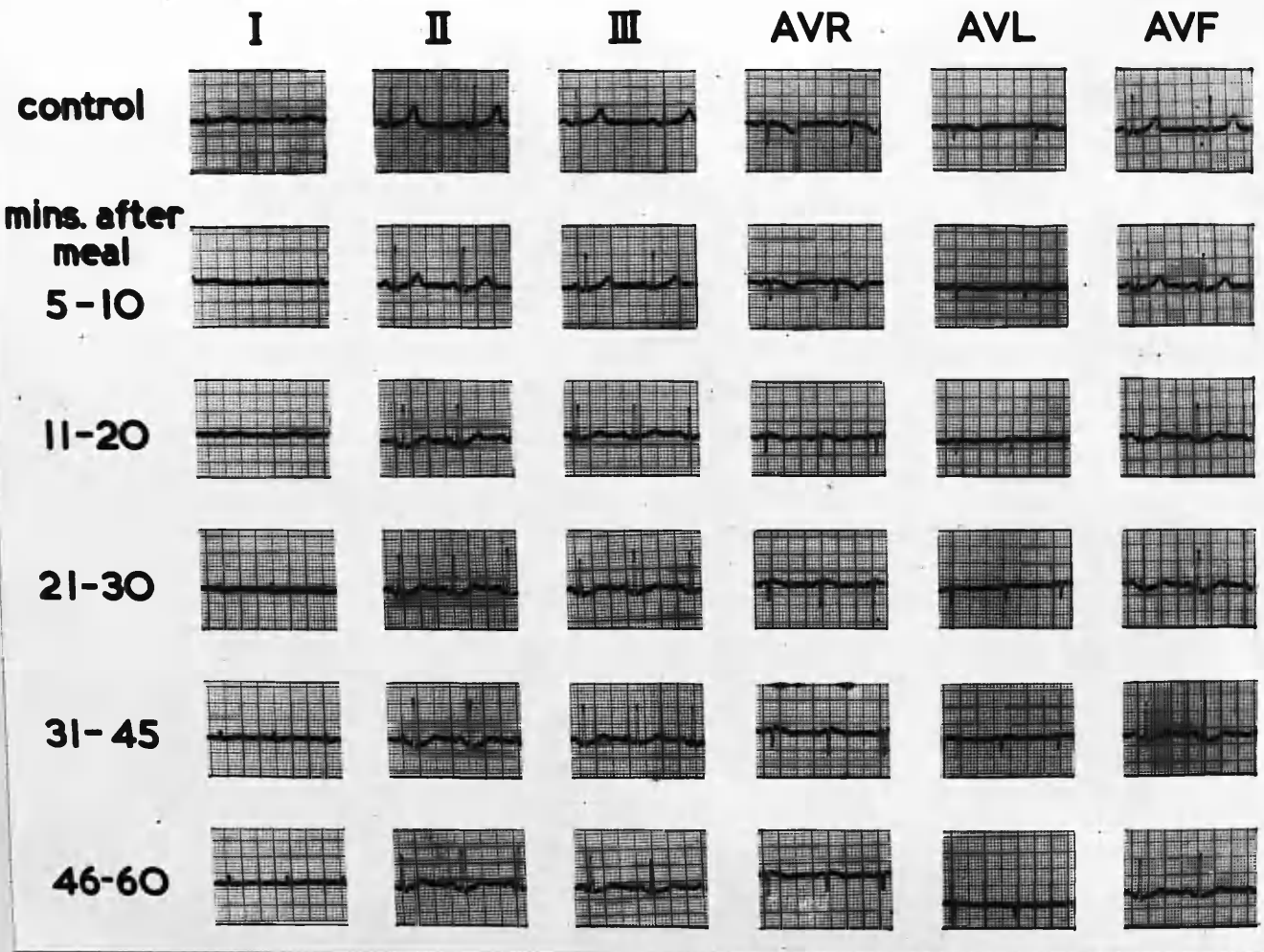


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 19.

PATIENT 6.

TRACINGS	-	Control and at 5, 18, 23, 35 and 60 mins.
HEART RATE	-	Increased from 75 to 88
P	-	No change from the control
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter in I, biphasic then inverted in II, more negative in III and biphasic then inverted in aVF at 18 - 60 mins. Less negative in aVR at 18 - 60 mins.
U	-	No change from the control Maximal changes at 23 mins.

AFTER POLYA GASTRECTOMY No: 20

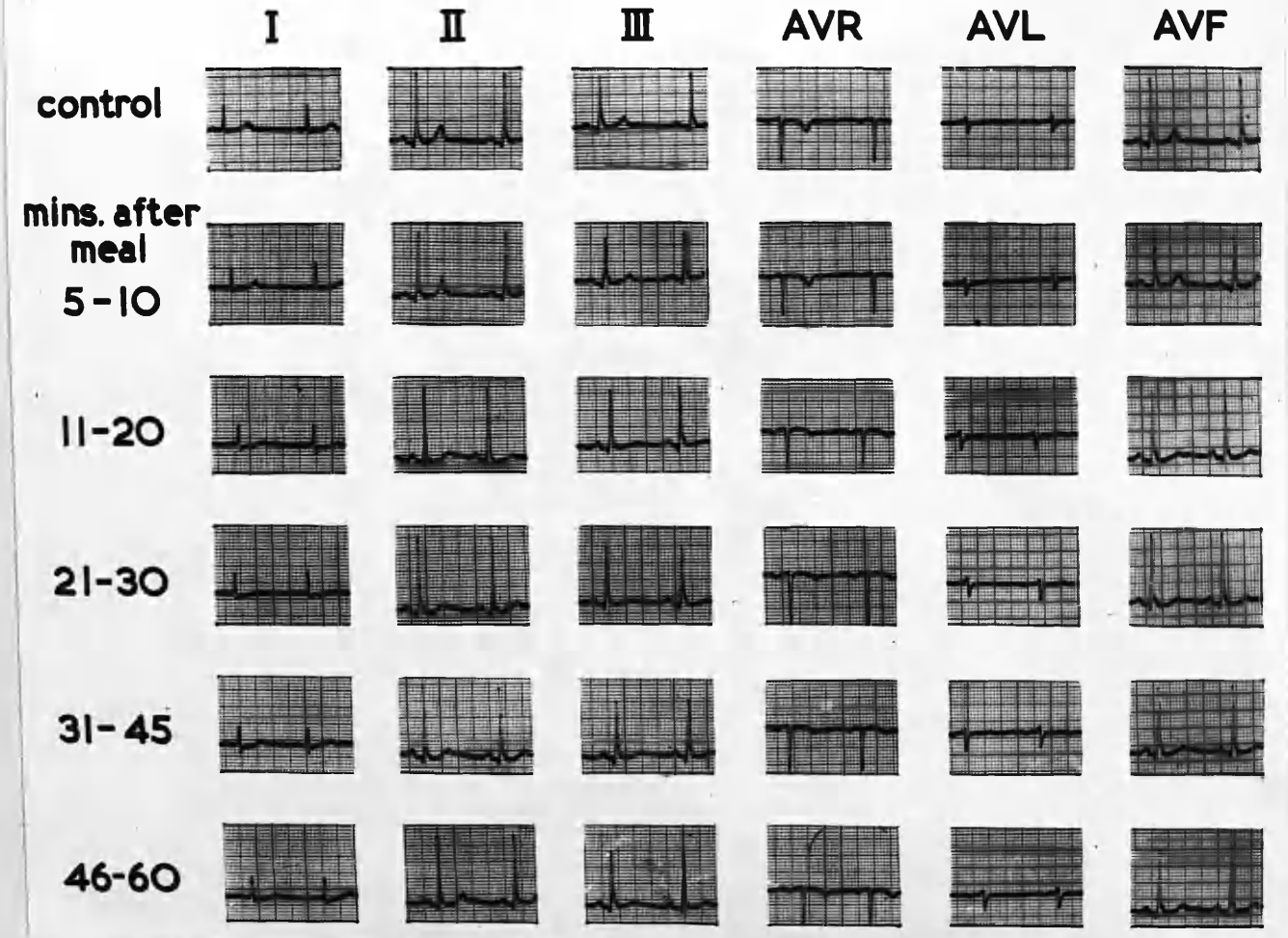


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 20.

PATIENT 37.

TRACINGS	-	Control and at 5, 17, 28, 35 and 46 mins.
HEART RATE	-	Increased from 62 to 88
P	-	No change from the control
QRS	-	Increased in R II and III at 35 mins.
RT	-	ST low take off in II, III and aVF at 28 and 35 mins.
T	-	Flatter and broader in II, III and aVF at 5 - 46 mins. Less negative in aVR at 17 - 46 mins.
U	-	Increased in II, III and aVF at 17 - 46 mins. Maximal changes at 28 mins.

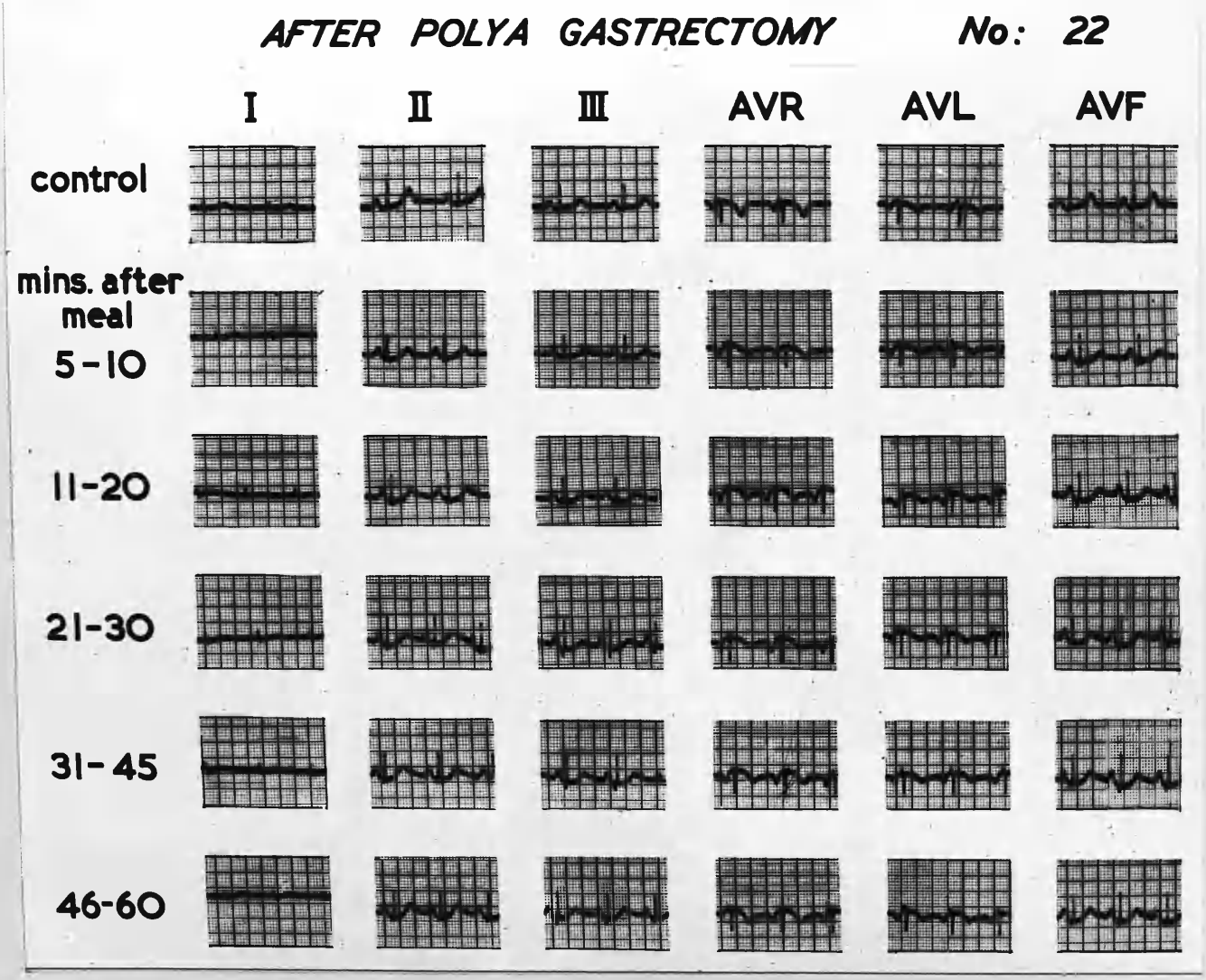
AFTER POLYA GASTRECTOMY No: 21



AFTER POLYA GASTRECTOMY - EXAMPLE NO. 21.

PATIENT 29.

TRACINGS	-	Control and at 6, 17, 26, 38 and 60 mins.
HEART RATE	-	Increased from 58 to 74
P	-	Peaked in II at 26 and 38 and in aVF at 38 mins.
QRS	-	R in II, III and aVF increased at 26 and 38 mins.
RT	-	No change from the control
T	-	Flatter and broader in I, II, III and aVF and less negative in aVR at 6 - 60 mins.
U	-	Increased in II and aVF at 26 - 60 mins. Maximal changes at 38 mins.

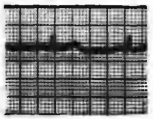
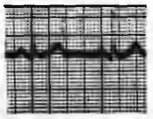
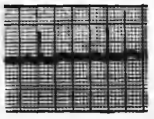
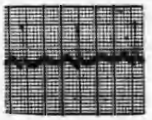
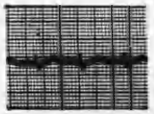
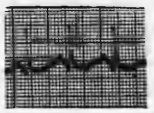
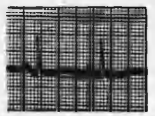
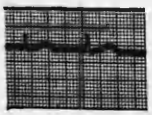
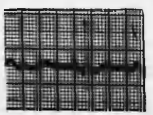
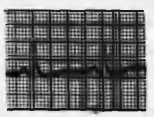
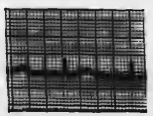
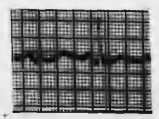

AFTER POLYA GASTRECTOMY
No: 22
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 22.

PATIENT 18.

TRACINGS	-	Control and at 9, 19, 25, 35 and 47 mins.
HEART RATE	-	Increased from 72 to 92
P	-	Peaking in II, III and aVF at 25 - 47 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Broader and flatter at 9 mins. in II, III, and aVF. Broader II, III and aVF at 25 - 47 mins.
U	-	No change from the control Maximal changes at 25 mins.

AFTER POLYA GASTRECTOMY No: 23

	I	II	III	AVR	AVL	AVF
control						
mins. after meal 5-10						
11-20						
21-30						
31-45						
46-60						

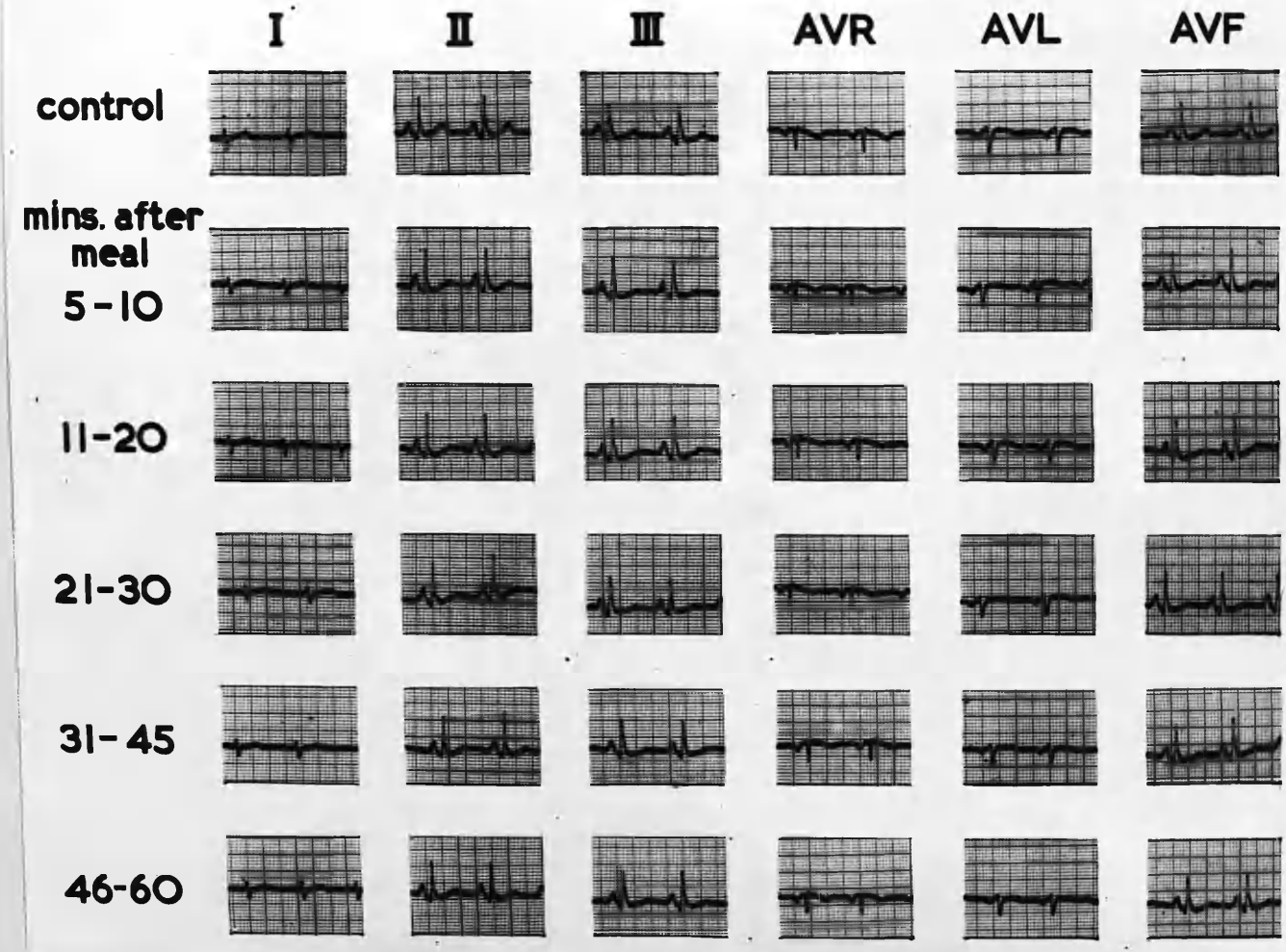
NO RECORD

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 23.

PATIENT 14.

TRACINGS	-	Control and at 10, 20, 25, 37 and 47 mins.
HEART RATE	-	Increased from 72 to 122
P	-	Peaked in II and III at 20 and 25 mins.
QRS	-	Increased in R II at 25 and 37 and R III at 20 - 47 mins.
RT	-	ST sagging below isoelectric line in II at 20 and 25 mins.
T	-	Flatter in II at 10 - 47 mins., and in III with biphasic at 20 mins.
U	-	No change from the control Maximal changes at 20 mins.

AFTER POLYA GASTRECTOMY *No: 24*



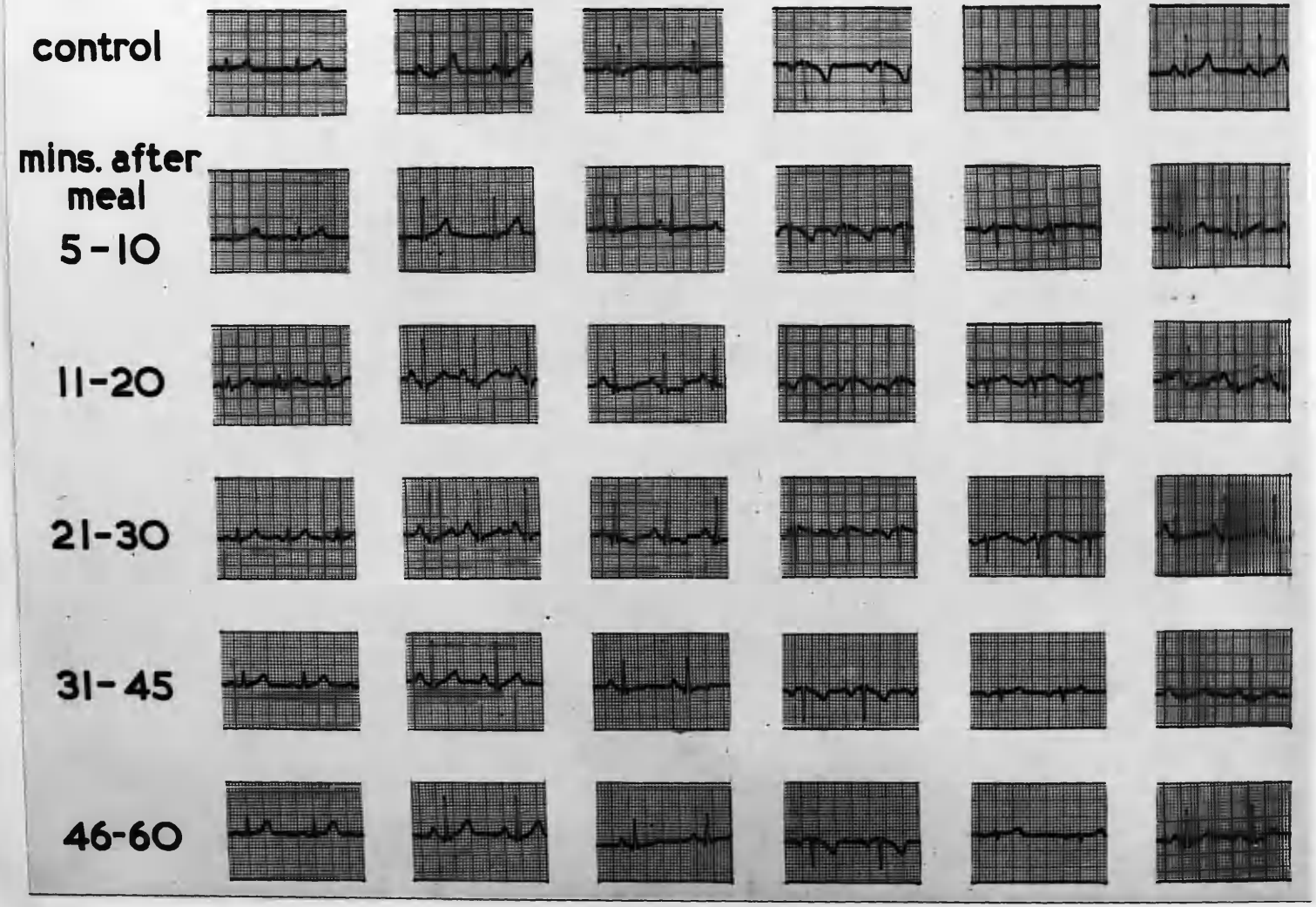
AFTER POLYA GASTRECTOMY - EXAMPLE NO. 24.

PATIENT 31.

TRACINGS	-	Control and at 10, 20, 25, 40 and 50 mins.
HEART RATE	-	Increased from 74 to 83
P	-	Peaked in II, III, and aVF at 10 - 50 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter in II, III and aVF and less negative aVR at 10 - 50 mins.
U	-	Increased in II at 10 - 50 mins.
		Maximal changes at 25 mins.

AFTER POLYA GASTRECTOMY

No: 25



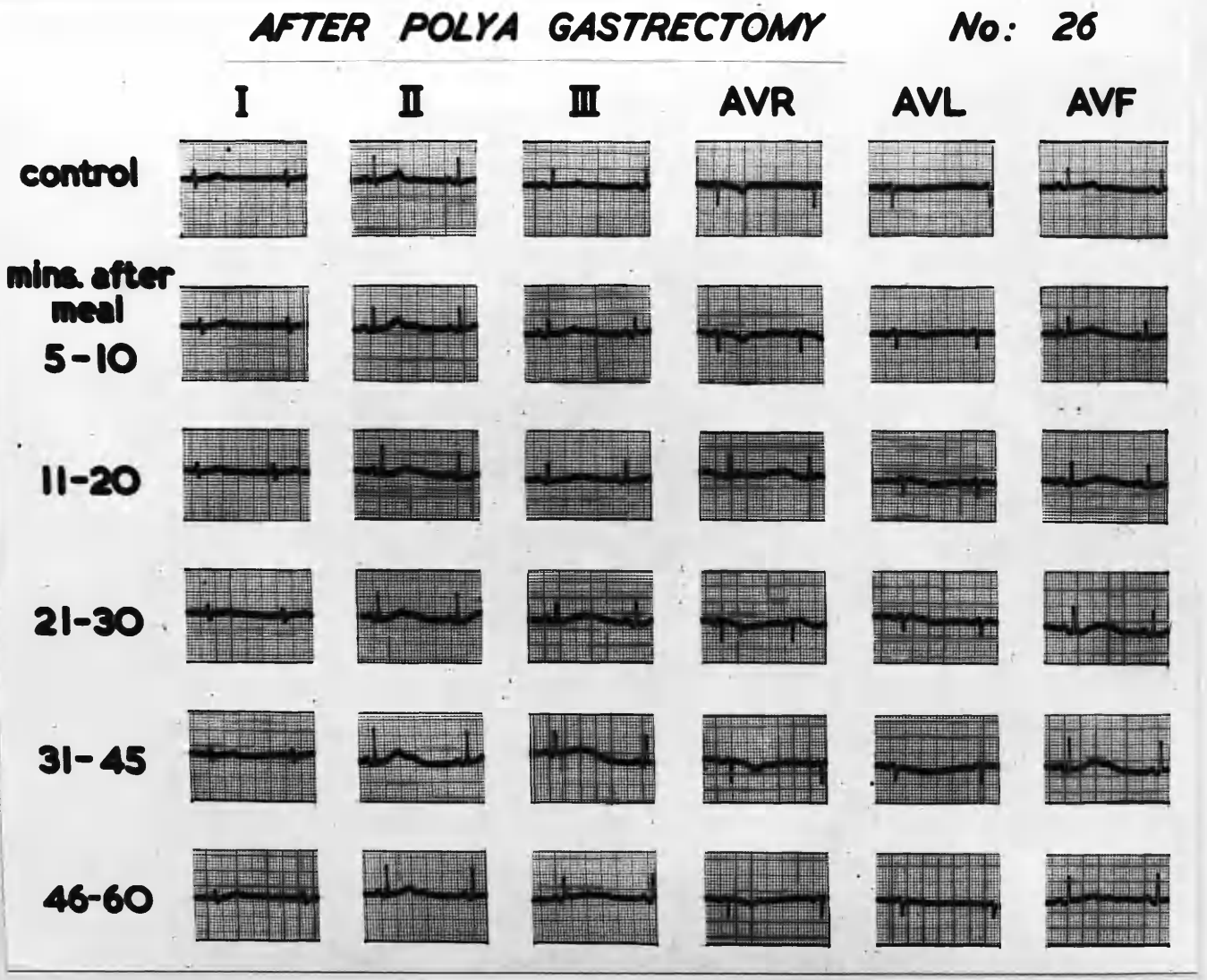
AFTER POLYA GASTRECTOMY - EXAMPLE NO. 25.

PATIENT 3.

TRACINGS	-	Control and at 6, 12, 25, 40 and 60 mins.
HEART RATE	-	Increased from 70 to 100
P	-	Peaked in II, III and aVF at 12 - 40 mins.
QRS	-	R increased in III and aVF at 12 and 25 mins. S less negative in aVL at 40 and 60 mins.
RT	-	ST in III below isoelectric line at 12 and 25 mins.
T	-	Flatter in II at 12 - 40 mins., becomes inverted III at 12 and 25 mins., and biphasic aVF at 12 and 25 mins. Less negative in aVR and 12 - 40 mins., and becomes positive aVL at 12 and 25 mins.
U	-	Increased in II and aVF at 40 mins.

Maximal changes at 25 mins.

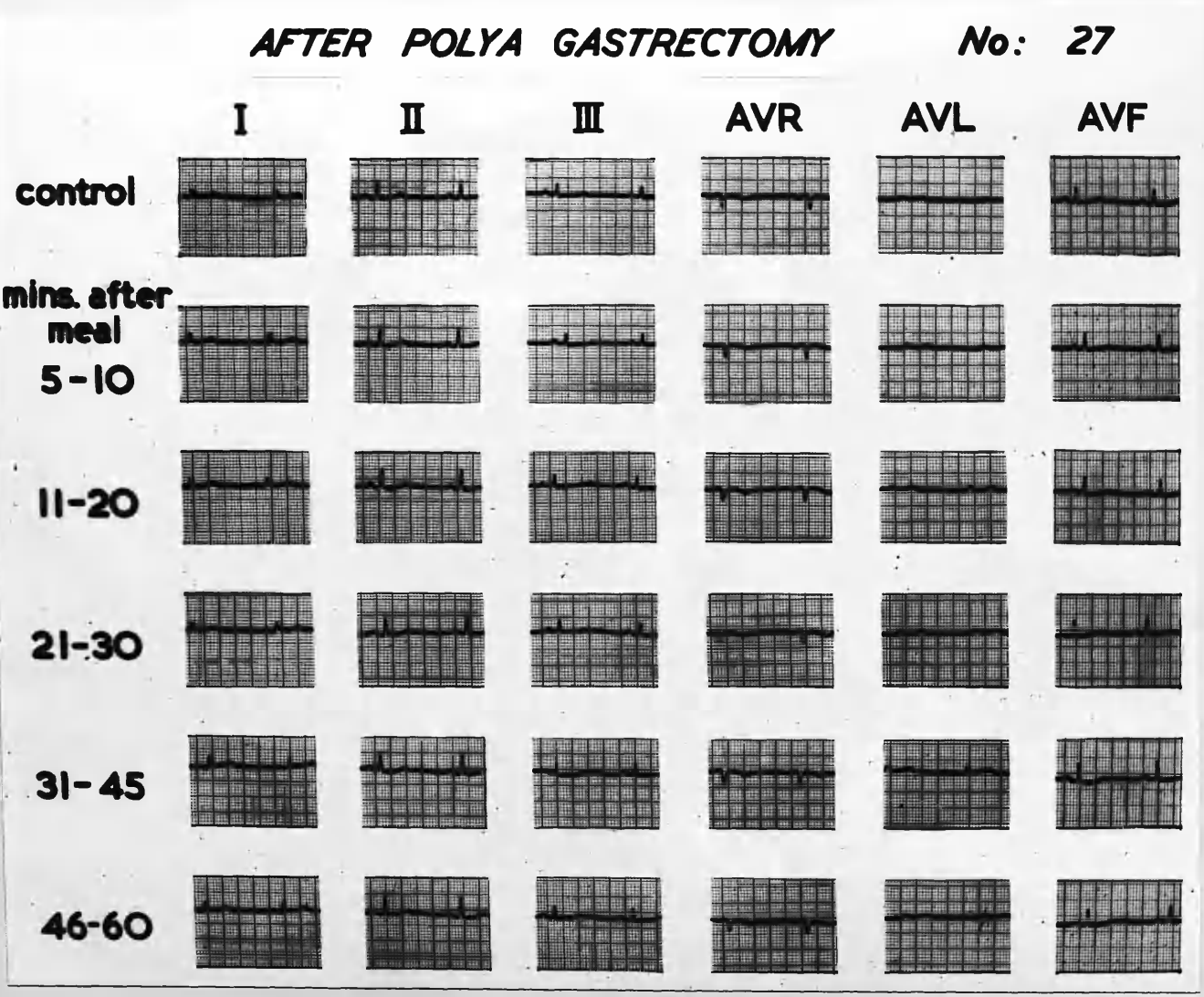
(Compare before operation Example No. 3).


AFTER POLYA GASTRECTOMY
No: 26
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 26.

PATIENT 26.

TRACINGS	-	Control and at 7, 12, 28, 40 and 60 mins.
HEART RATE	-	Increased from 54 to 60
P	-	No change from the control
QRS	-	Increased in R II, III and aVF at 28, 40 and 60 mins.
RT	-	No change from the control
T	-	Flatter and broader in II, III and aVF at 7 - 28 mins.
		when recovery of height but still broad. Less negative in aVR
U	-	Increased in II, aVR, and aVF at 7 - 60 mins.
		Maximal changes at 28 mins.

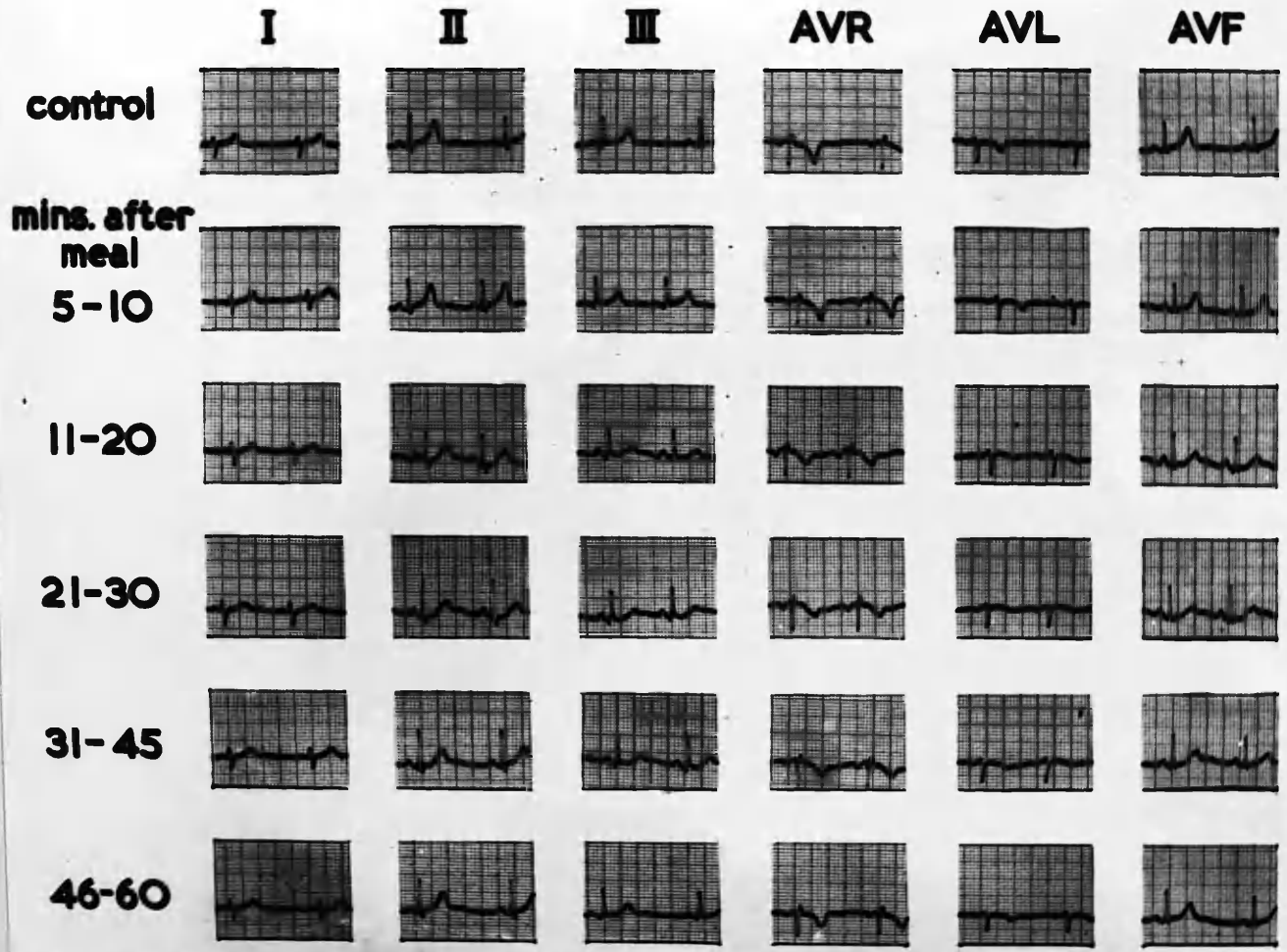

AFTER POLYA GASTRECTOMY
No: 27
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 27.

PATIENT 42.

TRACINGS	-	Control and at 5, 20, 36, 45, and 58 mins.
HEART RATE	-	Increased from 58 to 66
P	-	No change from the control
QRS	-	Low voltage
RT	-	ST segment sagging below isoelectric line in II, III, and aVF at 45 mins.
T	-	Flatter in II and aVF at 20 - 45 mins.
U	-	Increased in II at 5 - 58 mins. Maximal changes at 45 mins.

AFTER POLYA GASTRECTOMY No: 28

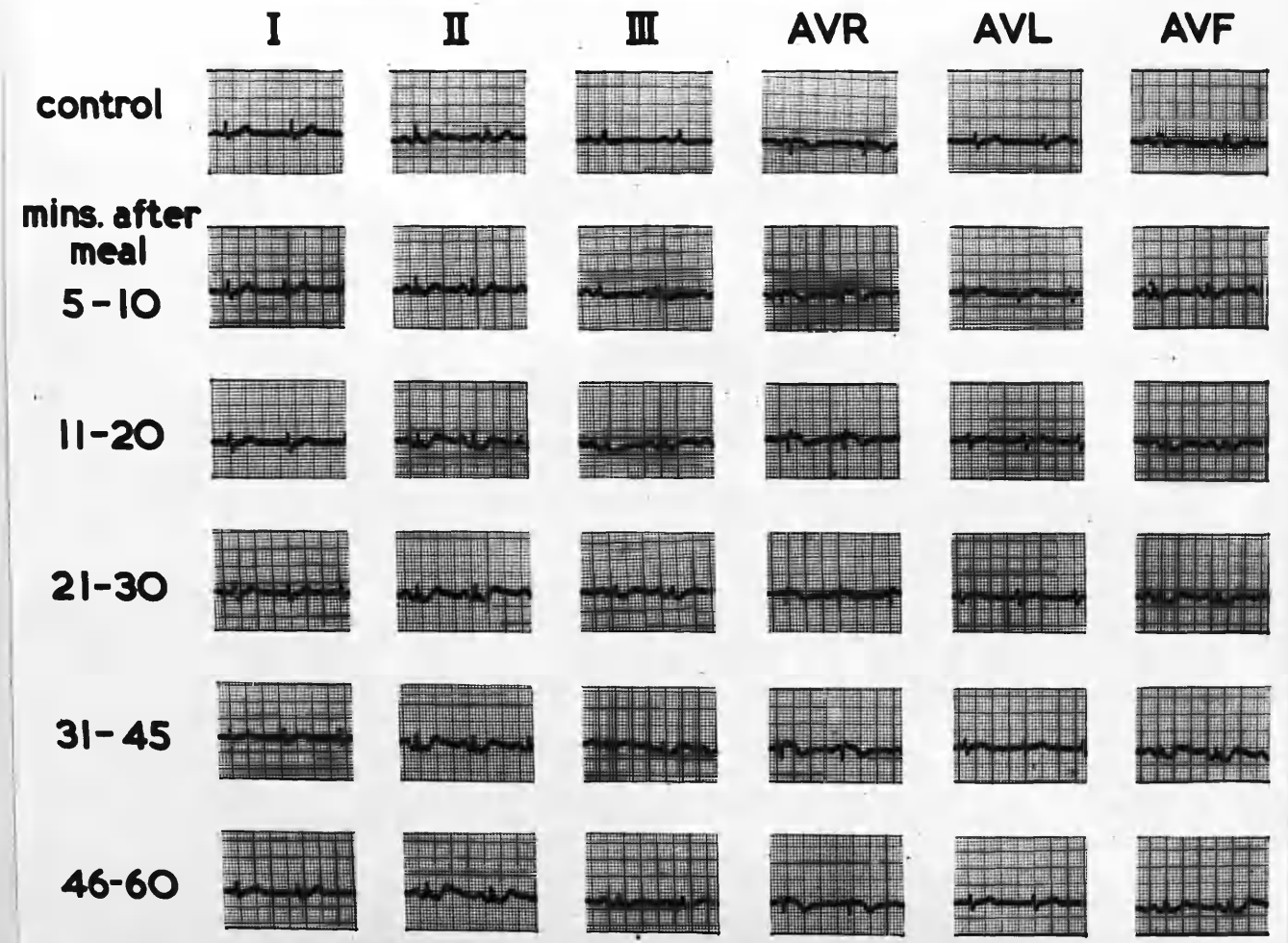


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 28.

PATIENT 25.

TRACINGS	-	Control and at 6, 18, 25, 40 and 60 mins.
HEART RATE	-	Increased from 54 to 84
P	-	Peaked in II and aVF at 18 and 25 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter in II, III and aVF and less negative in aVR at 12 - 60 mins.
U	-	Increased in II, III, aVR and aVF at 18 - 60 mins. Maximal changes at 25 mins.

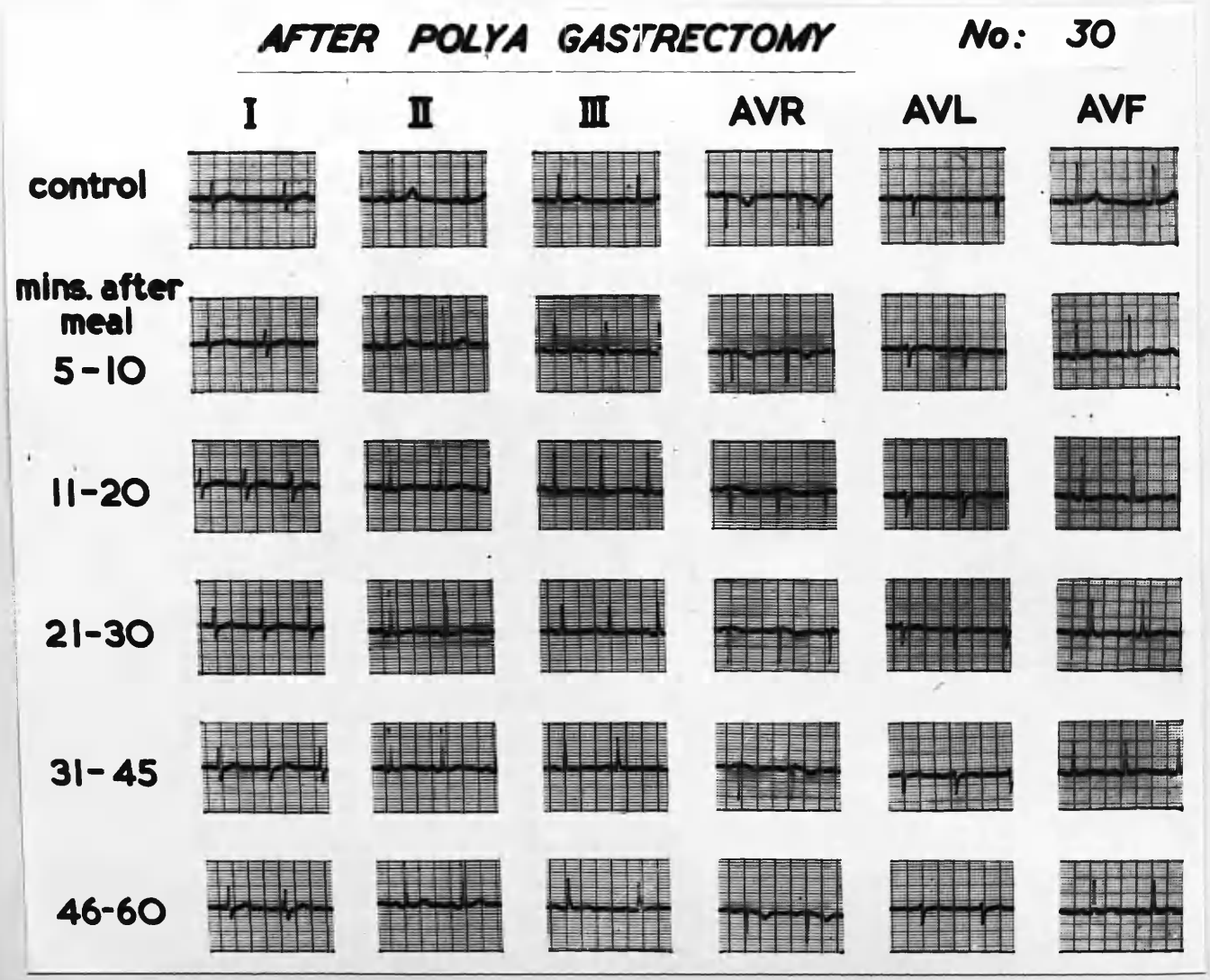
AFTER POLYA GASTRECTOMY No: 29



AFTER POLYA GASTRECTOMY - EXAMPLE NO. 29.

PATIENT 57.

TRACINGS	-	Control and at 5, 18, 26, 37 and 55 mins.
HEART RATE	-	Increased from 68 to 94
P	-	No change from the control
QRS	-	No change from the control
RT	-	ST sagging below isoelectric line in II, III and aVF and take off ST in aVR elevated at 18 - 26 mins.
T	-	Broader and runs into U wave
U	-	Increased in II, III and aVF at 18 - 55 mins. Maximal changes at 18 mins.

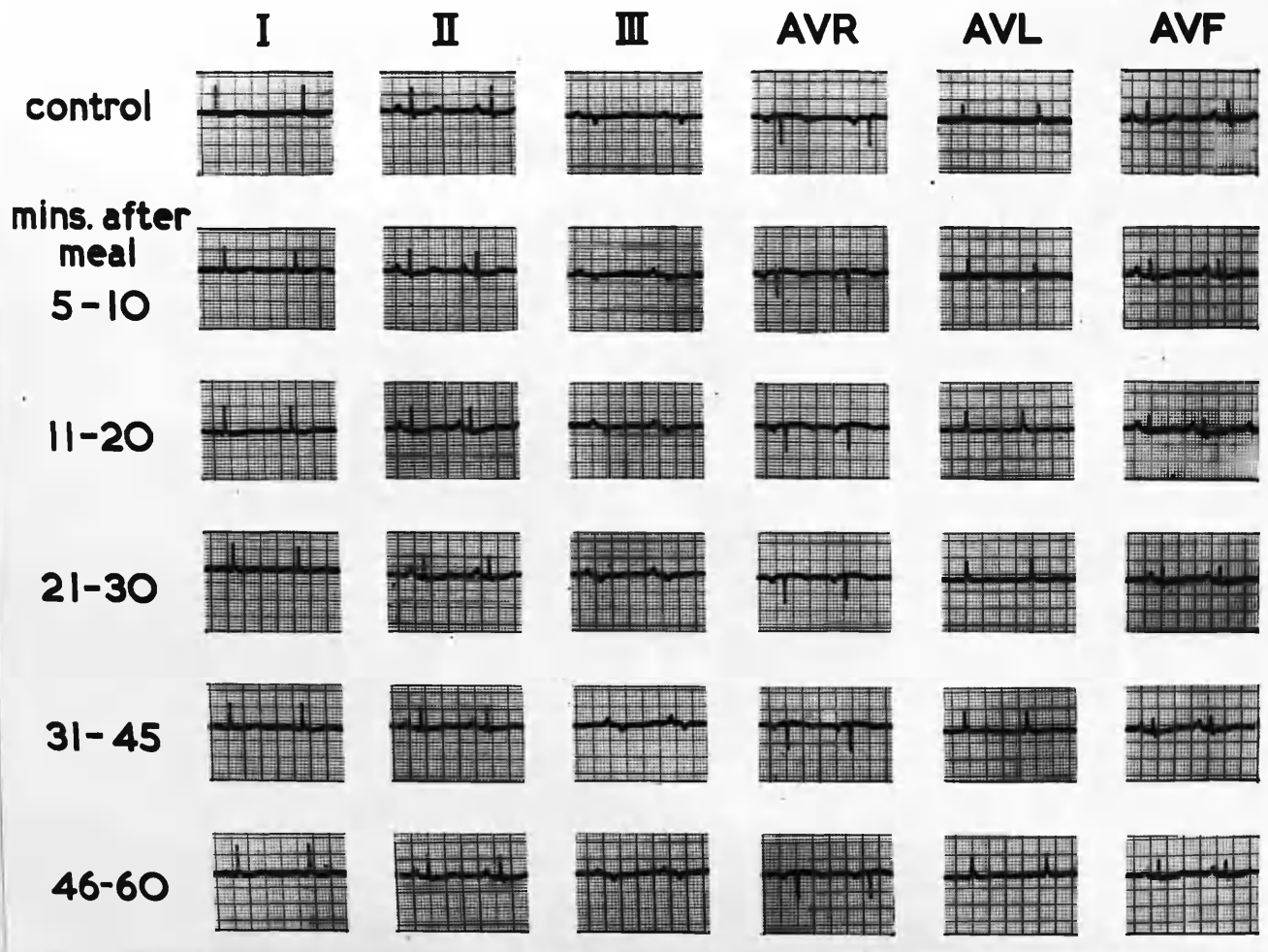

AFTER POLYA GASTRECTOMY
No: 30
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

AFTER POLYA GASTRECTOMY - EXAMPLE NO. 30.

PATIENT 58.

TRACINGS	-	Control and at 6, 13, 25, 45 and 60 mins.
HEART RATE	-	Increased from 60 to 100
P	-	No change from the control
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter and broader in II, III and aVF at 6 - 60 mins., in III biphasic at 25 mins. aVR less negative at 6 - 60 mins., and aVL becomes positive at 6 - 45 mins.
U	-	Increased in II and aVF at 6 - 60 mins. Maximal changes at 25 mins.

AFTER POLYA GASTRECTOMY No: 31

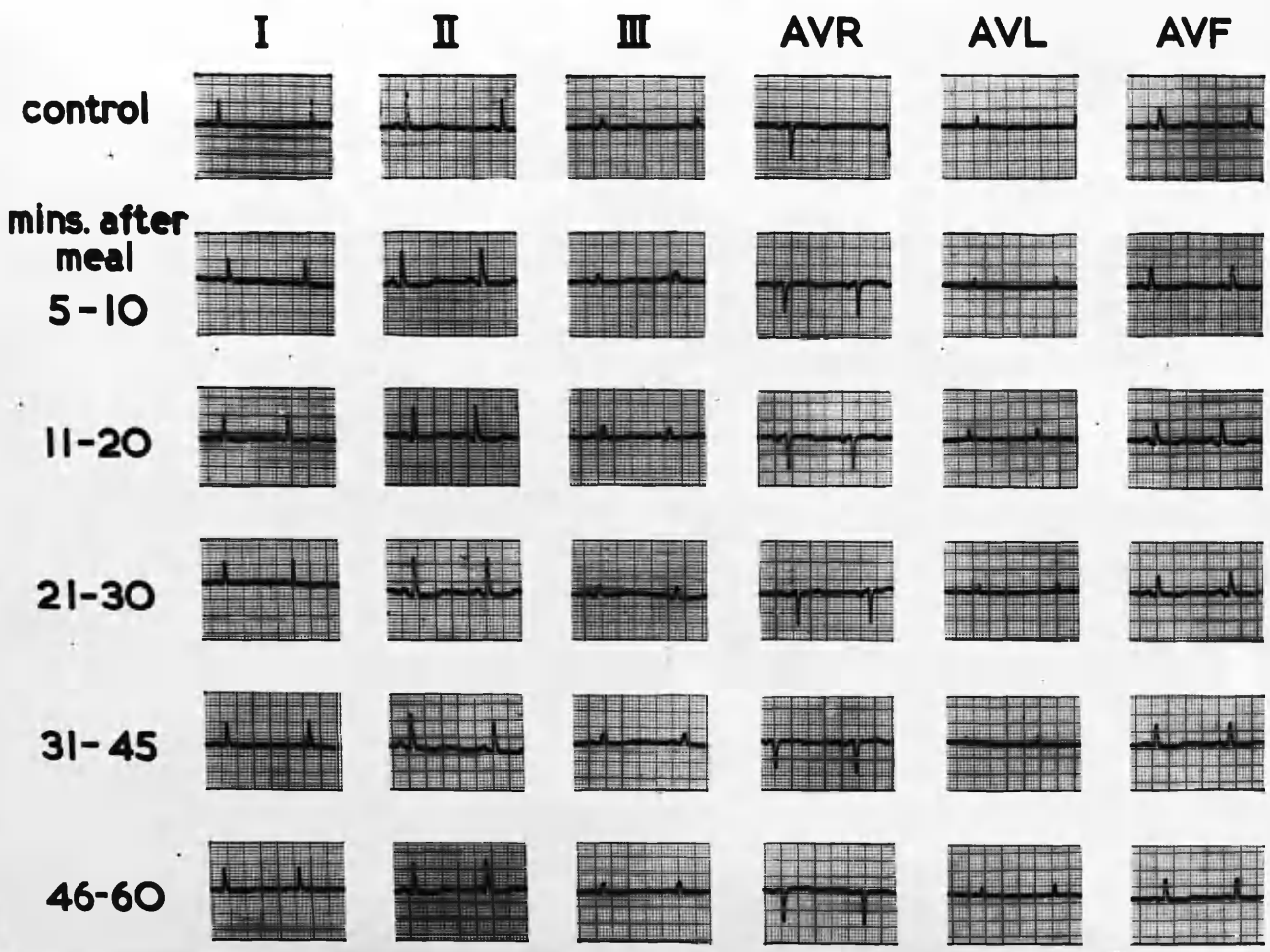


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 31.

PATIENT 38.

TRACINGS	-	Control and at 6, 12, 23, 36 and 55 mins.
HEART RATE	-	Increased from 64 to 88
P	-	Peaked in II, III and aVF at 12 and 23 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter in I, II, III and aVF at 12 - 36 mins., and less negative in aVR at 36 mins.
U	-	No change from the control Maximal changes at 23 mins.

AFTER POLYA GASTRECTOMY No: 32

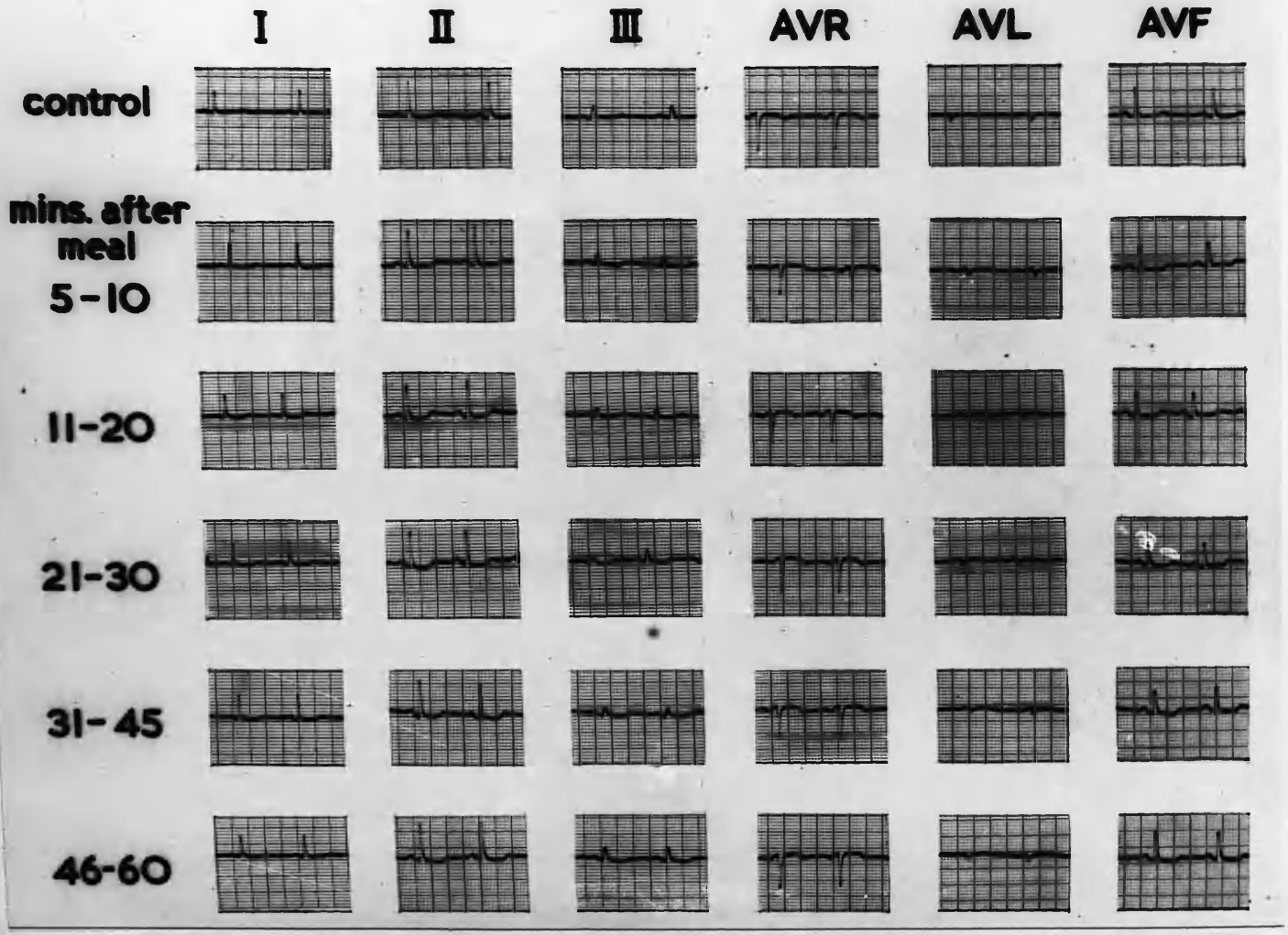


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 32.

PATIENT 13.

TRACINGS	-	Control and at 10, 20, 25, 40 and 58 mins.
HEART RATE	-	Increased from 54 to 78
P	-	Peaking in II and aVF at 20 and 25 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Biphasic in II, aVR and aVF at 10 - 58 mins.
U	-	No change from the control
		Maximal changes at 25 mins.

AFTER POLYA GASTRECTOMY No: 33

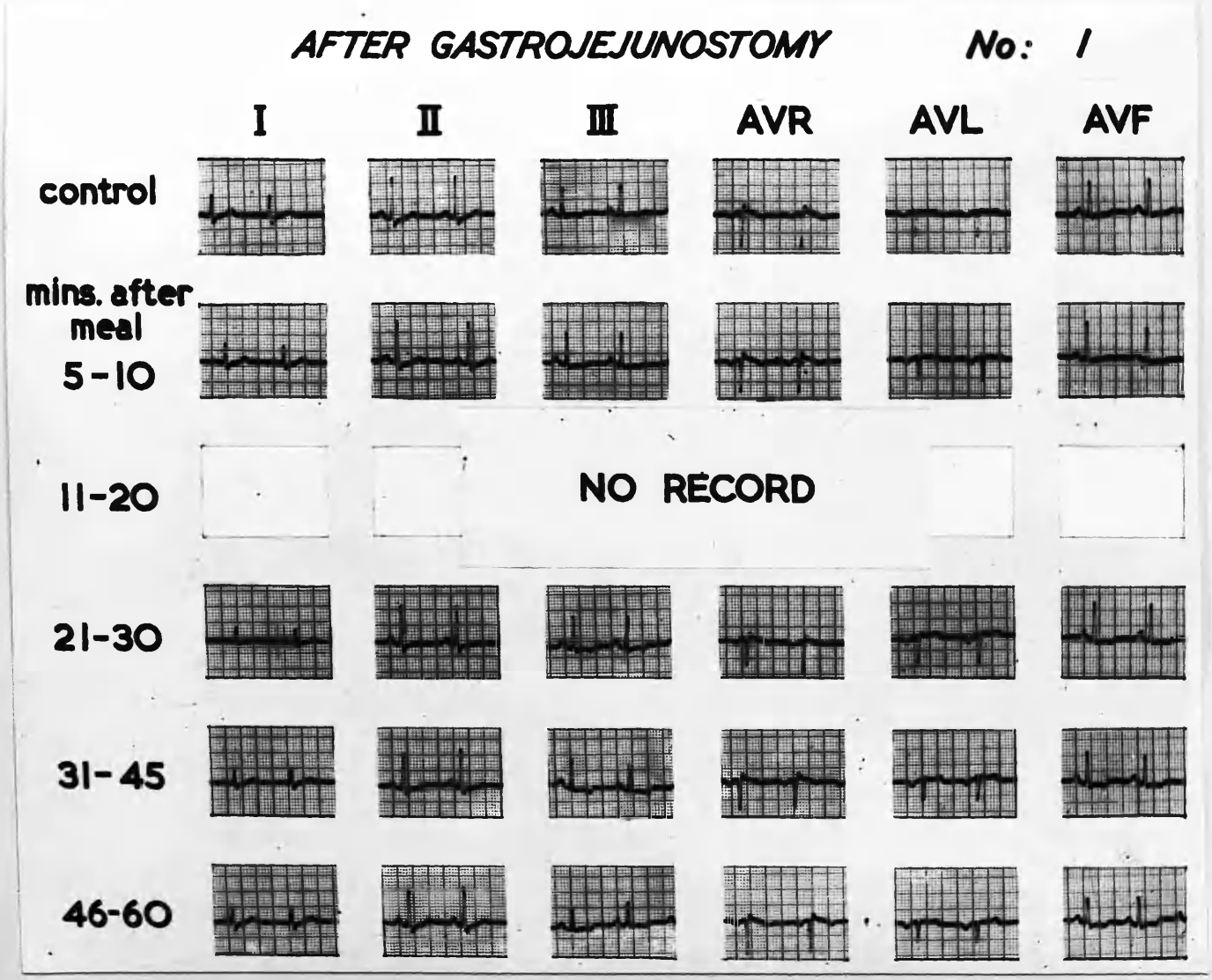


AFTER POLYA GASTRECTOMY - EXAMPLE NO. 33.

PATIENT 13.

TRACINGS	-	Control and at 10, 20, 25, 40 and 60 mins.
HEART RATE	-	Increased from 66 to 88
P	-	Peaking in II and aVF at 20 and 25 mins.
QRS	-	No change from control
RT	-	ST sagging below isoelectric line in II at 25 mins.
T	-	Biphasic in II, III and aVF at 10-60 mins.
U	-	No change from control
		Maximal changes at 25 mins.

ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE IN FIVE PATIENTS AFTER GASTROJEJUNOSTOMY WITH VAGOTOMY.


AFTER GASTROJEJUNOSTOMY
No: 1
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
NO RECORD
21-30
31-45
46-60

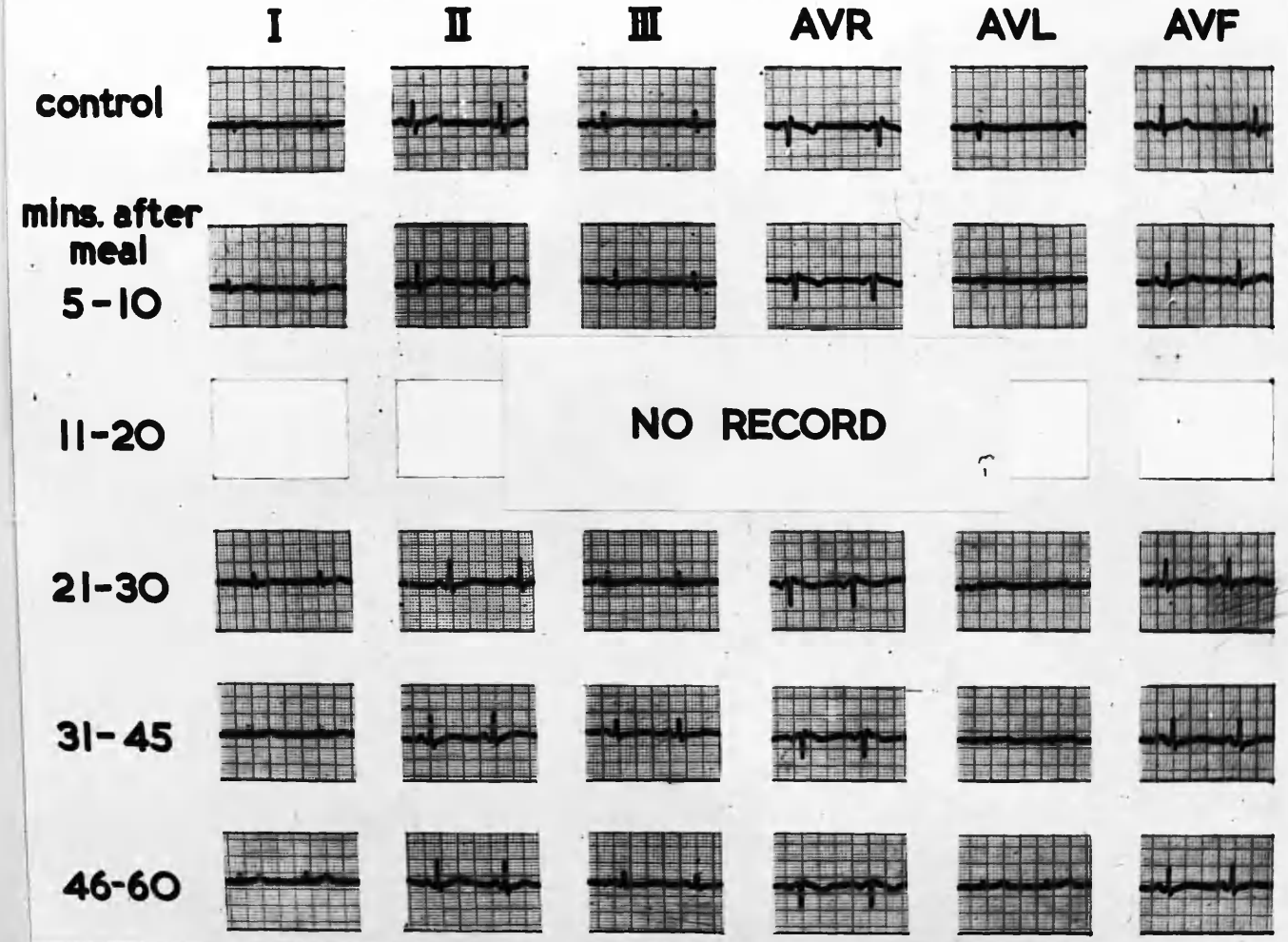
AFTER GASTROJEJUNOSTOMY - EXAMPLE NO. 1.

PATIENT 46.

TRACINGS	-	Control and at 5, 25, 37, and 60 mins.
HEART RATE	-	Increased from 74 to 84
P	-	Peaking in II, III and aVF at 25 and 37 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	I flatter at 25 mins., II biphasic at 25 and 38 mins., III more negative, aVL becomes positive at 25 - 60 mins., and aVF inverted at 25 - 60 mins.
U	-	No change from the control
		Maximal changes at 25 mins.

AFTER GASTROJEJUNOSTOMY

No: 2



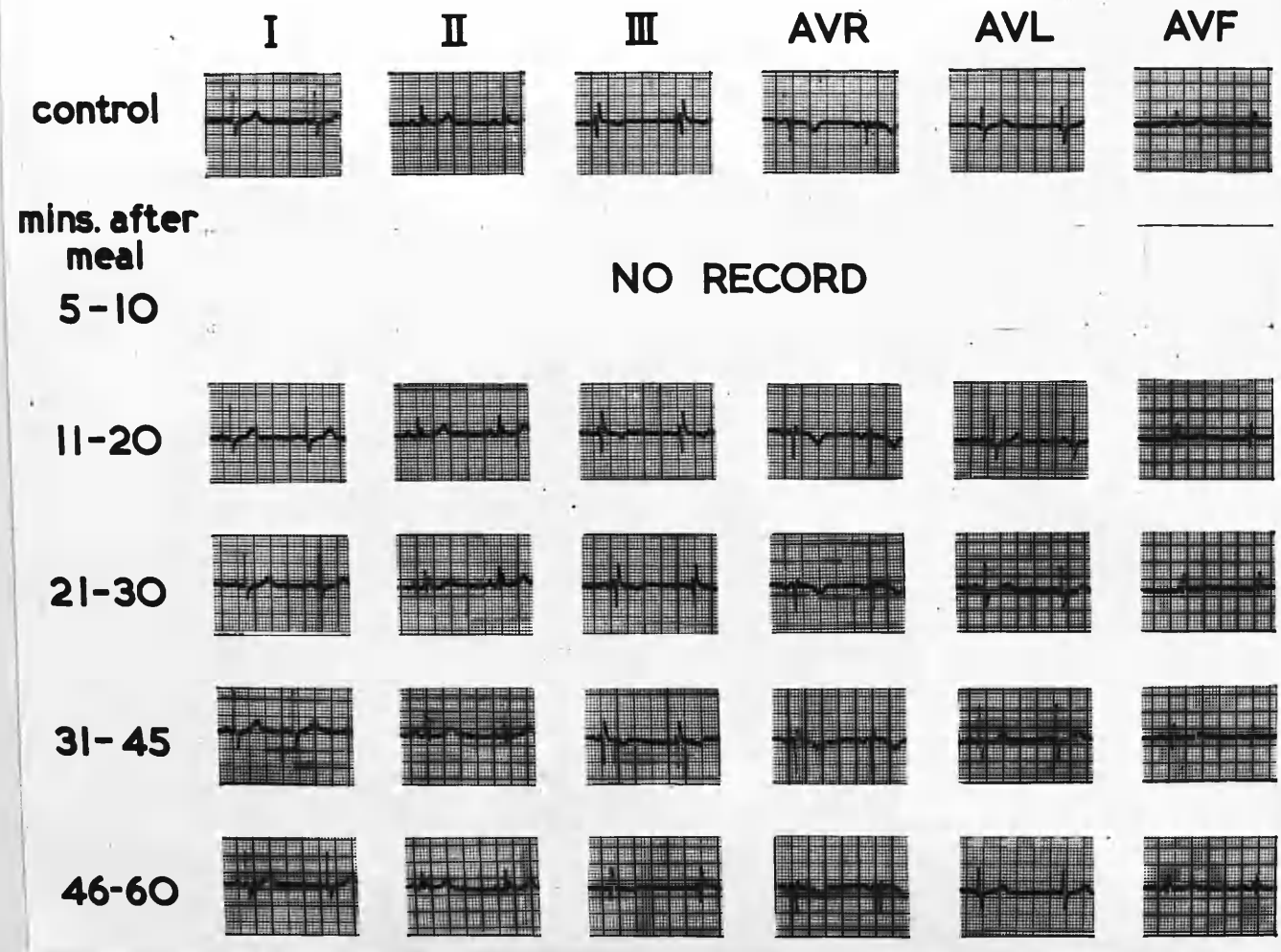
AFTER GASTROJEJUNOSTOMY - EXAMPLE NO. 2.

PATIENT 1.

TRACINGS	-	Control and 10, 28, 35 and 50 mins.
HEART RATE	-	Increased from 58 to 75
P	-	Peaked in II and aVF at 35 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter in II and aVF, less negative in aVR, biphasic in III at 28 and 35 mins.
U	-	No change from the control Maximal changes at 28 mins.

(Compare before operation Example No. 1).

AFTER GASTROJEJUNOSTOMY *No: 3*

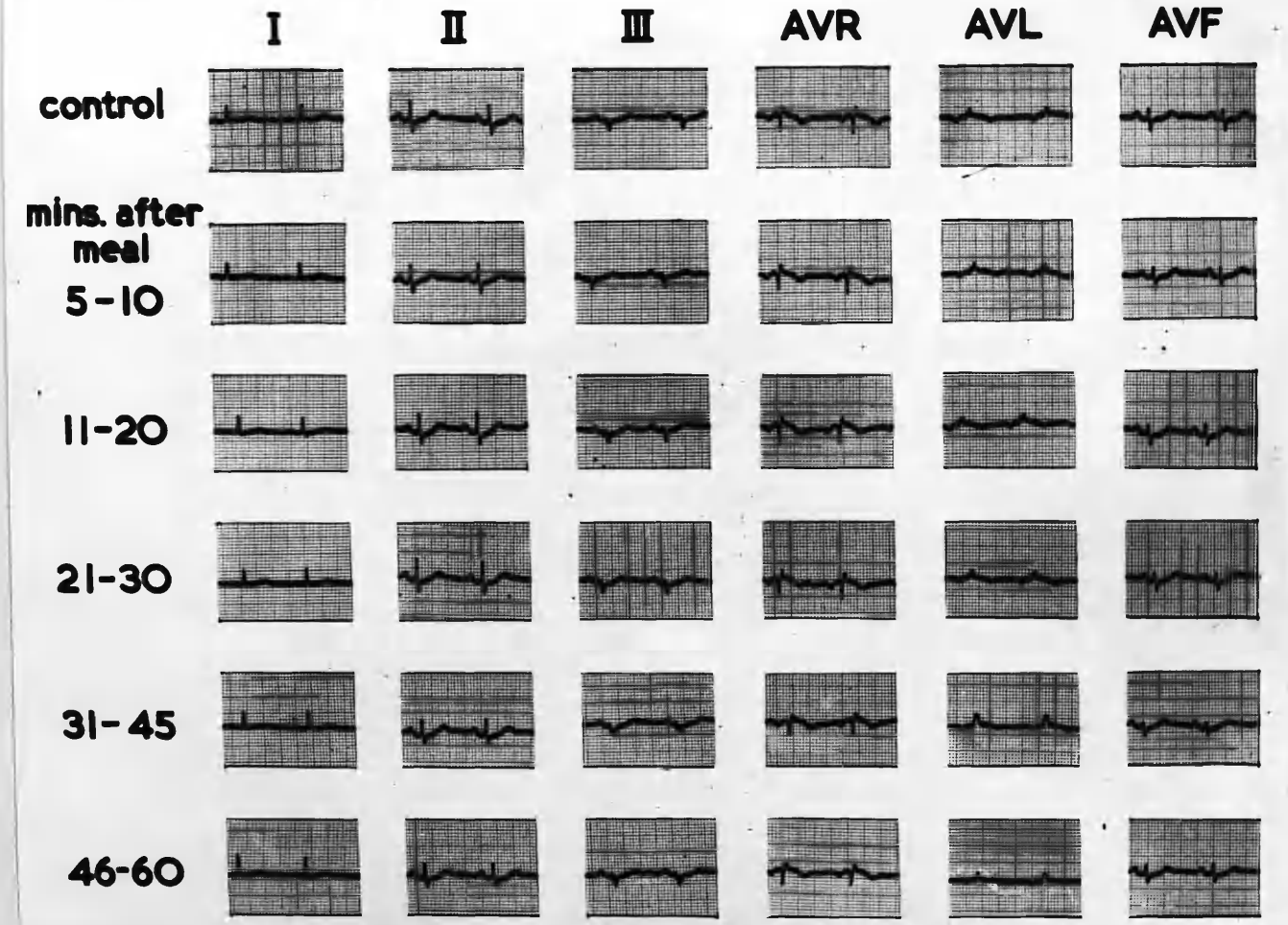


AFTER GASTROJEJUNOSTOMY - EXAMPLE NO. 3.

PATIENT 47.

TRACINGS	-	Control and at 18, 25, 38 and 60 mins.
HEART RATE	-	Increased from 60 to 68
P	-	Peaked in II at 38 mins.
QRS	-	No change from the control
RT	-	No change from the control
T	-	III becomes inverted at 38 mins., II flatter and aVR less negative at 25 mins.
U	-	Increased in II and aVF at 18 - 60 mins. Maximal changes at 38 mins.

AFTER GASTROJEJUNOSTOMY No: 4

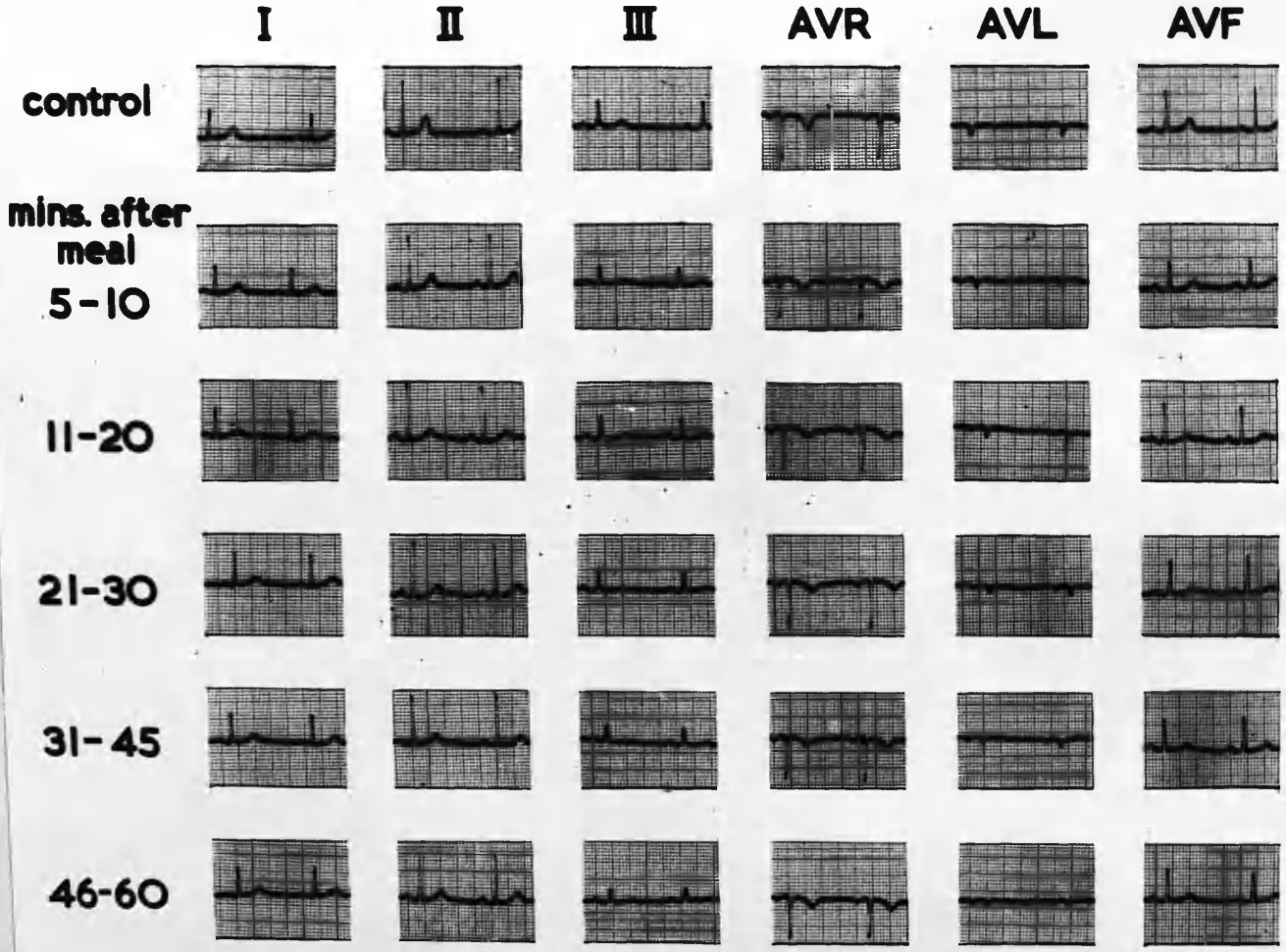


AFTER GASTROJEJUNOSTOMY - EXAMPLE NO. 4.

PATIENT 59.

TRACINGS	-	Control and at 7, 15, 25, 35 and 52 mins.
HEART RATE	-	Increased from 68 to 86
P	-	No change from the control
QRS	-	No change from the control
RT	-	ST take off below isoelectric line in II and aVF at 15 and 25 mins.
T	-	Flatter in II at 15 - 35 mins.
U	-	No change from the control
		Maximal changes at 25 mins.

AFTER GASTROJEJUNOSTOMY *No: 5*



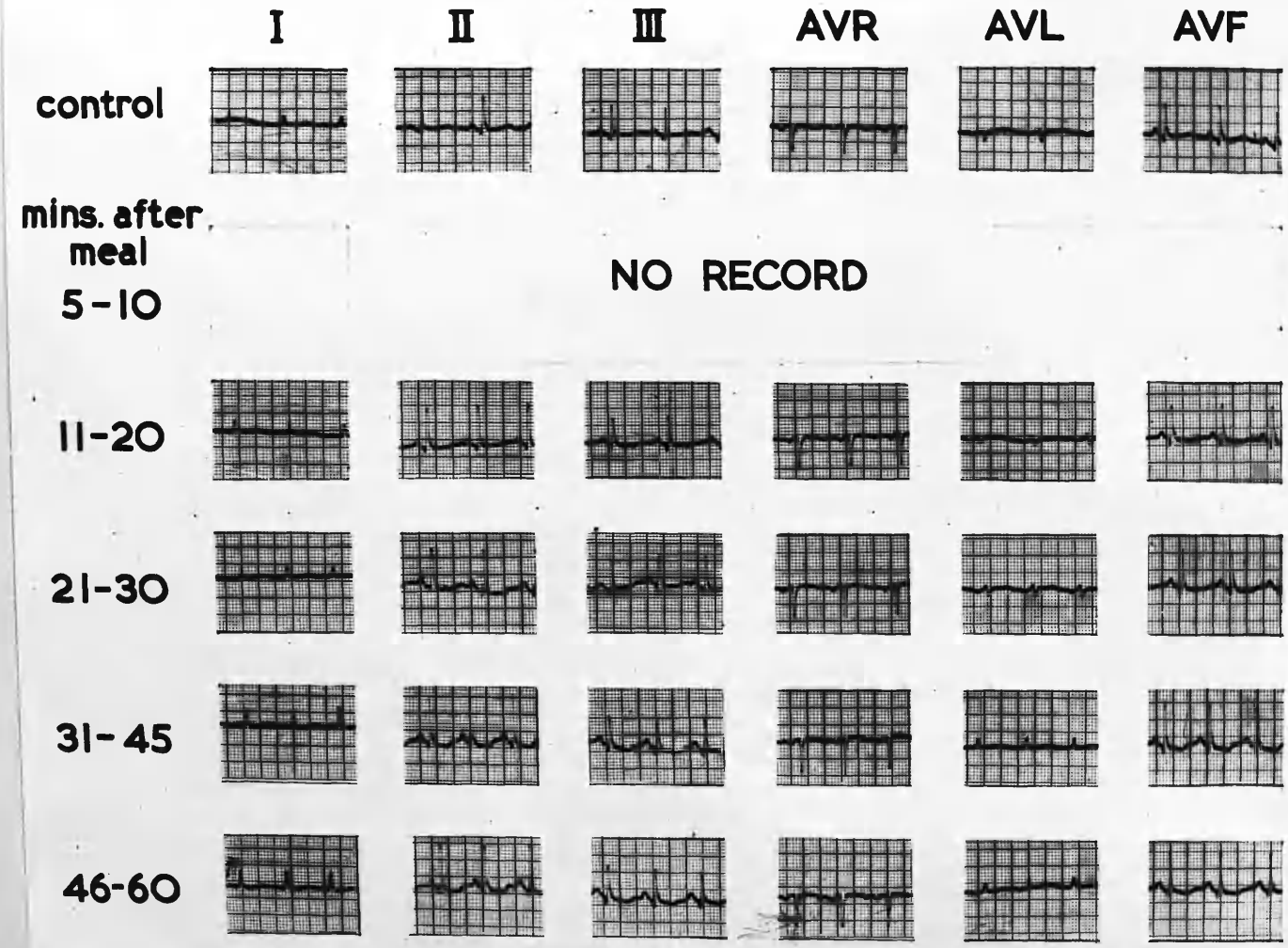
AFTER GASTROJEJUNOSTOMY - EXAMPLE NO. 5.

PATIENT 50.

TRACINGS	-	Control and at 10, 19, 25, 45 and 60 mins.
HEART RATE	-	Increased from 54 to 62
P	-	No change from the control
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter in I, II, III and aVF and less negative in aVR at 10 - 60 mins.
U	-	Increased in II and aVF at 19 - 45 mins. Maximal changes at 25 mins.

ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE IN FIVE PATIENTS AFTER BILLROTH I GASTRECTOMY.

AFTER BILLROTH I GASTRECTOMY *No: 1*



AFTER BILLROTH I GASTRECTOMY - EXAMPLE NO. 1.

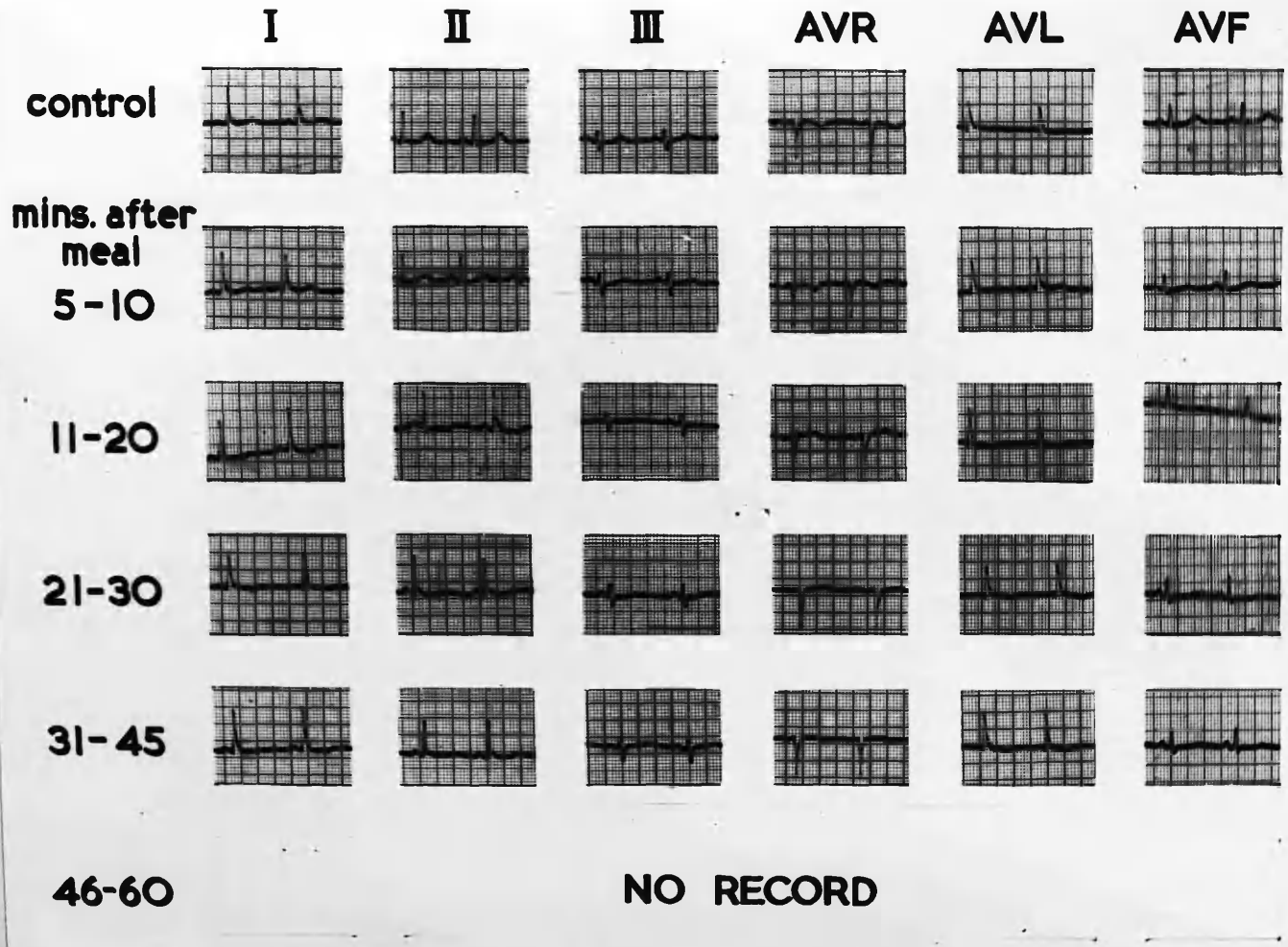
PATIENT 34.

TRACINGS	-	Control and at 20, 25, 35 and 48 mins.
HEART RATE	-	Increased from 86 to 116
P	-	Peaking in II, III and aVF at 25 - 48 mins.
QRS	-	R in II , III and aVF increased at 25 - 48 mins.
RT	-	ST sagging below isoelectric line in II, III and aVF at 25 - 48 mins.
T	-	Biphasic in II, III and aVF at 35 and 48 mins.
U	-	No change from the control

Maximal changes at 35 mins.

(Compare after Polya gastrectomy Example No. 2).

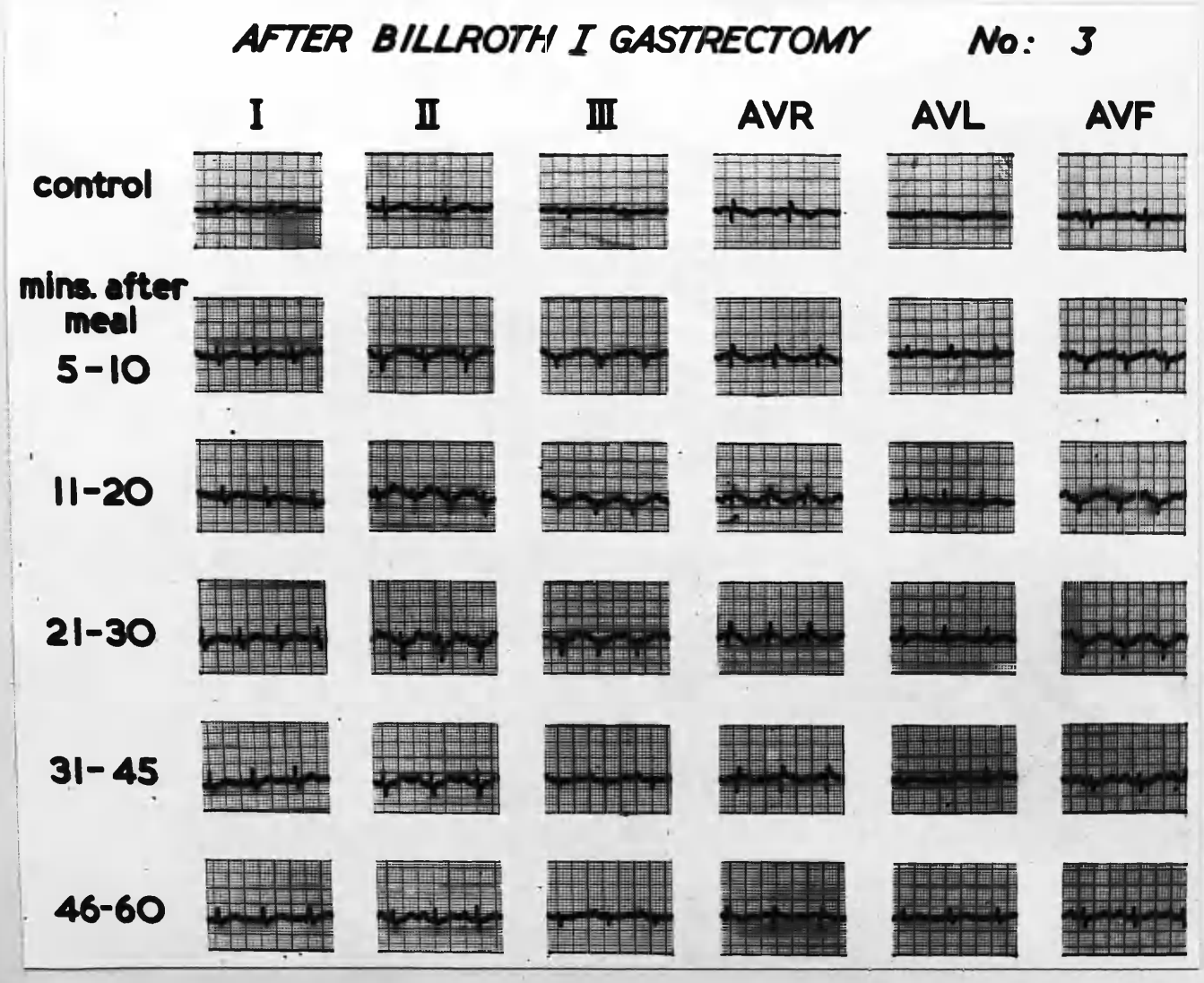
AFTER BILLROTH I GASTRECTOMY No: 2



AFTER BILLROTH I GASTRECTOMY - EXAMPLE NO. 2.

PATIENT 44.

TRACINGS	-	Control and at 5, 15, 25 and 40 mins.
HEART RATE	-	Unchanged at 76
P	-	No change from the control
QRS	-	R II increased at 15 mins.
RT	-	ST in II below isoelectric line
T	-	Flatter and then inverted in I, II and aVL and flatter in III and aVF. Becomes positive in aVR
U	-	No change from the control
		Maximal changes at 15 mins.


AFTER BILLROTH I GASTRECTOMY
No: 3
I
II
III
AVR
AVL
AVF
control
mins. after meal
5-10
11-20
21-30
31-45
46-60

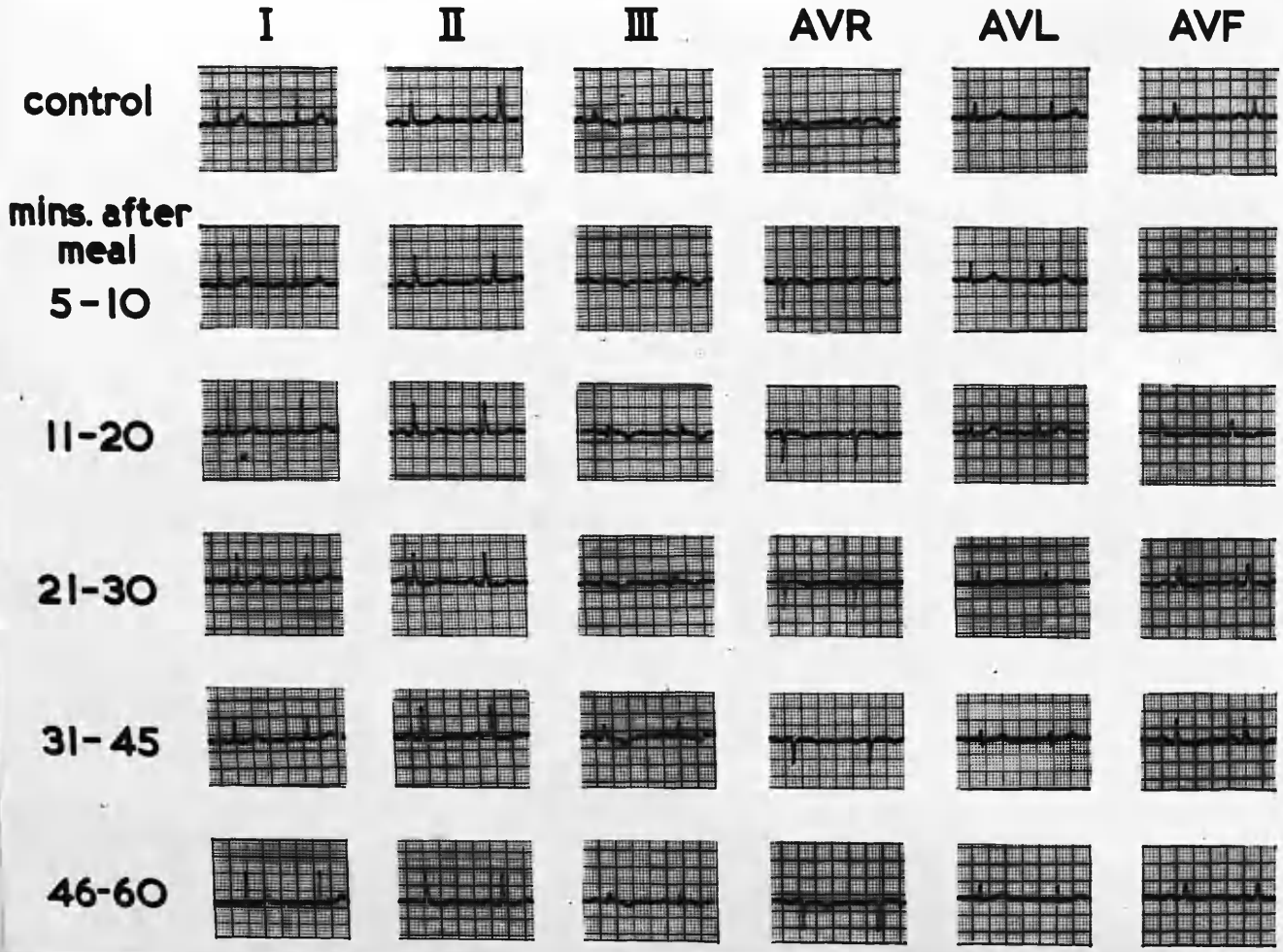
AFTER BILLROTH I GASTRECTOMY - EXAMPLE NO. 3.

PATIENT 15.

TRACINGS	-	Control and at 8, 13, 22, 38 and 53 mins.
HEART RATE	-	Increased from 66 to 124
P	-	Peaked in II, III and aVF at 13 - 38 mins.
QRS	-	S in II, III and aVF increased at 13 - 38 mins.
RT	-	ST low take off in II and sagging in III at 22 mins.
T	-	Slightly increased in II and III at 13 and 22 mins.
U	-	No change from the control
		Maximal changes at 22 mins.

(Compare after Polya gastrectomy Example No. 5).

AFTER BILLROTH I GASTRECTOMY No: 4

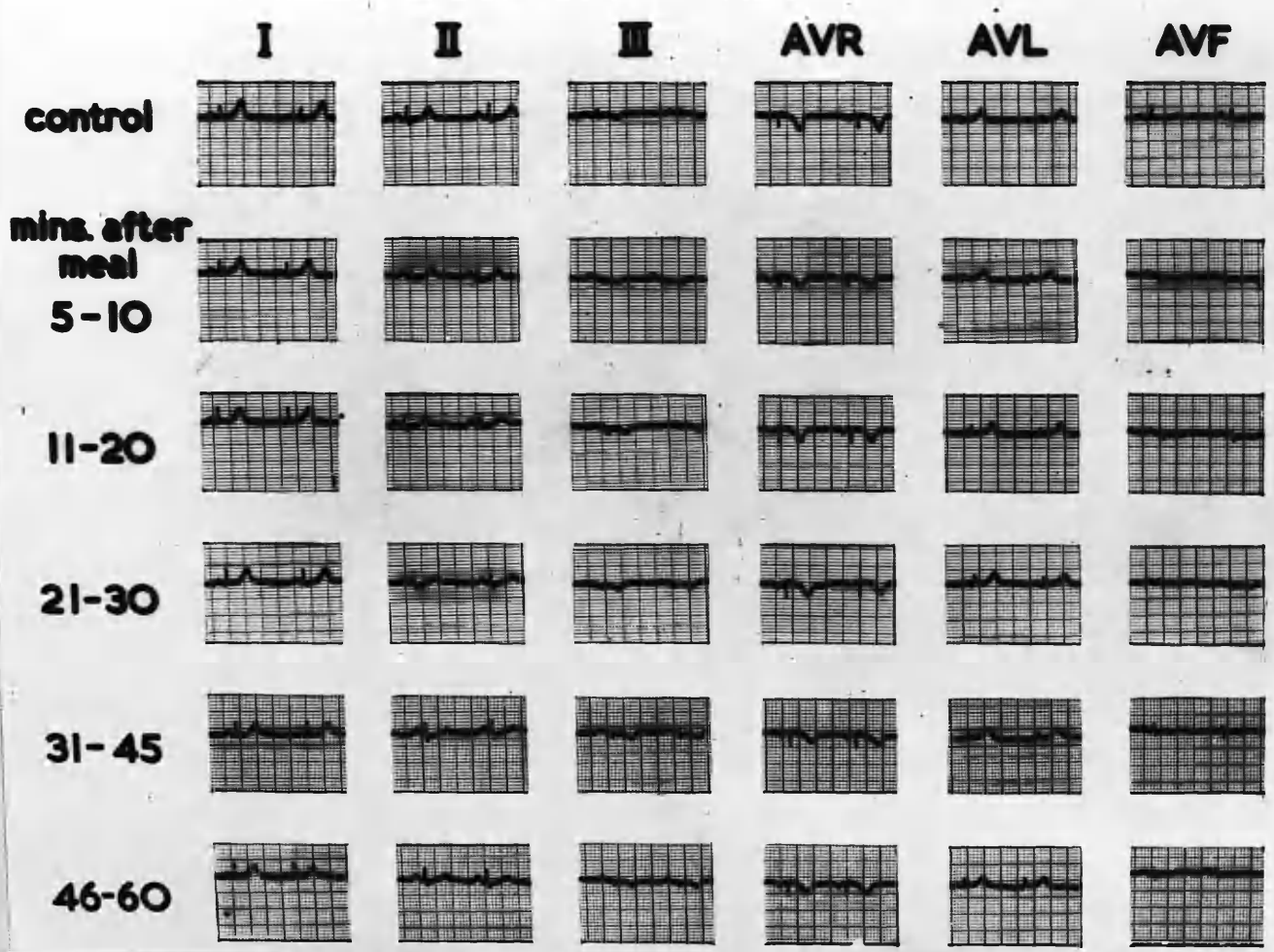


AFTER BILLROTH I GASTRECTOMY - EXAMPLE NO. 4.

PATIENT 41.

TRACINGS	-	Control and at 8, 12, 21, 35 and 63 mins.
HEART RATE	-	Increased from 62 to 70.
P	-	No change from the control
QRS	-	No change from the control
RT	-	No change from the control
T	-	Flatter to become biphasic in II at 35 mins., III inverted at 8 - 35 mins. aVR less negative at 21 and 35 mins.
U	-	Increased in II at 21 - 63 mins. Maximal changes at 35 mins.

AFTER BILLROTH I GASTRECTOMY No: 5



AFTER BILLROTH I GASTRECTOMY - EXAMPLE NO. 5.

PATIENT 22.

TRACINGS	-	Control and at 5, 12, 25, 39 and 60 mins.
HEART RATE	-	Increased from 62 to 72
P	-	No change from the control
QRS	-	No change from the control
RT	-	No change from the control
T	-	II flatter at 25 - 60 mins., and aVF biphasic at 5 - 60 mins.
U	-	Increased in II at 12 - 60 mins.
		Maximal changes at 39 mins.

PATIENT	CONTROL			AFTER GLUCOSE			Q-T RATIO INCREASED AFTER GLUCOSE
	RR Sec	Q-T Sec	QT RATIO	RR Sec	Q-T Sec	QT RATIO	
2	0.87	0.38	1.00	0.76	0.37	1.06	+
3	0.84	0.37	1.00	0.61	0.35	1.12	+
5	1.00	0.37	0.92	0.84	0.34	0.92	-
6	0.76	0.37	1.02	0.72	0.36	1.06	+
10	0.80	0.37	1.03	0.72	0.38	1.11	+
12	0.78	0.35	0.98	0.68	0.36	1.04	+
13	1.12	0.46	1.08	0.74	0.41	1.19	+
14	0.92	0.40	1.02	0.65	0.36	1.12	+
15	0.72	0.37	1.08	0.55	0.32	1.08	-
18	0.92	0.42	1.04	0.72	0.36	1.06	-
23	1.18	0.43	0.97	1.12	0.44	1.03	+
24	0.90	0.38	0.99	0.76	0.36	1.03	+
25	0.88	0.38	1.00	0.68	0.37	1.12	+
26	1.18	0.44	1.00	0.81	0.48	1.32	+
27	0.78	0.36	1.01	0.62	0.34	1.08	+
29	1.06	0.42	1.00	0.76	0.40	1.15	+
30	0.96	0.41	1.04	0.70	0.39	1.16	+
31	0.78	0.38	1.02	0.68	0.37	1.12	+
32	0.79	0.36	1.00	0.71	0.38	1.13	+
34	0.71	0.38	1.13	0.54	0.33	1.12	-
37	1.00	0.40	0.99	0.66	0.35	1.08	+
38	0.96	0.37	0.94	0.68	0.36	1.09	+
39	0.78	0.37	1.05	0.68	0.36	1.09	+
40	0.78	0.35	0.98	0.62	0.33	1.04	+
42	1.04	0.40	0.97	1.00	0.38	0.95	-
43	0.92	0.40	1.04	0.78	0.38	1.08	+
45	0.69	0.34	1.02	0.53	0.32	1.10	+
51	0.76	0.36	1.03	0.49	0.32	1.14	+
56	1.04	0.41	1.00	0.80	0.40	1.10	+
57	0.84	0.36	0.97	0.66	0.34	1.05	+
58	0.96	0.37	0.94	0.65	0.36	1.11	+

THE Q-T RATIOS OF 31 PATIENTS AFTER GASTRECTOMY WHO HAD ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ml. 50 per cent GLUCOSE.

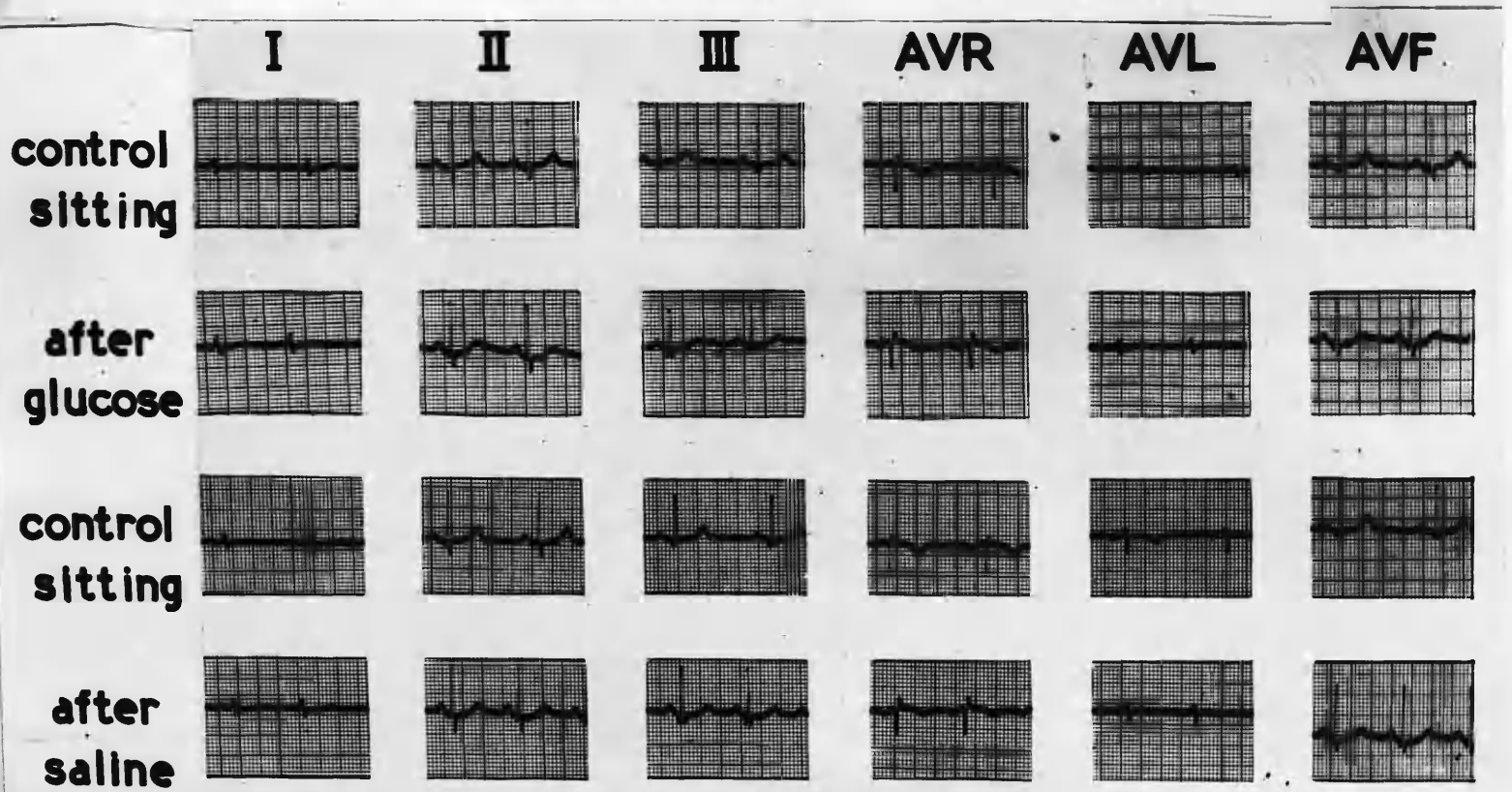
ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE.

ADDITIONAL EXPERIMENTS TO ELUCIDATE THE MECHANISM OF THE CHANGES.

In these tracings, the maximal electrocardiographic changes resulting from the additional experiments are compared with the maximal changes following a meal of 100 ml. 50 per cent glucose in the sitting position in the same patient.

EFFECT OF	ADDITIONAL EXPERIMENT EXAMPLE NOS.
HYPERTONIC SALINE	1 - 3
ADRENALINE	4 - 9
POSTURE	8 - 17
ATROPINE	13 - 17
GLUCOSE IN RECUMBENCY	18 - 22
EFFECT OF ERGOTAMINE	EXAMPLES NOS. 1 - 9

ADDITIONAL EXPERIMENT NO. 1



ADDITIONAL EXPERIMENT - EXAMPLE NO. 1.

PATIENT 27.

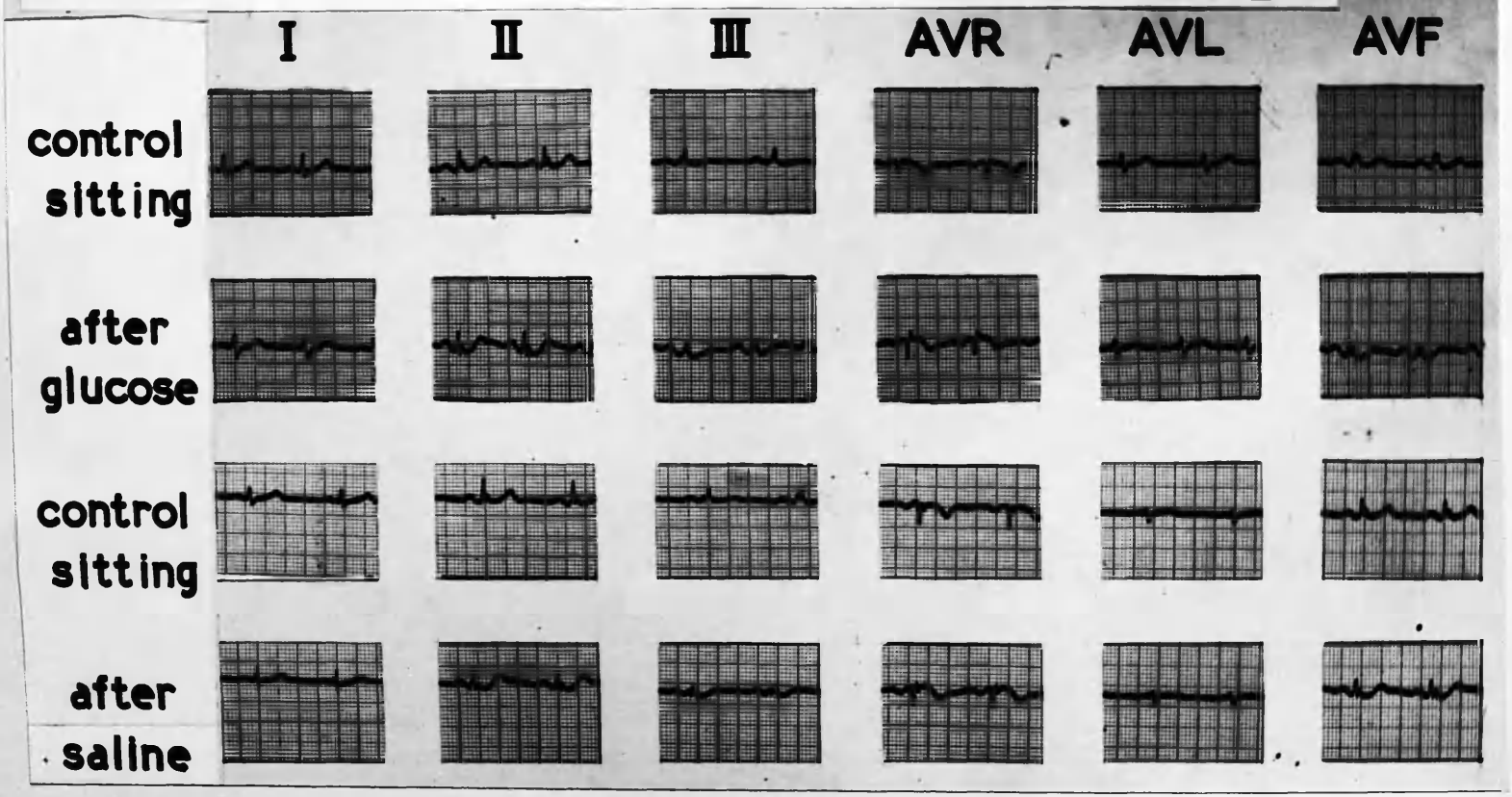
GLUCOSE Control and 23 mins.

Heart rate increased from 66 to 94: P:- peaked II, III and aVF: QRS:- No change from the control: RT:- ST segment sagging below isoelectric line II and aVF and less negative aVR: U:- II, III and aVF.

HYPERTONIC SALINE Control and 20 mins.

Heart rate increased from 66 to 88: P:- II, III and aVR: QRS:- No change from the control: RT:- ST low take off aVF: T:- flatter in II, III and aVF and less negative aVR: U:- No change from the control.

ADDITIONAL EXPERIMENT NO. 2



ADDITIONAL EXPERIMENT - EXAMPLE NO. 2.

PATIENT 57.

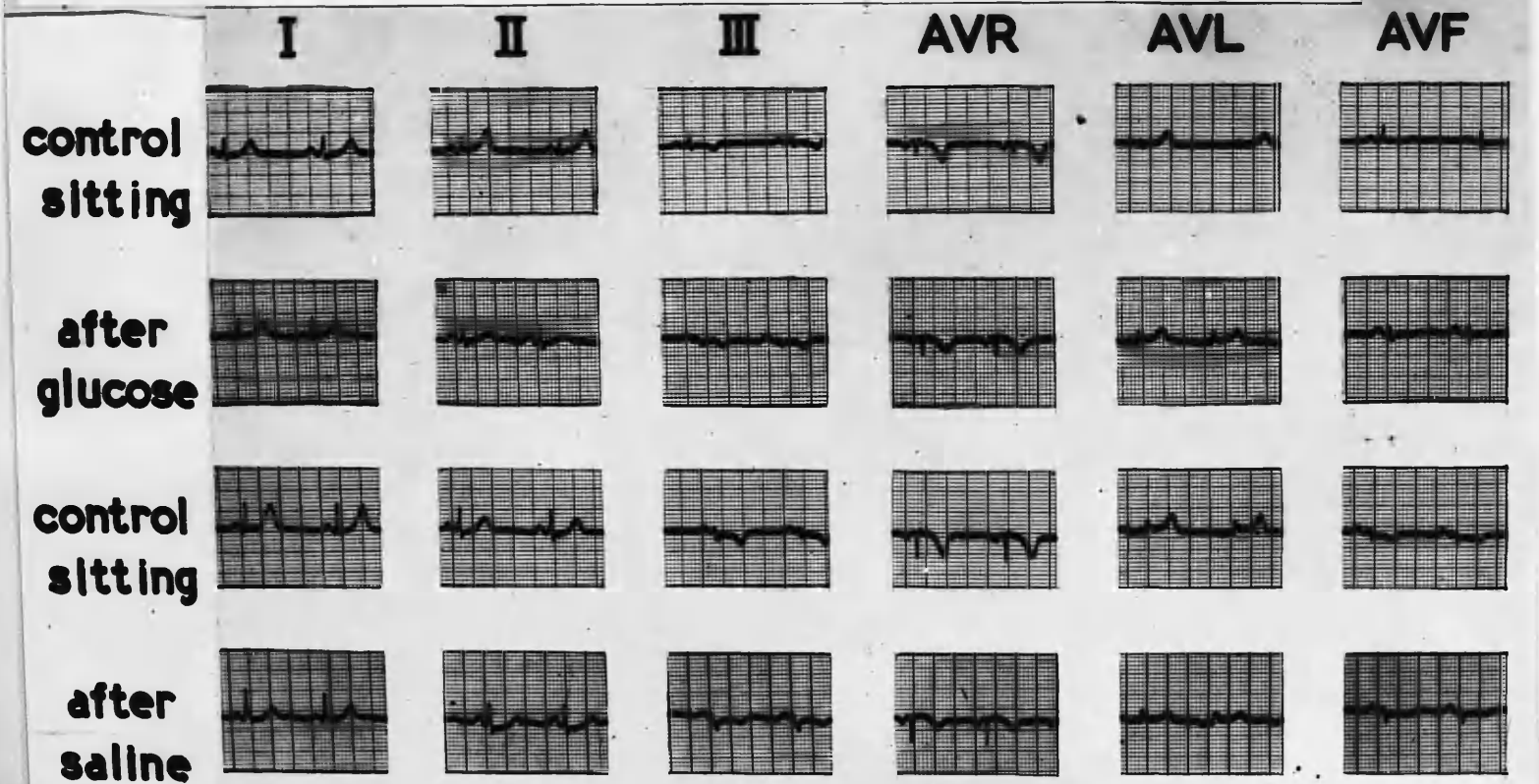
GLUCOSE Control and 18 mins.

Heart rate increased from 68 to 94: P:- No change from the control: QRS:- No change from the control: RT:- ST sagging below the isoelectric line II, III and aVF and take off ST aVR elevated: T:- broader and runs into U wave: U:- increased II, III and aVF

HYPERTONIC SALINE Control and 18 mins.

Heart rate increased from 66 to 75: P:- No change from the control: QRS:- No change from the control: RT:- ST sagging below isoelectric line II, III and aVF: T:- broader and runs into U waves: U:- increased II, III and aVF.

ADDITIONAL EXPERIMENT NO. 3



ADDITIONAL EXPERIMENT - EXAMPLE NO. 3.

PATIENT 22.

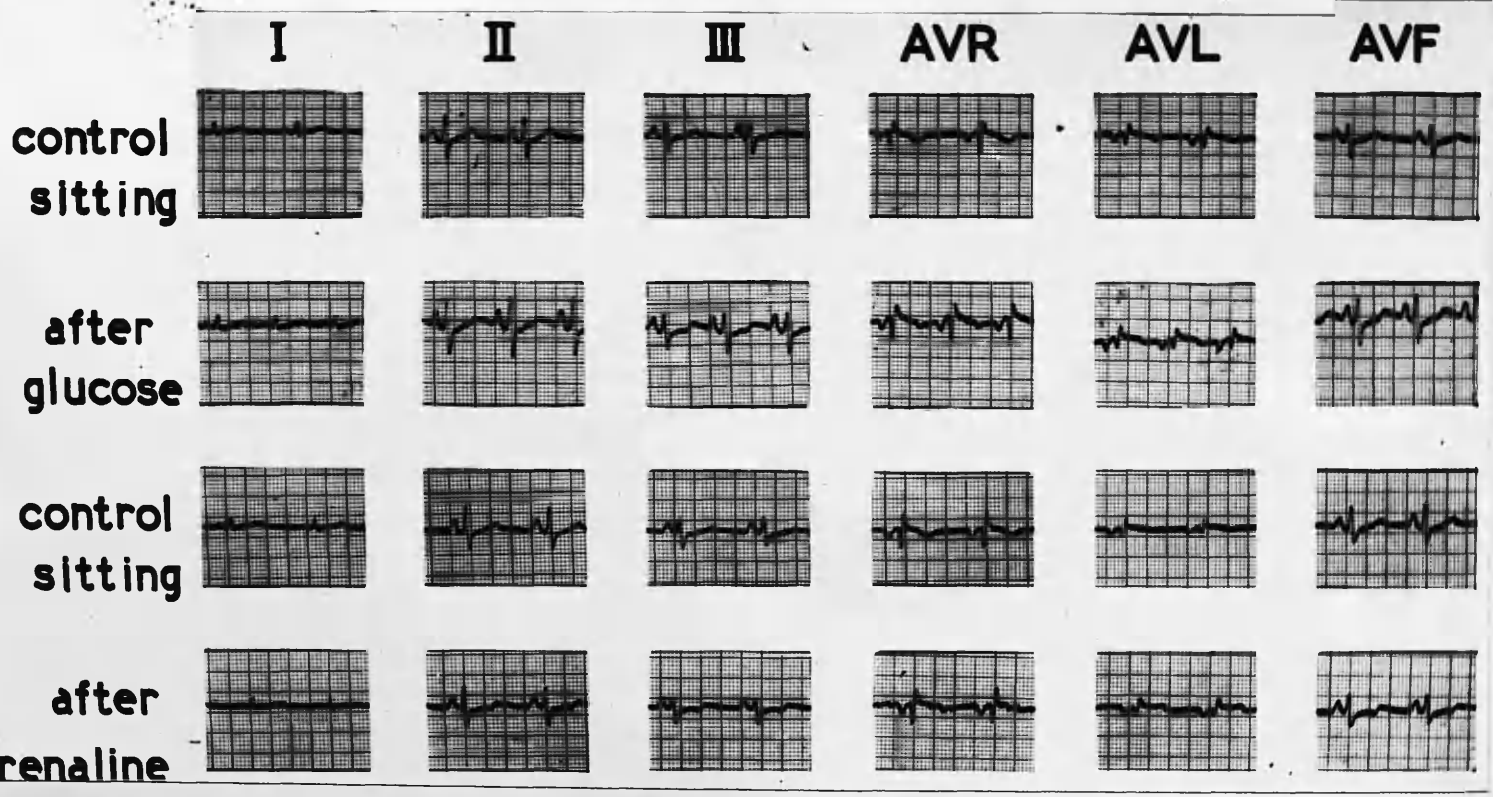
GLUCOSE Control and 39 mins.

Heart rate increased from 62 to 72: P:- peaked II: QRS:- No change from the control: RT:- No change from the control: T:- flatter II and biphasic aVF: less negative aVR: U:- increased in II.

HYPERTONIC SALINE Control and 35 mins.

Heart rate increased from 66 to 77: P:- peaked II: QRS:- No change from the control: RT:- ST below isoelectric line II: T:- flatter I, II, aVL and less negative aVR: U:- increased in II.

ADDITIONAL EXPERIMENT NO. 4



ADDITIONAL EXPERIMENT - EXAMPLE NO. 4.

PATIENT 40.

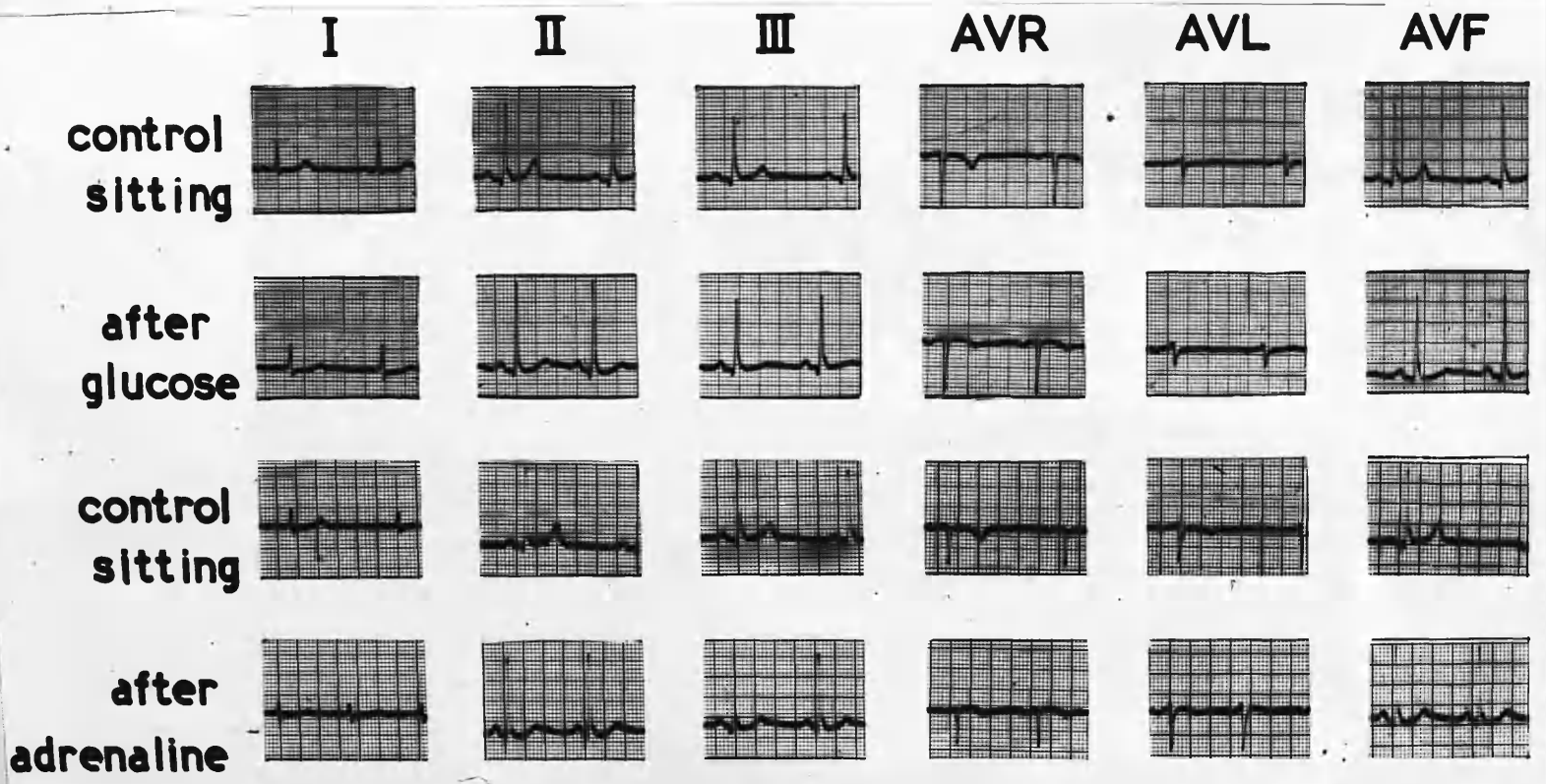
GLUCOSE Control and 30 mins.

Heart rate increased from 72 to 100: P:- peaked II, III and aVF: QRS:- increased II, III and aVF: RT:- ST below isoelectric line in II, III and aVF: T:- flatter and broader II, III and aVF, biphasic III, less negative aVR, biphasic aVL: U:- No change from the control

ADRENALINE Control and 33 mins.

Heart rate increased from 72 to 76: P:- peaked aVF: QRS:- No change from the control: RT:- ST below isoelectric line II, III and aVF: elevated aVL: T:- biphasic II, III and aVR, aVL and aVF: U:- No change from the control.

ADDITIONAL EXPERIMENT NO. 5



ADDITIONAL EXPERIMENT - EXAMPLE NO. 5.

PATIENT 29.

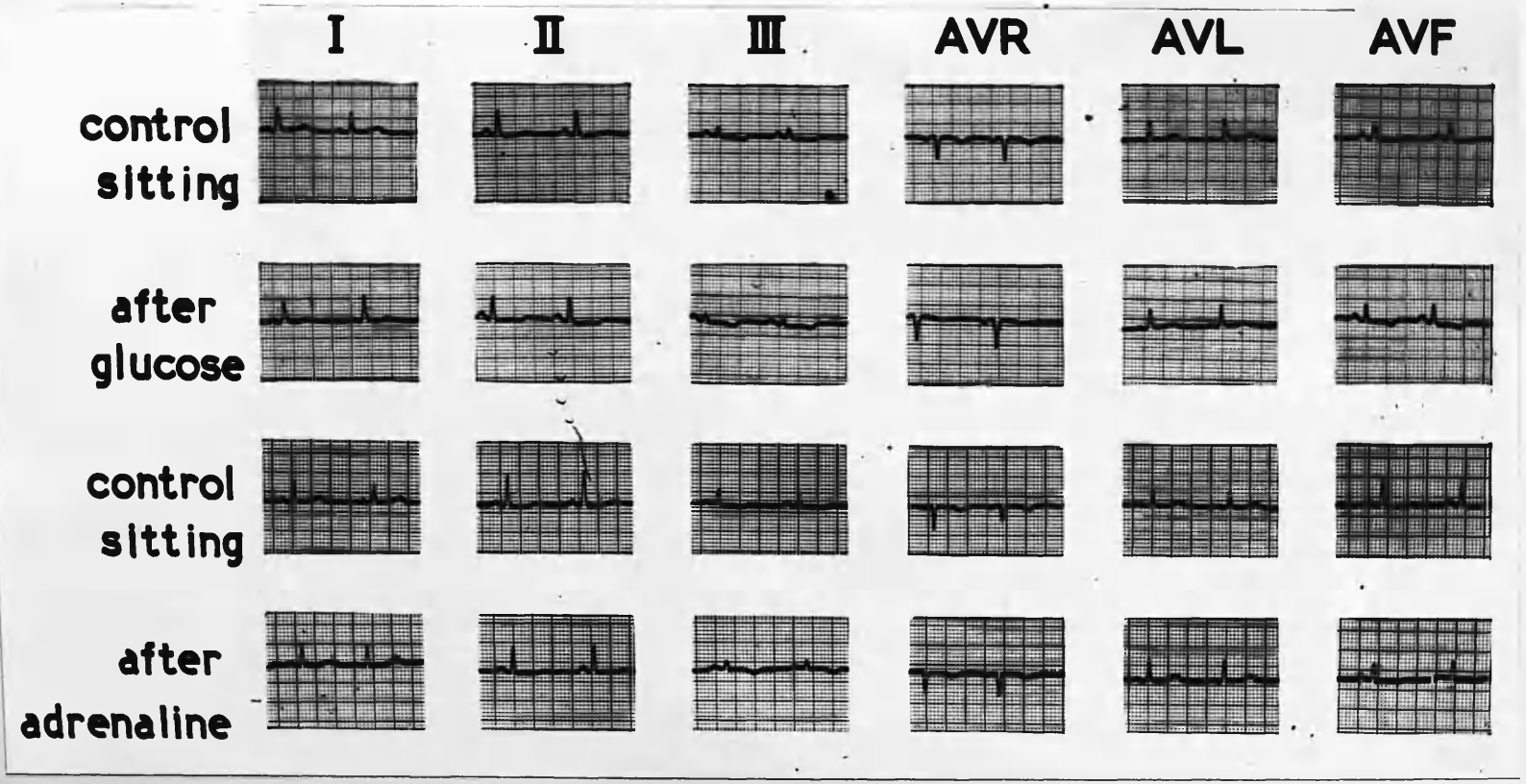
GLUCOSE Control and 38 mins.

Heart rate increased from 58 to 74: P:- peaked II and aVF: QRS:- R II, III and aVF increased: RT:- No change from the control: T:- flatter and broader I, II, III and aVF and less negative aVR: U:- increased II and aVF.

ADRENALINE Control and 34 mins.

Heart rate increased from 54 to 75: P:- peaked II, III and aVF: QRS:- No change from the control: RT:- ST sagging below isoelectric line II and aVF: T:- I, II, III and aVF flatter and broader and less negative aVL and aVR: U:- increased II and aVF.

ADDITIONAL EXPERIMENT NO. 6



ADDITIONAL EXPERIMENT - EXAMPLE NO. 6.

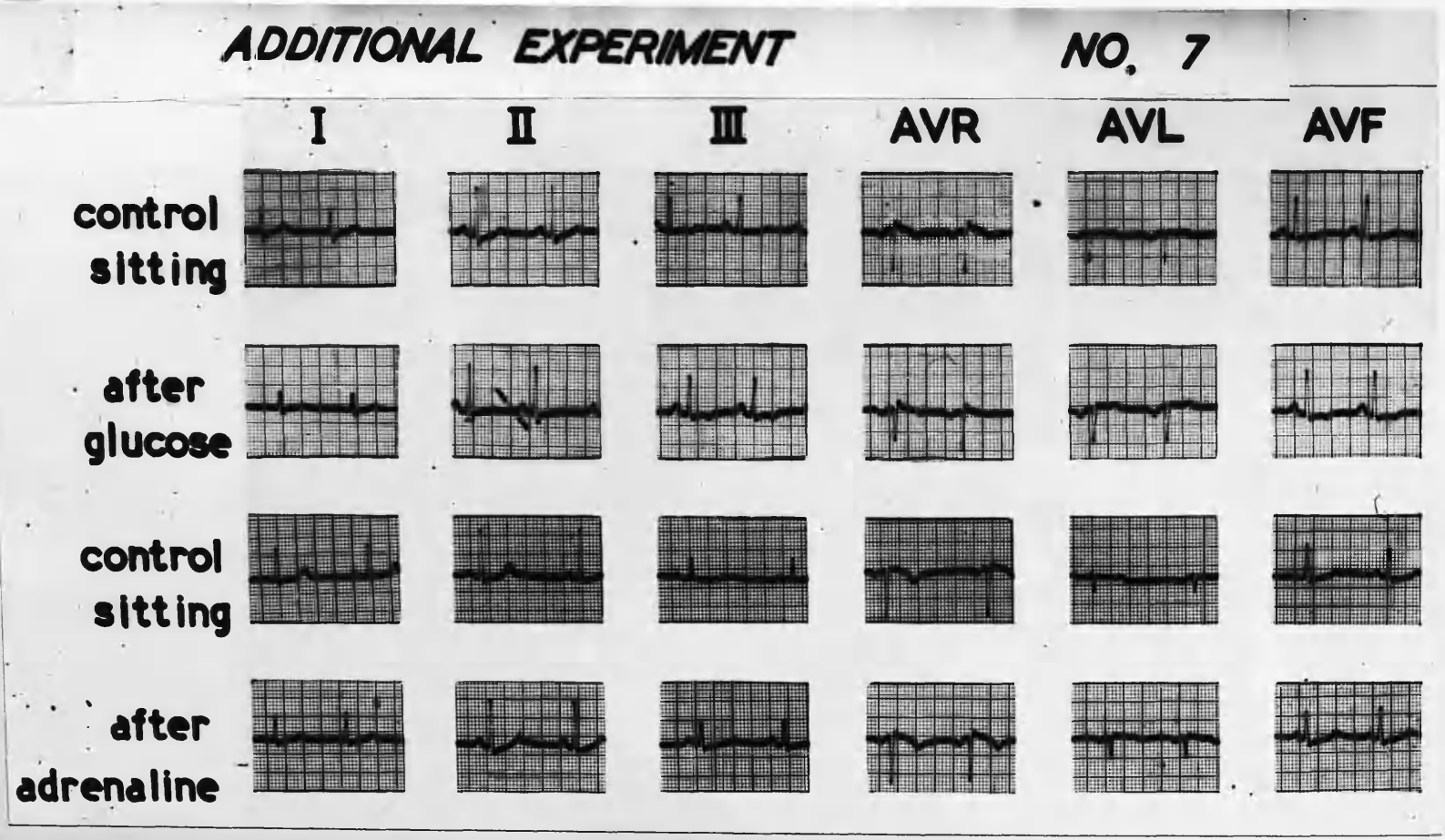
PATIENT 6.

GLUCOSE Control and 23 mins.

Heart rate increased from 75 to 88: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- flatter I, biphasic II, more negative III, less negative aVR, inverted aVF: U:- No change from the control.

ADRENALINE Control and 12 mins.

Heart rate increased from 76 to 79: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- flatter II, inverted III, biphasic aVF: U:- No change from the control.


ADDITIONAL EXPERIMENT
NO. 7
I
II
III
AVR
AVL
AVF
control sitting
after glucose
control sitting
after adrenaline

ADDITIONAL EXPERIMENT - EXAMPLE NO. 7.

PATIENT 46.

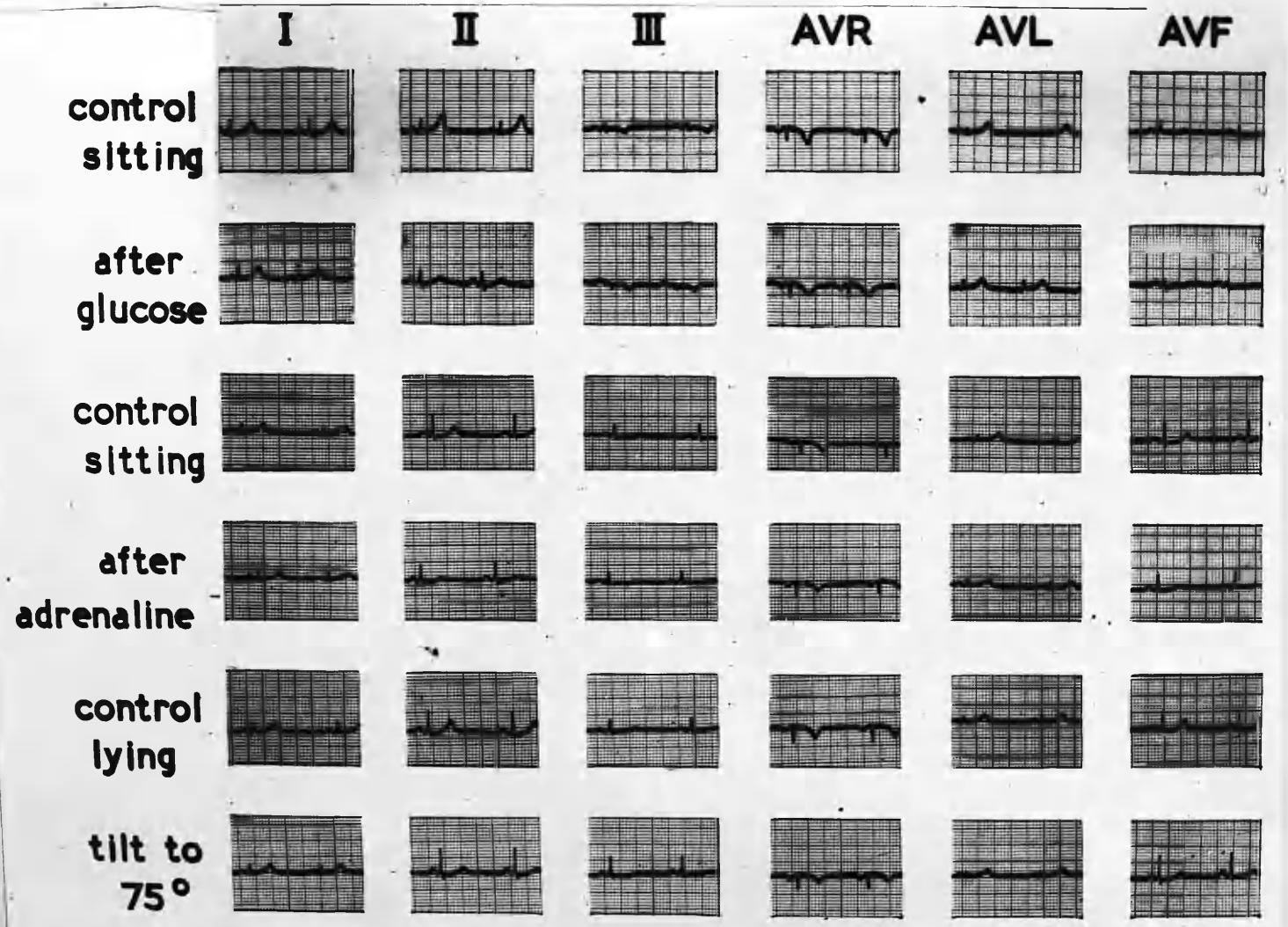
GLUCOSE Control and 25 mins.

Heart rate increased from 74 to 84: P:- peaking II, III and aVF: QRS:- No change from the control: RT:- No change from the control: T:- flatter I, biphasic II, more negative III, aVR less negative, aVL positive: aVR inverted: U:- No change from the control.

ADRENALINE Control and 33 mins.

Heart rate increased from 58 to 72: P:- peaking III: QRS:- No change from the control: RT:- low ST take off II, III and aVF: T:- flatter I, II, and less negative aVR: U:- No change from the control.

ADDITIONAL EXPERIMENT NO. 8



ADDITIONAL EXPERIMENT - EXAMPLE NO. 8.

PATIENT 22.

GLUCOSE Control and 39 mins.

Heart rate increased from 62 to 84: P:- peaked II and aVF: QRS:- No change from the control: RT:- No change from the control: T:- flatter II, less negative aVR, inverted aVF: U:- II.

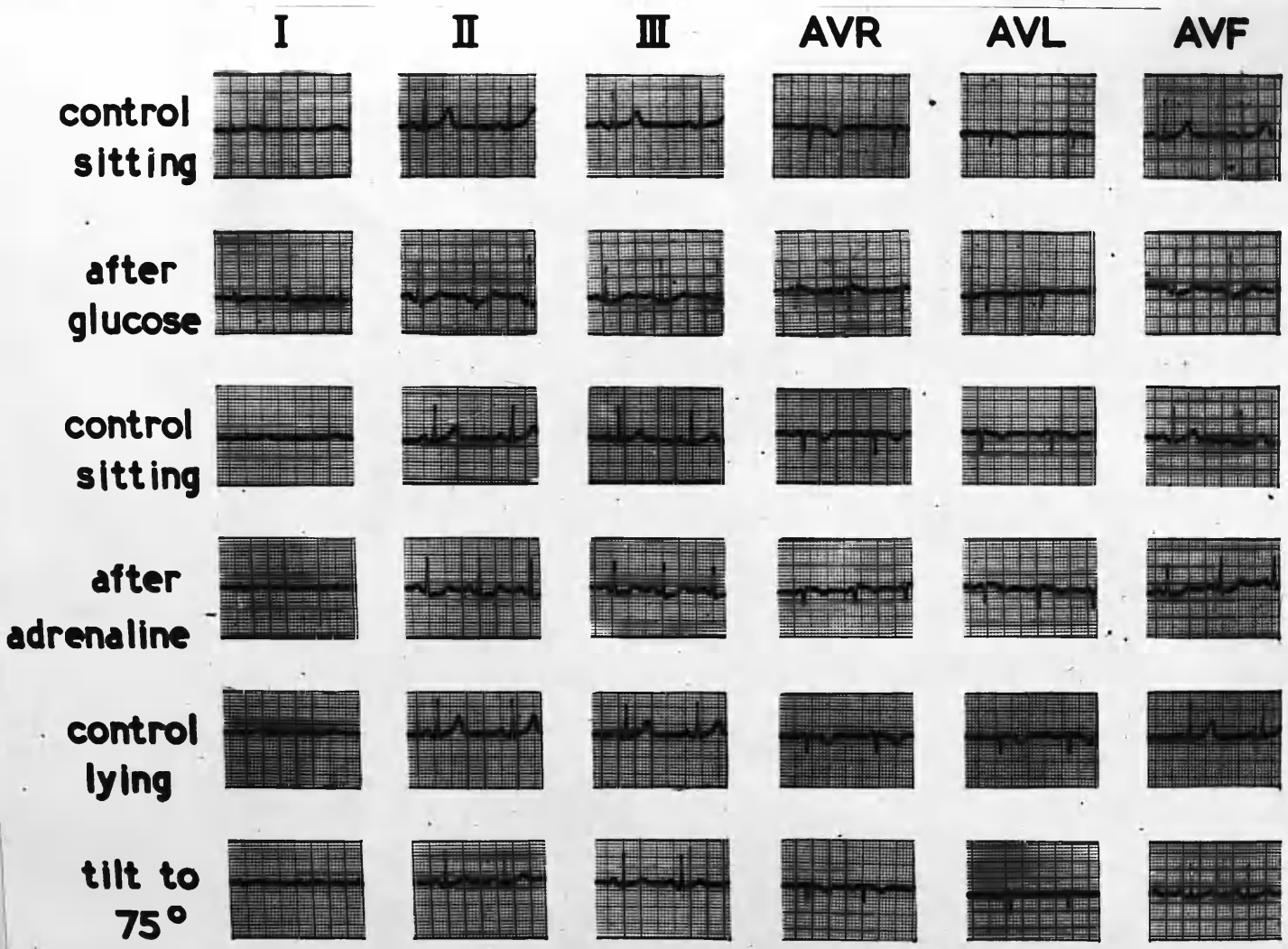
ADRENALINE Control and 20 mins.

Heart rate increased from 60 to 67: P:- No change from the control: QRS:- No change from the control: T:- flatter I, II and aVF, more biphasic III, less negative aVR: U:- No change from the control.

POSTURE Control and 5 mins.

Heart rate increased from 60 to 68: P:- peaked II and III: QRS:- No change from the control: RT:- No change from the control: T:- flatter II, biphasic III, less negative aVR, flatter aVF: U:- II.

ADDITIONAL EXPERIMENT NO. 9



ADDITIONAL EXPERIMENT - EXAMPLE NO. 9.

PATIENT 37.

GLUCOSE Control and 28 mins.

Heart rate increased from 62 to 88: P:- No change from the control: QRS:- No change from the control: RT:- ST low take off II, III and aVF: T:- flatter I, II, III and aVF and less negative aVR and aVL: U:- II, III and aVF.

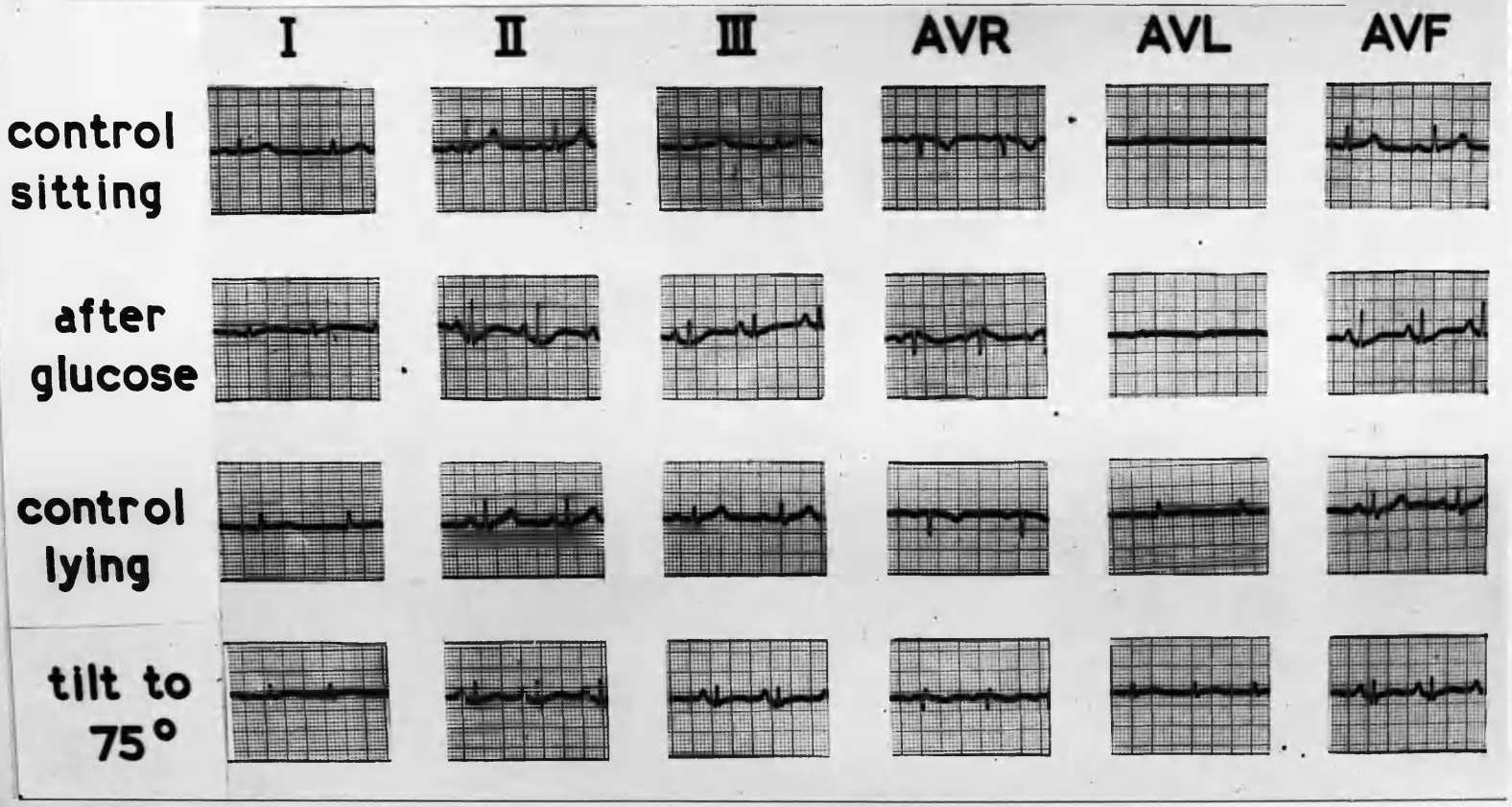
ADRENALINE Control and 15 mins.

Heart rate increased from 68 to 100: P:- No change from the control: QRS:- No change from the control: RT:- ST below isoelectric line II, III and aVF: T:- flatter I, II, III and aVF and less negative aVR and aVL: U:- III.

POSTURE Control and 5 mins.

Heart rate increased from 66 to 88: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- flatter II, III and aVF and less negative aVR and aVL: U:- II, III and aVF.

ADDITIONAL EXPERIMENT NO. 10



ADDITIONAL EXPERIMENT - EXAMPLE NO. 10.

PATIENT 2.

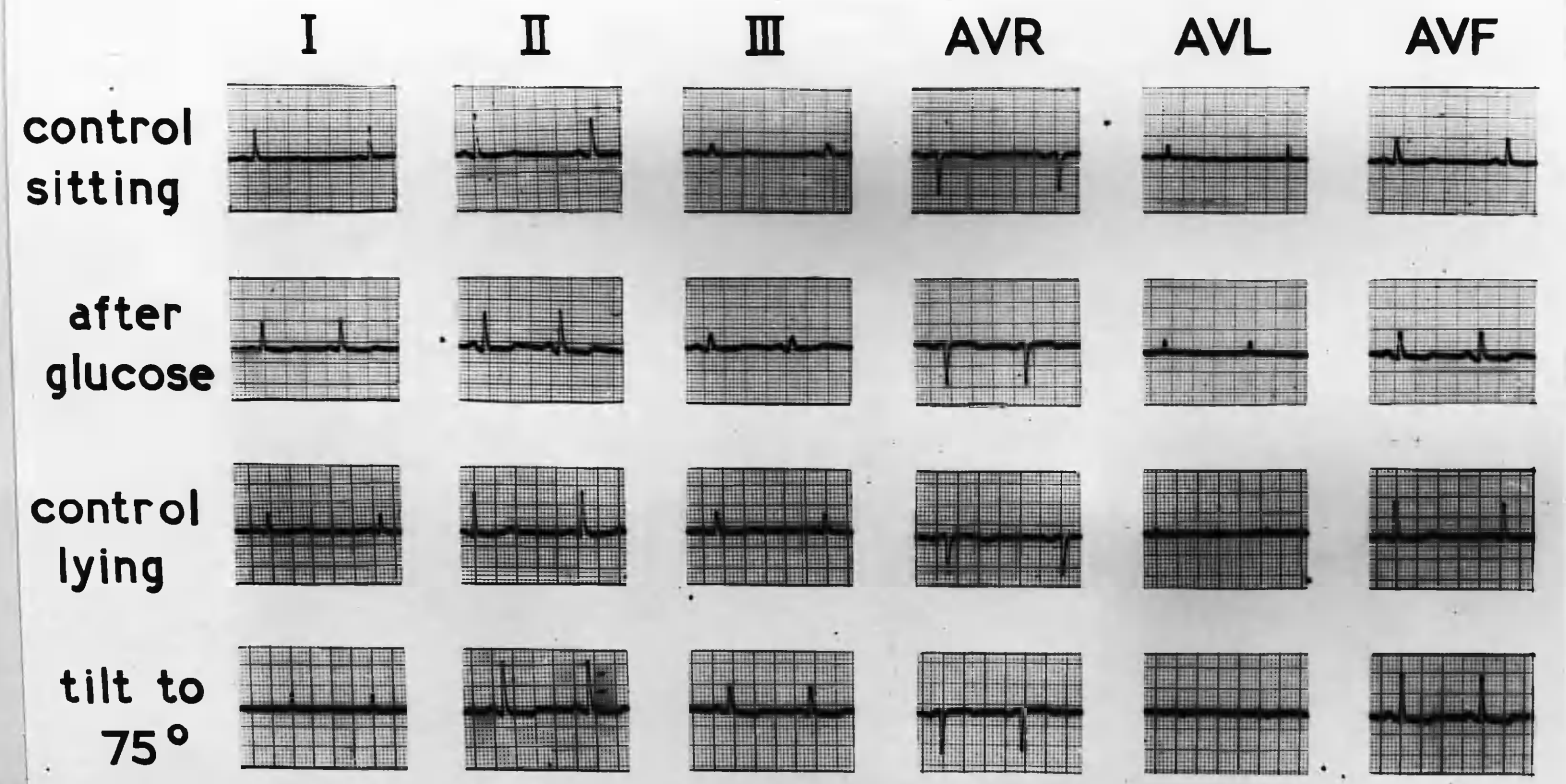
GLUCOSE Control and 36 mins.

Heart rate increased from 72 to 84: P:- peaked II, III and aVF: QRS:- RII and aVF increased: RT:- No change from the control: T:- flatter I, II, III aVF and less negative aVR: U:- II.

POSTURE Control and 5 mins.

Heart rate increased from 70 to 94: P:- peaked II, III and aVF: QRS:- RII and aVF decreased, S aVL: RT:- No change from the control: T:- flatter II, III and aVF and less negative aVR: U:- No change from the control.

ADDITIONAL EXPERIMENT NO. II



ADDITIONAL EXPERIMENT - EXAMPLE NO. 11.

PATIENT 13.

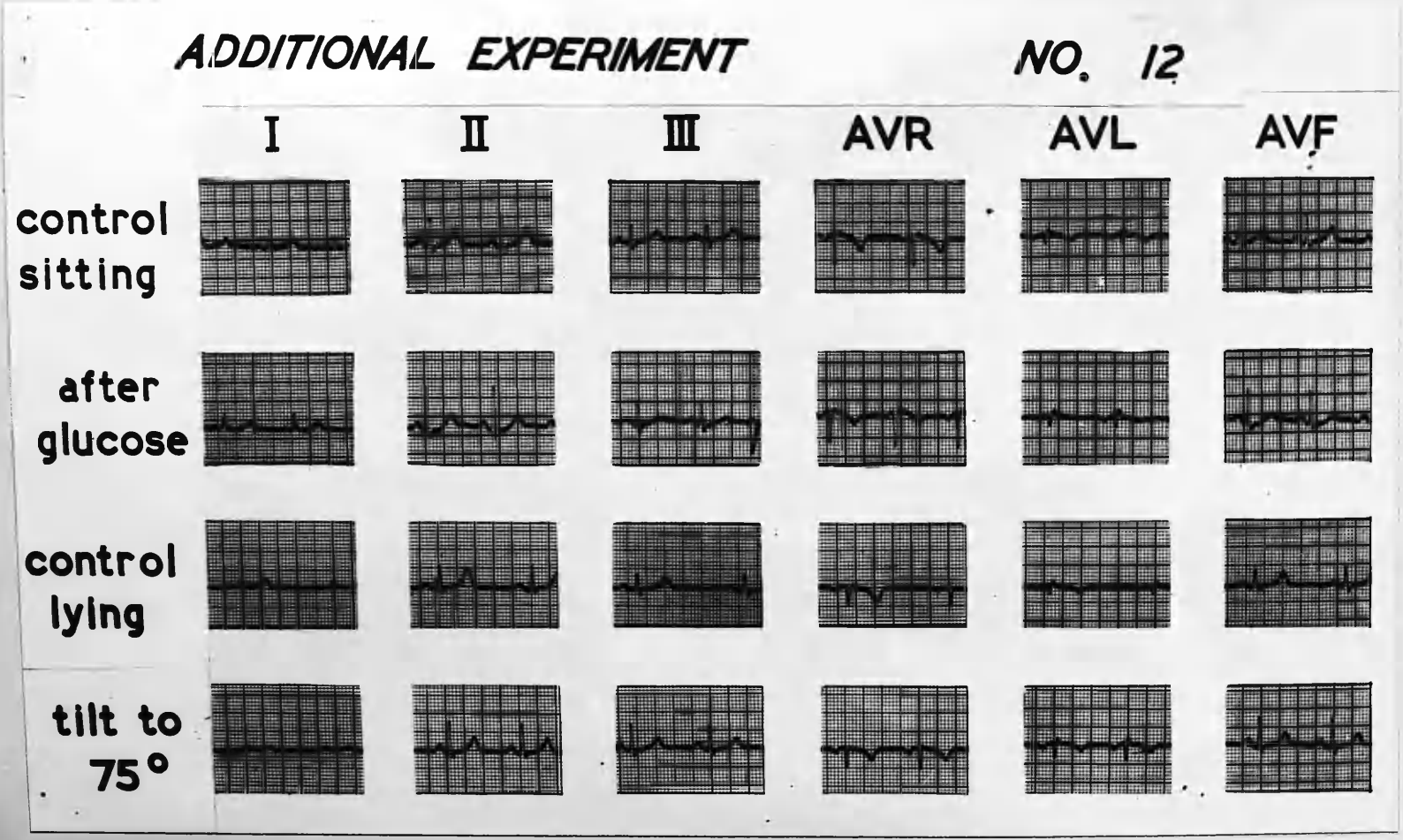
GLUCOSE Control and 20 mins.

Heart rate increased from 54 to 78: P:- peaking II and aVF: QRS:- No change from the control: RT:- No change from the control: T:- II, III, aVR and aVF: U:- No change from the control.

POSTURE Control and 5 mins.

Heart rate increased from 56 to 77: P:- peaking II and aVF: QRS:- S aVL: RT:- No change from the control: T:- biphasic II, III, aVR and aVF: U:- No change from the control.

ADDITIONAL EXPERIMENT NO. 12



ADDITIONAL EXPERIMENT - EXAMPLE NO. 12.

PATIENT 39.

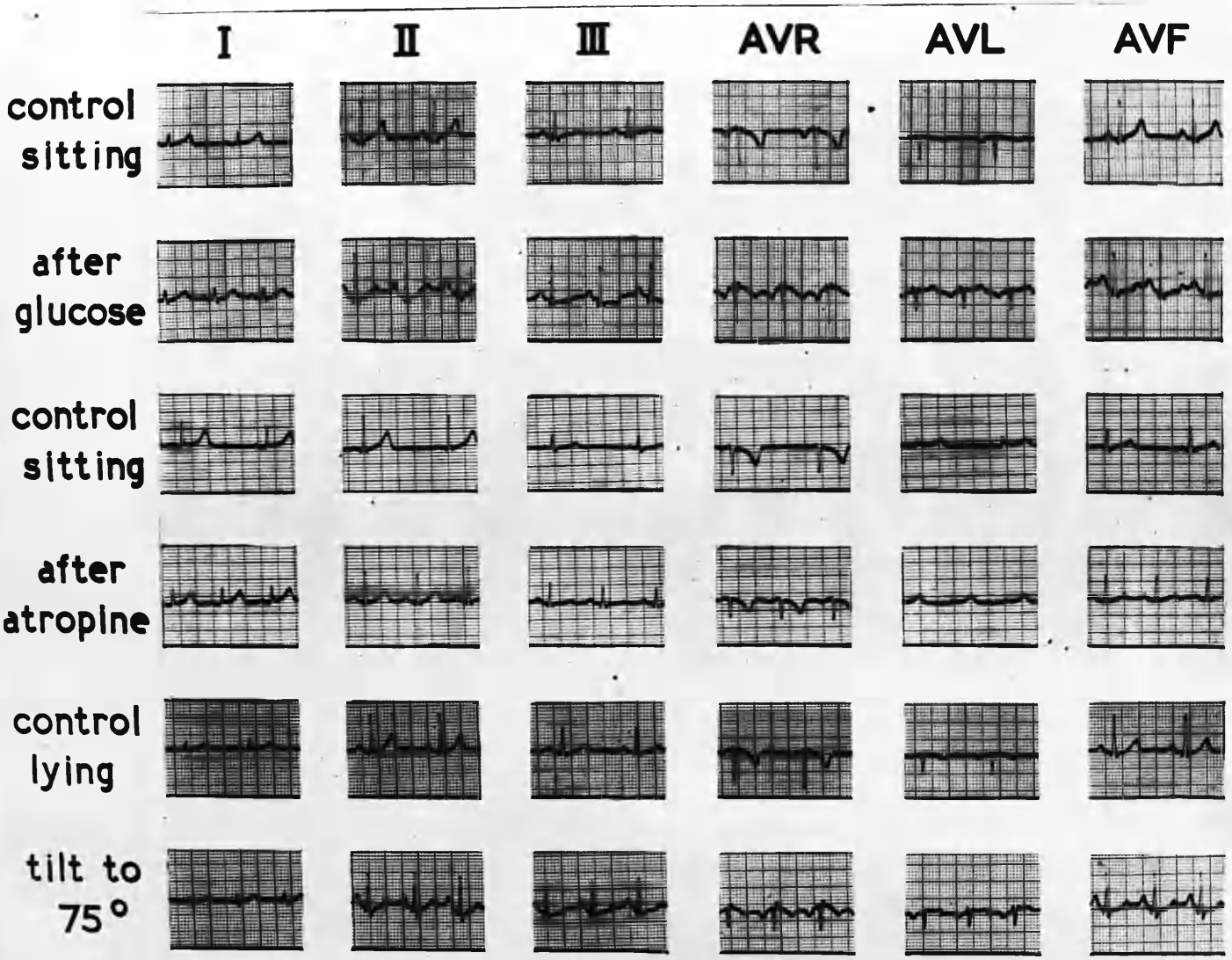
GLUCOSE Control and 30 mins.

Heart rate increased from 72 to 94: P:- No change from the control: QRS:- increased R II and aVF: R^T:- ST low take off II and aVF: T:- flatter II, III and aVF and less negative aVR and aVL: U:- aVF.

POSTURE Control and 5 mins.

Heart rate increased from 56 to 75: P:- peaked aVF: QRS:- increased R II and aVF, S aVL increased: RT:- No change from the control: T:- flatter I, II, III and aVF and less negative aVR: U:- aVF.

ADDITIONAL EXPERIMENT NO. 13



ADDITIONAL EXPERIMENT - EXAMPLE NO. 13.

PATIENT 3.

GLUCOSE Control and 25 mins.

Heart rate increased from 70 to 100: P:- peaked II, III and aVF: QRS:- R II and III increased: RT:- ST III below isoelectric line: T:- flatter I, II, inverted III, biphasic aVF, less negative aVR, positive aVL: U:- No change from the control.

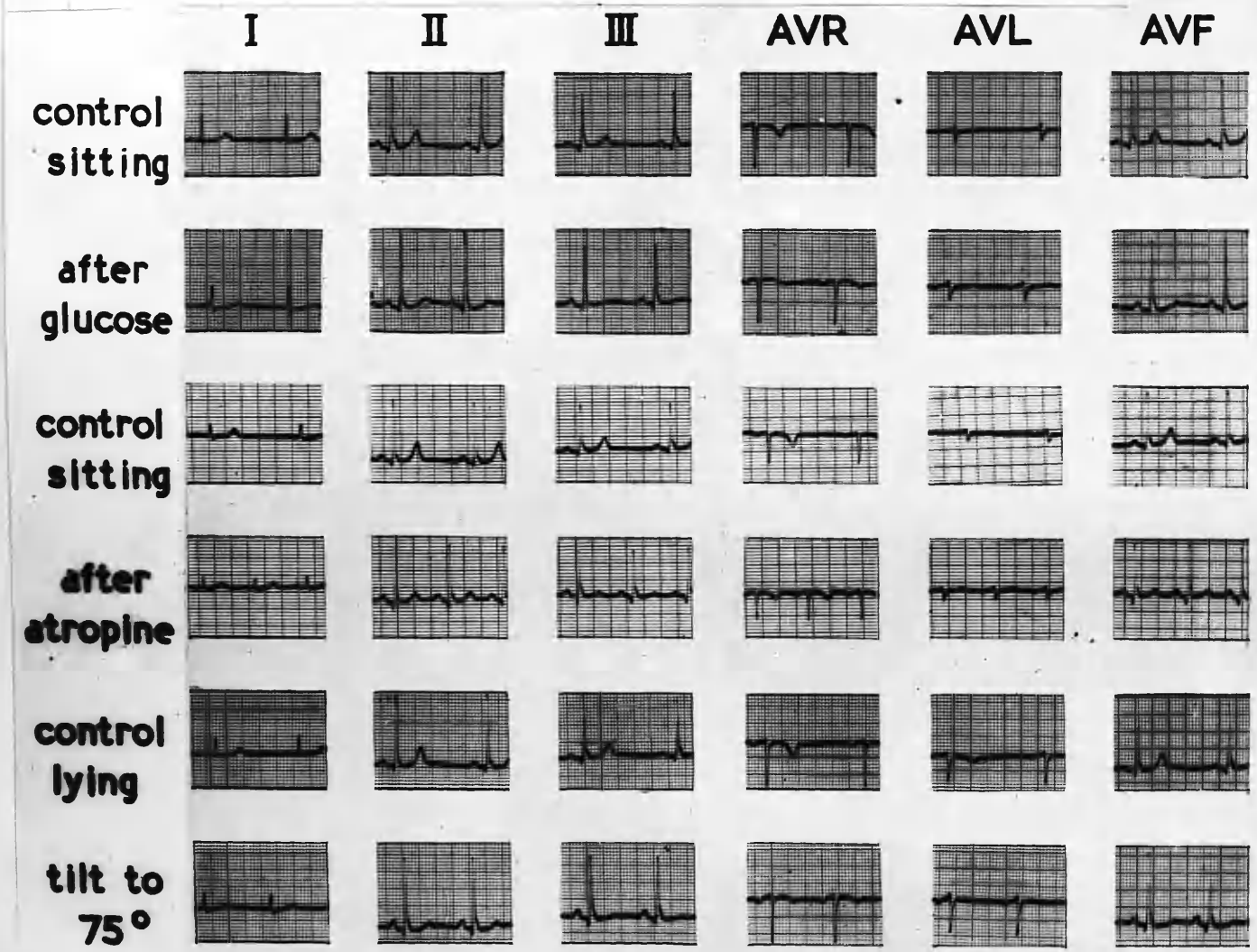
ATROPINE Control and 15 mins.

Heart rate increased from 60 to 100: P:- peaked II, III and aVF: QRS:- No change from the control: RT:- No change from the control: T:- slightly flatter I, II, III and aVF: U:- No change from the control.

POSTURE Control and 5 mins.

Heart rate increased from 70 to 107: P:- peaking II, III and aVF: QRS:- S aVR decreased: RT:- ST low take off II and aVF, below isoelectric line III: T:- flatter I, II, biphasic III, less negative aVR; biphasic aVL, flatter aVF: U:- No change from the control.

ADDITIONAL EXPERIMENT NO. 14



ADDITIONAL EXPERIMENT - EXAMPLE NO. 14.

PATIENT 29.

GLUCOSE Control and 38 mins.

Heart rate increased from 58 to 74: P:- peaked aVF:- QRS:- increased R II and III: RT:- No change from the control: T:- flatter I, II, III and aVF and less negative aVR: U:- II and aVF.

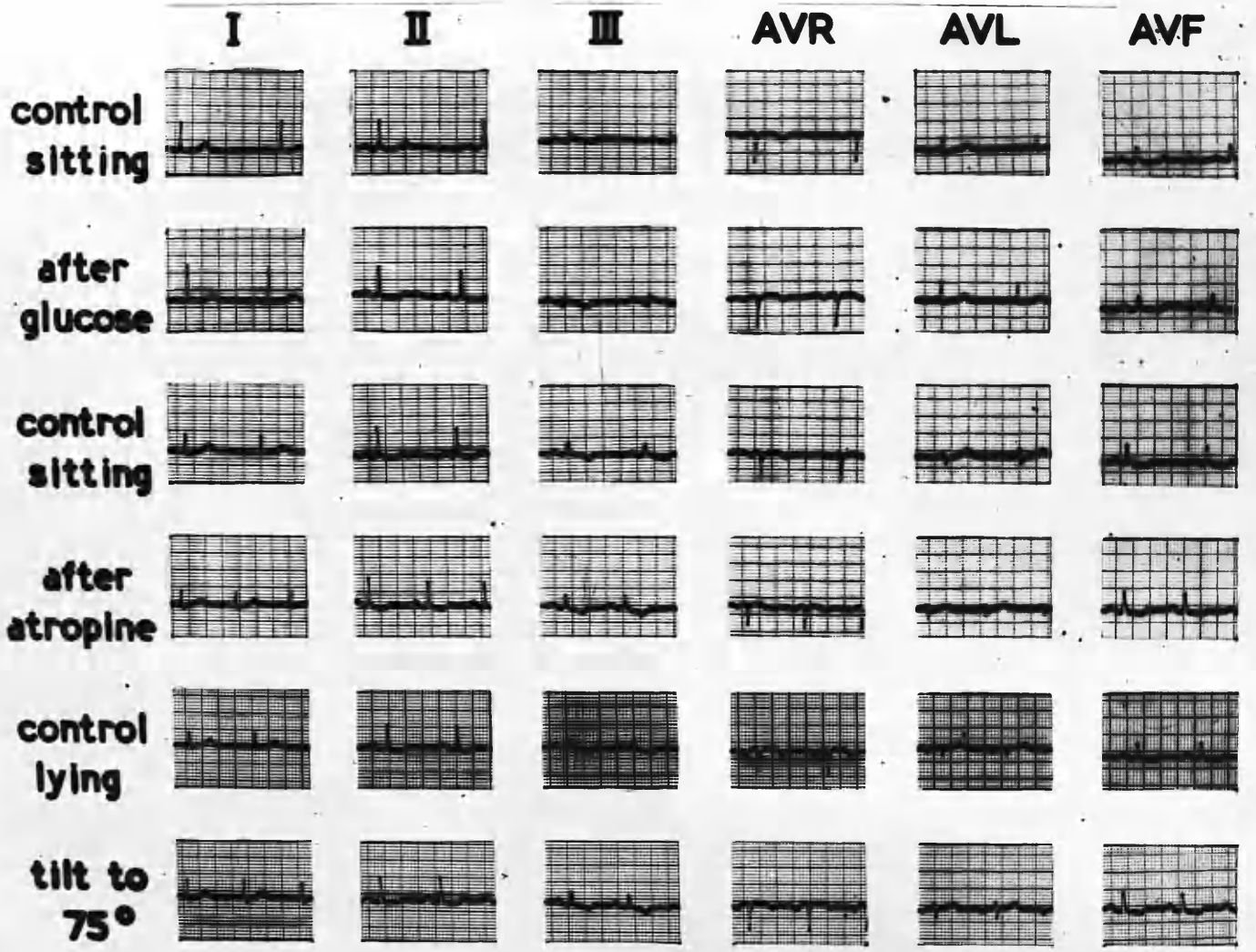
ATROPINE Control and 15 mins.

Heart rate increased from 58 to 94: P:- peaked aVF: QRS:- No change from the control: RT:- No change from the control: T:- Flatter I, II, III and aVF and less negative aVR: U:- No change from the control.

POSTURE Control and 5 mins.

Heart rate increased from 54 to 72: P:- peaked II, III and aVF: QRS:- No change from the control: RT:- No change from the control: T:- flatter I, II, III and aVF and less negative aVR and aVL: U:- II.

ADDITIONAL EXPERIMENT NO. 15



ADDITIONAL EXPERIMENT - EXAMPLE NO. 15.

PATIENT 41.

GLUCOSE Control and 25 mins.

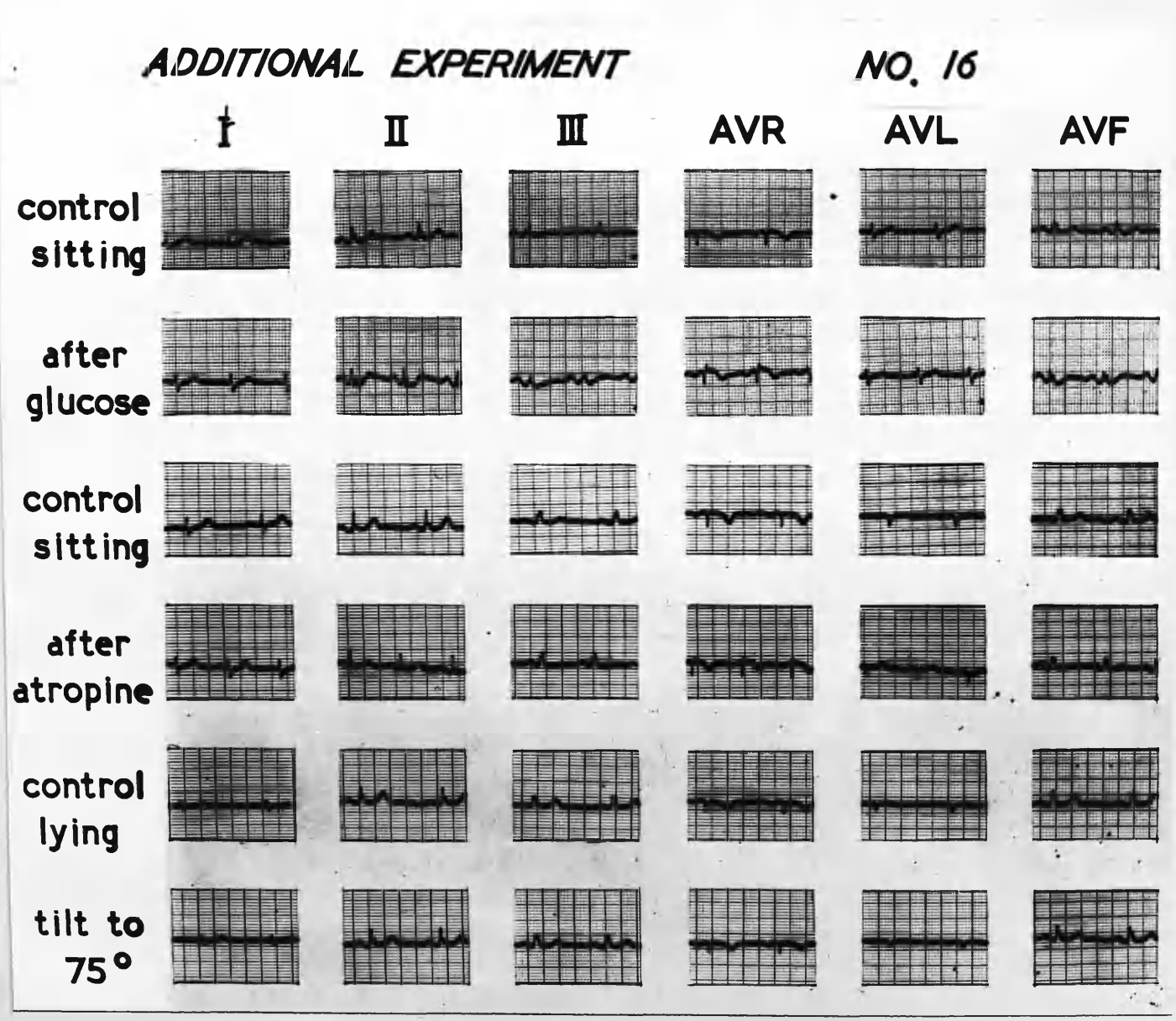
Heart rate increased from 52 to 62: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- flatter II, more negative III, less negative aVR: biphasic aVF: U:- II and aVF.

ATROPINE Control and 15 mins.

Heart rate increased from 66 to 90: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- biphasic II, more negative III: U:- No change from the control.

POSITIVE Control and 5 mins.

Heart rate increased from 75 to 86: P:- No change from the control: QRS:- S aVL increased: R^T:- No change from the control: T:- flatter II, more negative III, less negative aVR and more negative part of biphasic T aVF: U:- No change from the control.


ADDITIONAL EXPERIMENT NO. 16
I
II
III
AVR
AVL
AVF
control sitting
after glucose
control sitting
after atropine
control lying
tilt to 75°

ADDITIONAL EXPERIMENT - EXAMPLE NO. 16.

PATIENT 57.

GLUCOSE Control and 18 mins.

Heart rate increased from 68 to 94: P:- No change from the control: QRS:- No change from the control: RT:- ST sagging below isoelectric line II, III and aVF and take off ST aVR elevated: T:- broader and runs into U wave: U:- II, III and aVF.

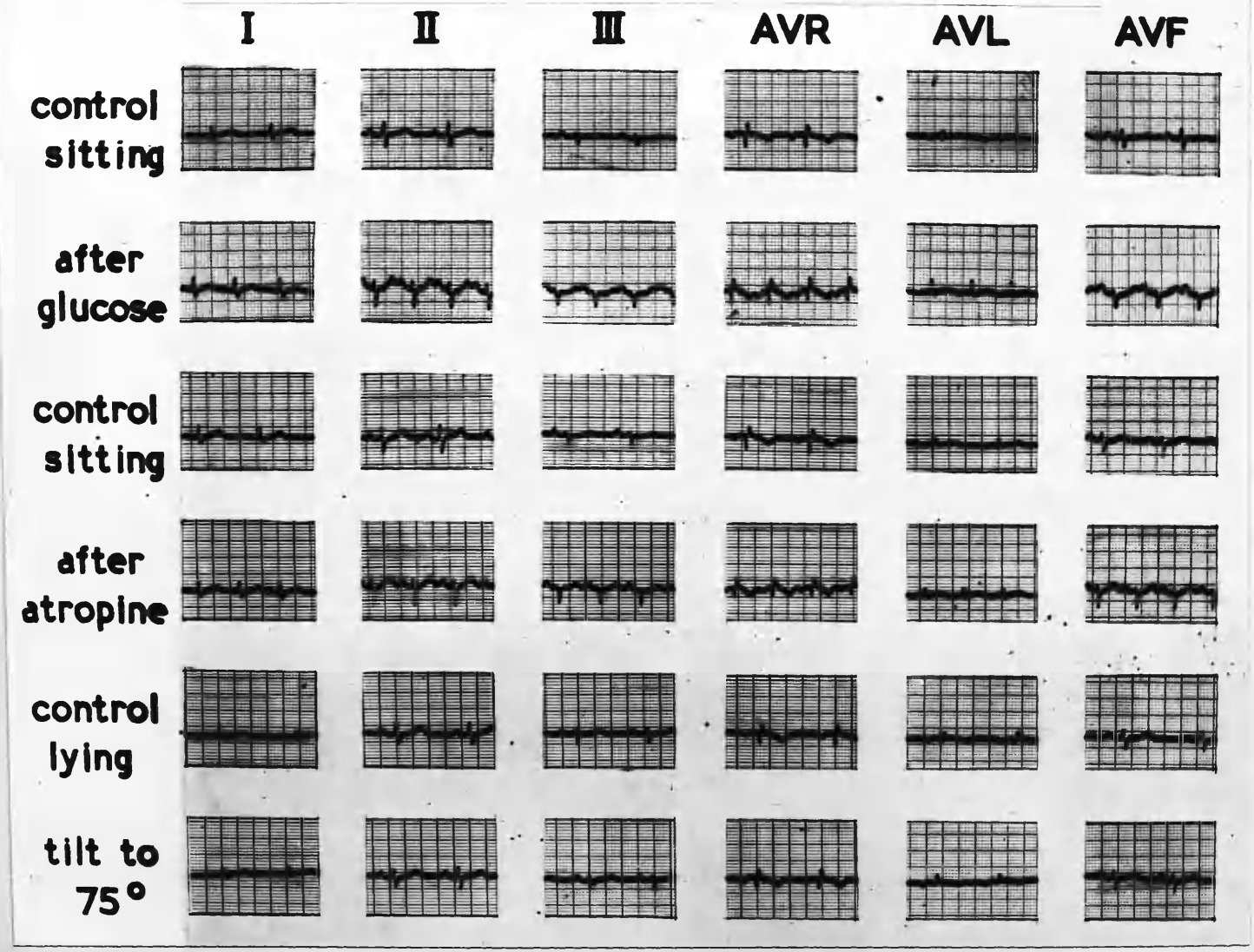
ATROPINE Control and 15 mins.

Heart rate increased from 64 to 92: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- flatter II, III and aVF and less negative aVR: U:- No change from the control.

POSTURE Control and 5 mins.

Heart rate increased from 60 to 67: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- flatter II, III and aVF: U:- aVF.

ADDITIONAL EXPERIMENT NO. 17



ADDITIONAL EXPERIMENT - EXAMPLE NO. 17.

PATIENT 15.

GLUCOSE Control and 22 mins.

Heart rate increased from 66 to 124: P:- peaked II, III and aVF: QRS:- S II, III and R aVL increased: RT:- ST low take off II and aVF: T:- slightly increased in II and III: U:- No change from the control.

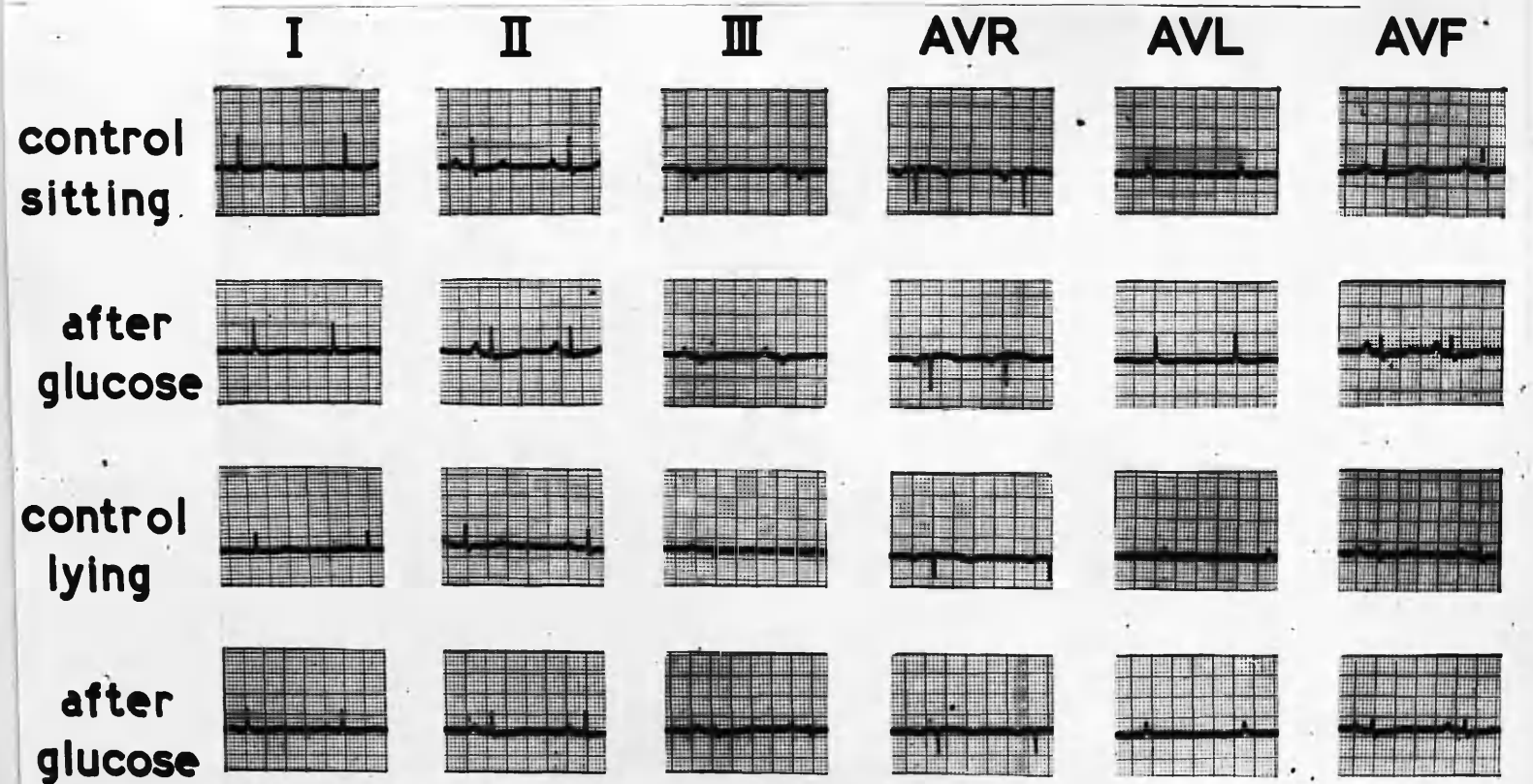
ATROPINE Control and 20 mins.

Heart rate increased from 78 to 118: P:- peaked II, III and aVF: QRS:- increased S II: RT:- No change from the control: T:- No change from the control: U: No change from the control.

POSTURE Control and 5 mins.

Heart rate increased from 68 to 79: P:- peaked aVF: QRS:- No change from the control: RT:- No change from the control: T:- slightly flatter II: U:- No change from the control.

ADDITIONAL EXPERIMENT NO. 18



ADDITIONAL EXPERIMENT - EXAMPLE NO. 18.

PATIENT 38.

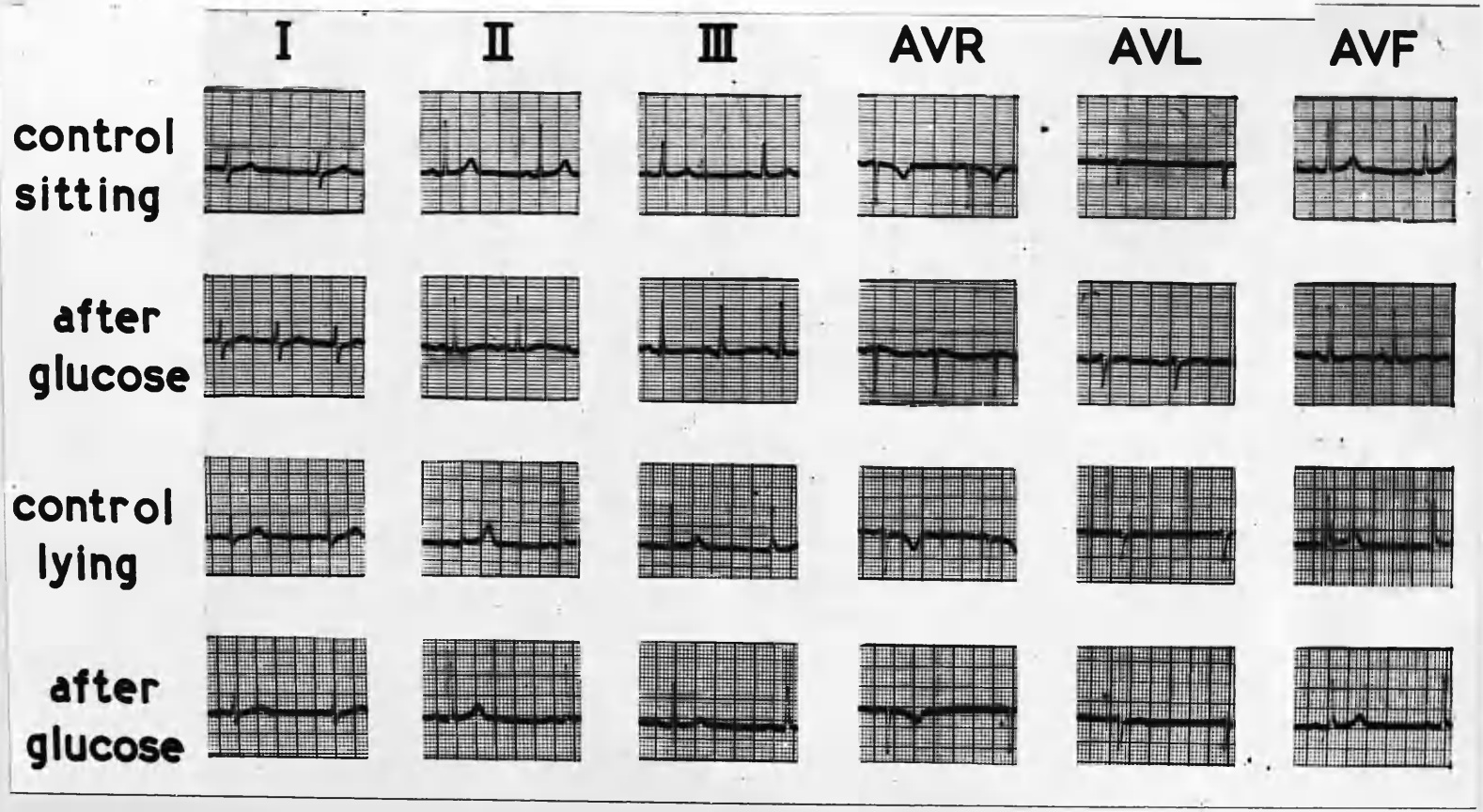
GLUCOSE SITTING Control and 23 mins.

Heart rate increased from 64 to 88: P:- peaked II, III and aVF: QRS:- No change from the control: RT:- No change from the control: T:- flatter I, II, III and aVF and less negative aVR: U:- No change from the control.

GLUCOSE LYING Control and 23 mins.

Heart rate increased from 54 to 63: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- slightly flatter II and aVF: U:- No change from the control.

ADDITIONAL EXPERIMENT NO. 19



ADDITIONAL EXPERIMENT - EXAMPLE NO. 19.

PATIENT 58.

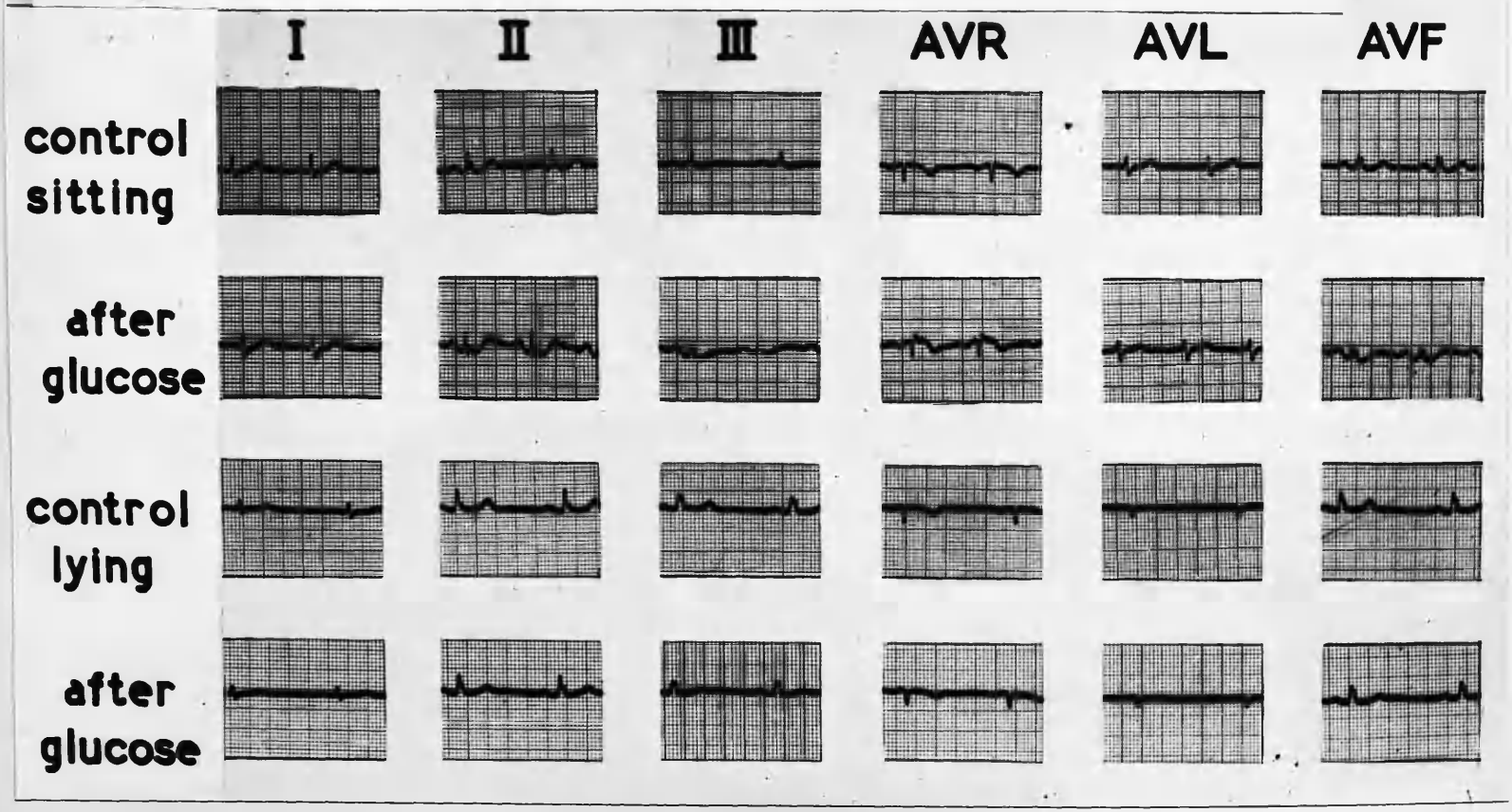
GLUCOSE SITTING Control and 25 mins.

Heart rate increased from 60 to 100: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- flatter and broader II, III, aVF, less negative aVR and positive aVL: U:- II and aVF.

GLUCOSE LYING Control and 25 mins.

Heart rate unchanged: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- slightly flatter II, III, aVF and slightly less negative aVR: U:- II.

ADDITIONAL EXPERIMENT NO. 20



ADDITIONAL EXPERIMENT - EXAMPLE NO. 20.

PATIENT 57.

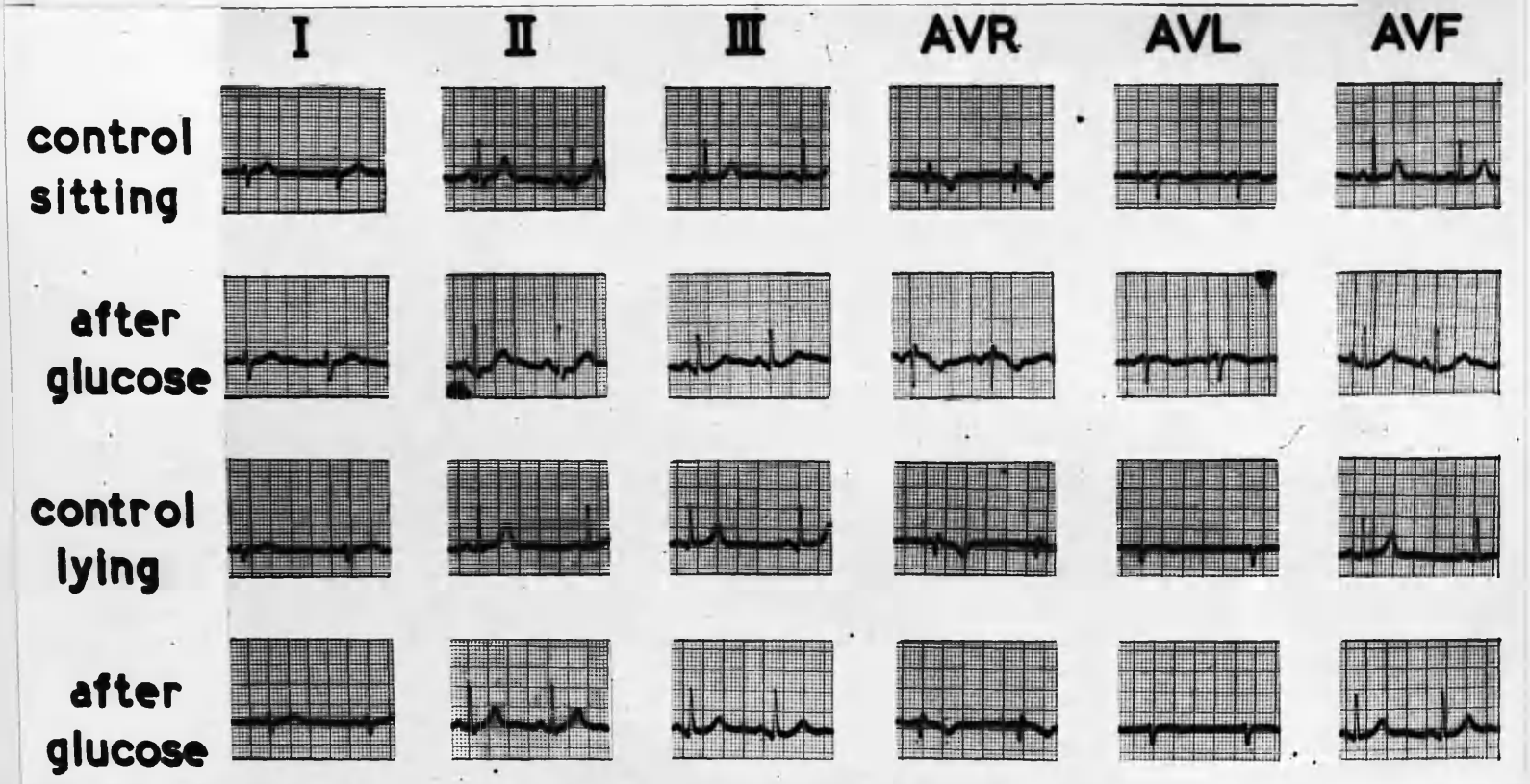
GLUCOSE SITTING Control and 18 mins.

Heart rate increased from 68 to 94: P:- No change from the control: QRS:- No change from the control: R_t:- ST sagging below isoelectric line in II, III and aVF: T:- broader and runs into U wave: U:- II, III and aVF.

GLUCOSE LYING Control and 18 mins.

Heart rate increased from 55 to 60: P:- No change from the control: QRS:- No change from the control: RT:- No change from the control: T:- slightly flatter II, III and aVF: U:- slightly increased II, III and aVF.

ADDITIONAL EXPERIMENT NO. 21



ADDITIONAL EXPERIMENT - EXAMPLE NO. 21.

PATIENT 25.

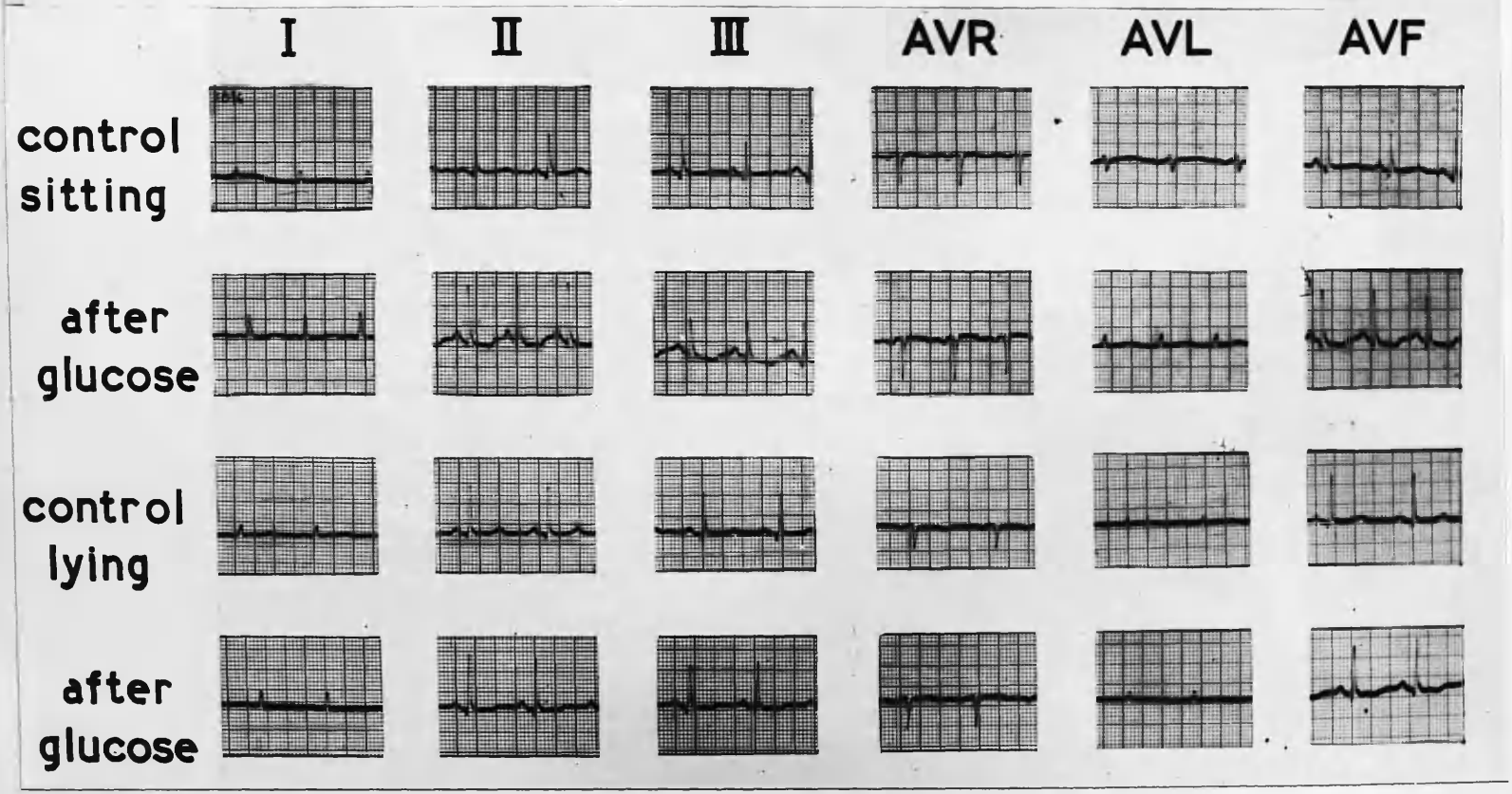
GLUCOSE SITTING Control and 25 mins.

Heart rate increased from 54 to 84: P:- peaked II and aVF: QRS:- No change from the control: RT:- No change from the control: T:- flatter in II, III and aVF, and less negative in aVR and aVL: U:- increased in II, III, aVR and aVF.

GLUCOSE LYING Control and 25 mins.

Heart rate increased from 56 to 72: P:- peaked II: QRS:- No change from the control: RT:- No change from the control: T:- slightly flatter II, III and aVF and slightly less negative aVL: U:- increased II, III and aVF.

ADDITIONAL EXPERIMENT NO. 22



ADDITIONAL EXPERIMENT - EXAMPLE NO. 22.

PATIENT 34.

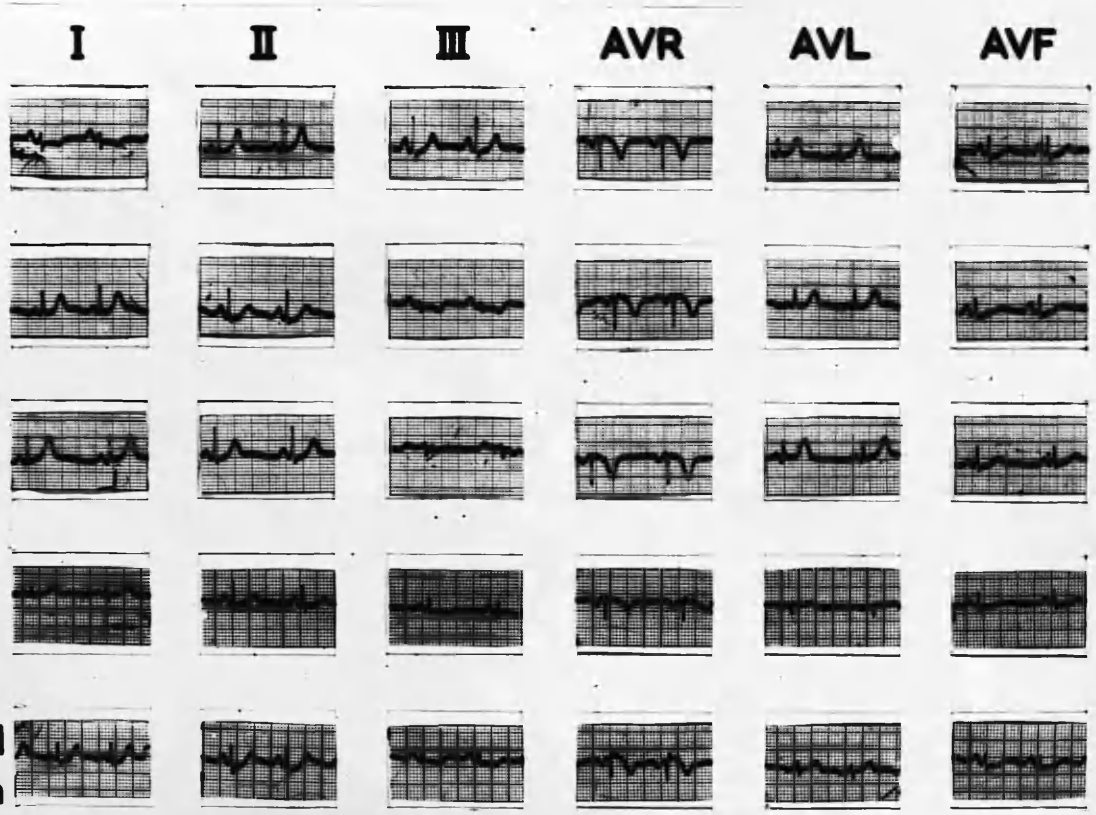
GLUCOSE SITTING Control and 35 mins.

Heart rate increased from 86 to 116: P:- peaking II, III and aVF: QRS:- R II, III and aVF increased: RT:- ST sagging below isoelectric line II, III and aVF: T:- biphasic II, III and aVF: U:- No change from the control.

GLUCOSE LYING Control and 35 mins.

Heart rate increased from 75 to 88: P:- peaked aVF: QRS:- No change from the control: RT:- No change from the control: T:- biphasic II, III and aVF: U:- No change from the control.

EFFECT OF ERGOTAMINE I.V. No: 1



EFFECT OF ERGOTAMINE - EXAMPLE NO. 1.

PATIENT 22.

1. GLUCOSE AND ERGOTAMINE.

Control (Tracings for Leads I and III inadvertently in wrong places)

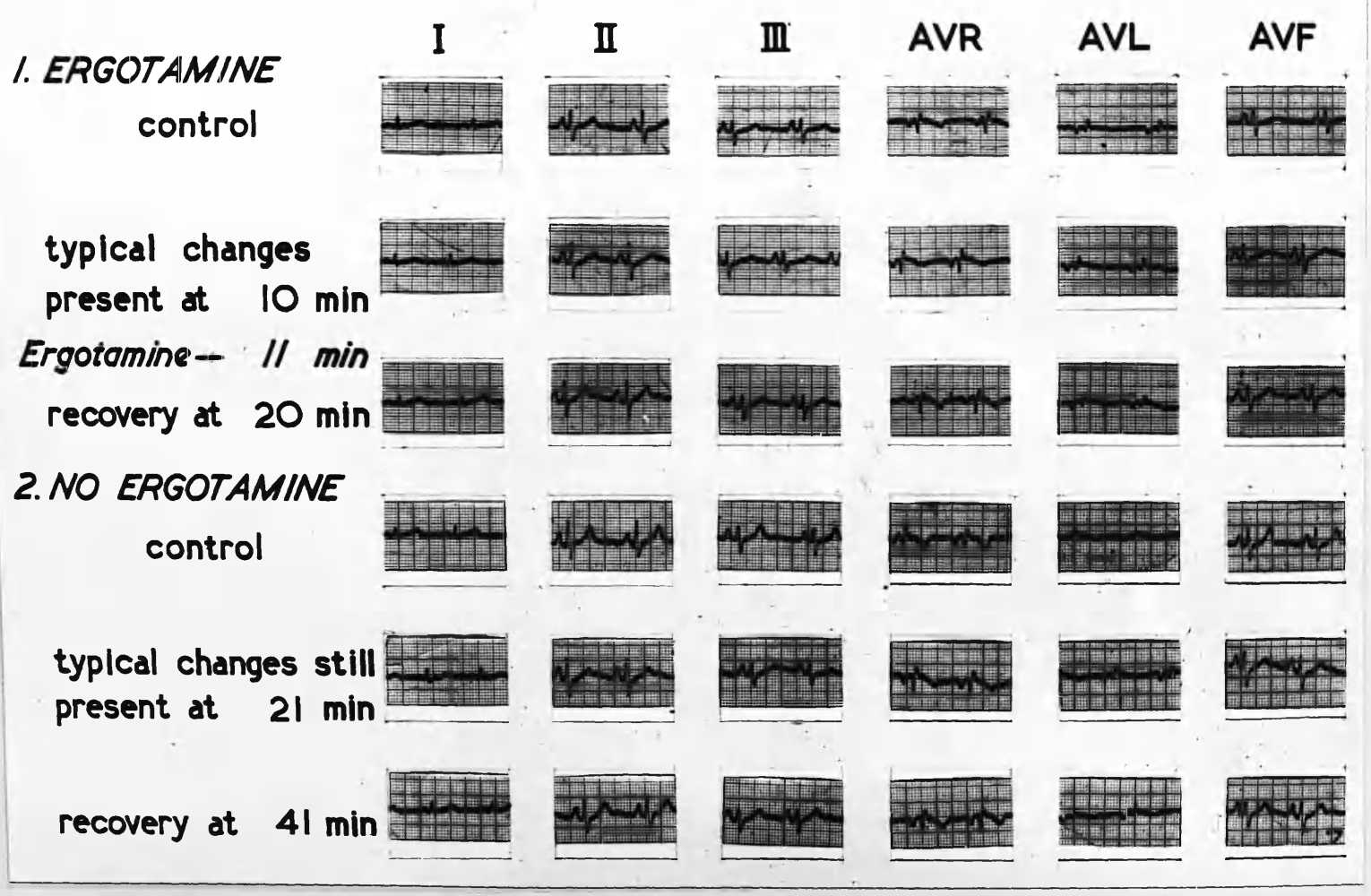
16 mins. Heart rate increased from 80 to 90: P:- peaked II: QRS: No change: RT:- ST sagging below isoelectric line in II and aVF: T:- flatter II, and aVF: inverted III: U:- increased II and aVR.

25 mins. Heart rate 68: P:- back to control: QRS:- No change: RT:- ST sagging below isoelectric line in II and aVF: T:- flatter II, and aVF, inverted III: U:- increased II and aVR.

2. GLUCOSE AND NO ERGOTAMINE.

37 mins. Heart rate increased from 72 to 88: P:- peaked II, III and aVF: QRS:- No change: RT:- ST sagging below isoelectric line I, II, III, and aVF: T:- larger I, inverted III: U:- increased II.

EFFECT OF ERGOTAMINE I.V. *No: 2*



EFFECT OF ERGOTAMINE - EXAMPLE NO. 2.

PATIENT 40.

1. GLUCOSE AND ERGOTAMINE

10 mins. Heart rate increased from 68 to 84: P:- peaked II: QRS:- increased in II: RT:- low take off ST II and aVF: T:- flatter I, II, III and aVF: less negative aVR: U:- increased in II, III and aVF.

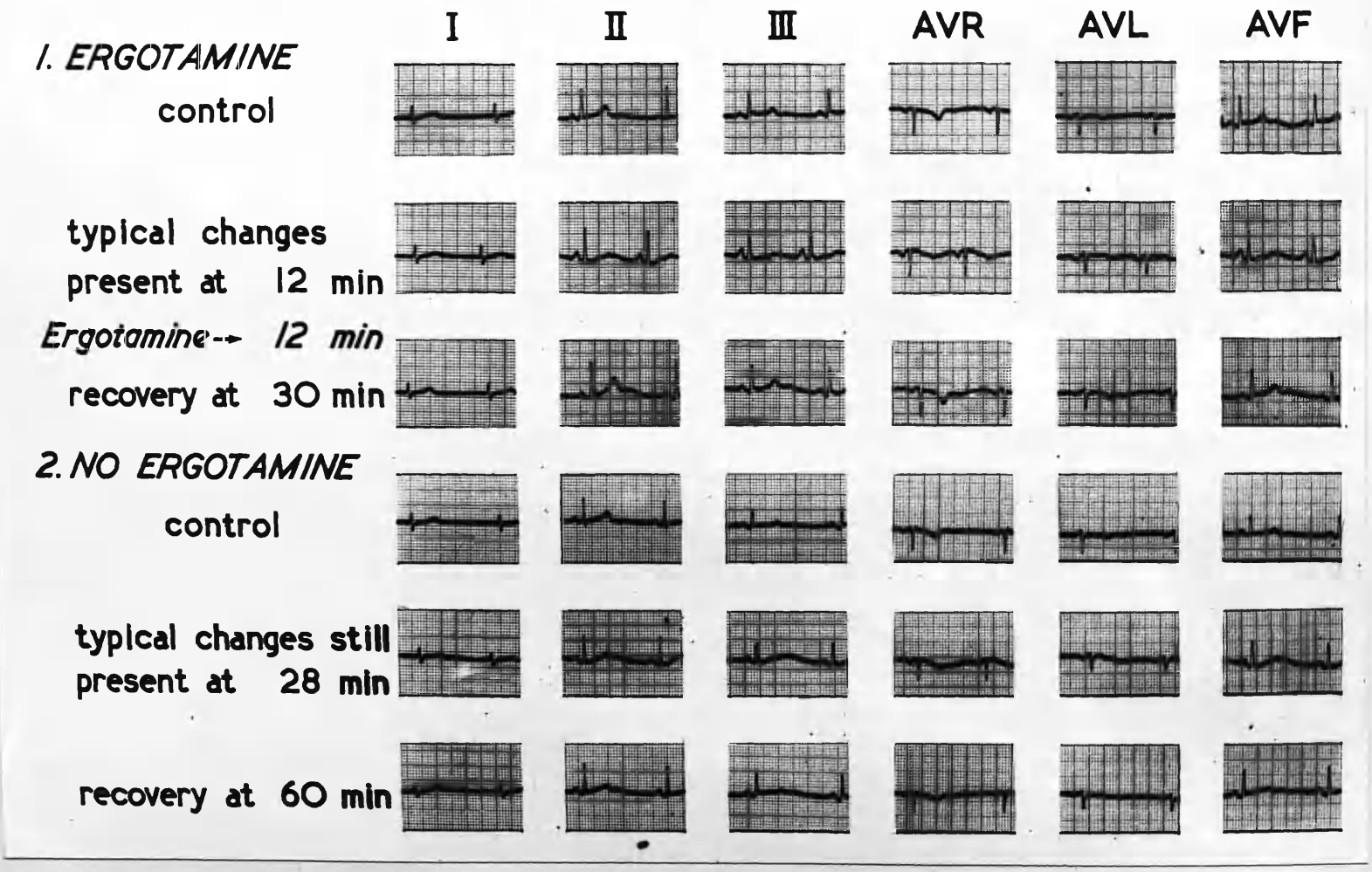
20 mins. Heart rate 75: P:- II back to control: QRS:- II still increased: T:- waves back to control height: U:- II, III, aVF still present.

2. GLUCOSE AND NO ERGOTAMINE.

21 mins. Heart rate increased from 65 to 82: P:- peaked II, III and aVF: QRS:- no change: RT:- ST low take off II and aVF: T:- flatter I, II, III and aVF and less negative aVR: U:- increased II, III and aVF.

41 mins. Heart rate 79: P:- returned to control: QRS:- No change: RT:- ST take off isoelectric: T:- waves control height: U:- still present II, III and aVF.

EFFECT OF ERGOTAMINE I.V. No: 3



EFFECT OF ERGOTAMINE - EXAMPLE NO. 3.

PATIENT 26.

1. GLUCOSE AND ERGOTAMINE.

12 mins. Heart rate increased from 56 to 75: P:- peaked II, III and aVF: QRS:- No change: RT:- No change: T:- flatter in I, II, biphasic III, aVL and aVF: less negative aVR: U:- increased II.

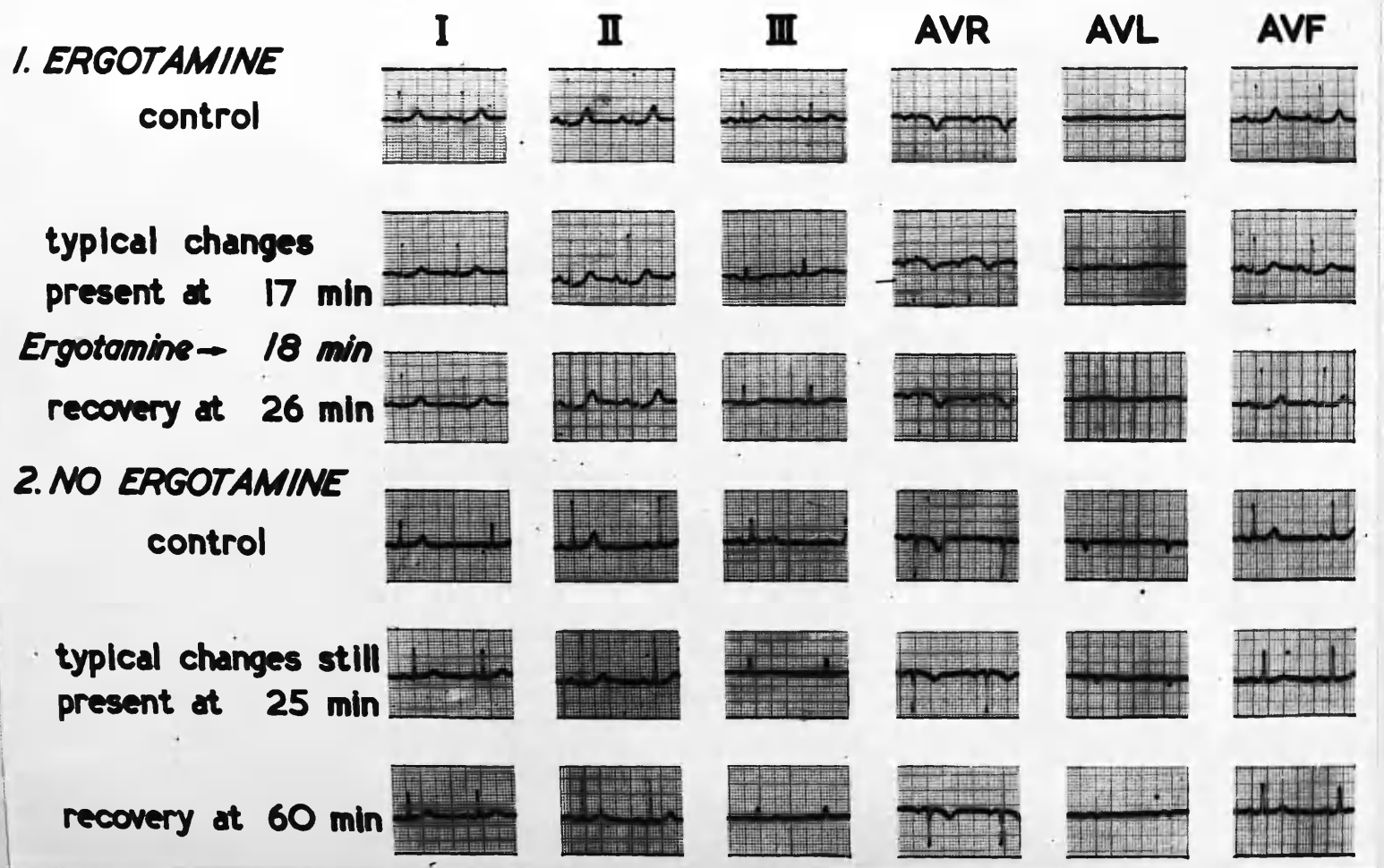
30 mins. Heart rate 56: P:- back to control: QRS:- No change: RT:- No change: T:- return to control voltage: U:- increased II, III and aVF.

2. GLUCOSE AND NO ERGOTAMINE.

28 mins. Heart rate increased from 50 to 60: P:- No change: QRS:- No change: RT:- No change: T:- flatter I, II, III and broader aVF, less negative aVR: U:- increased II, III and aVR.

60 mins. Heart rate 54: P:- No change: QRS:- No change: RT:- No change: T:- returning but still flatter than control: U:- still increased II, III and aVF.

EFFECT OF ERGOTAMINE I.V. *No: 4*



EFFECT OF ERGOTAMINE - EXAMPLE NO. 4.

PATIENT 50.

1. GLUCOSE AND ERGOTAMINE.

17 mins. Heart rate increased from 70 to 79: P:- No change: QRS:- No change: RT:- ST sagging below isoelectric line in II and aVF: T:- flatter I, II, III, and aVF: less negative aVR: U:- increased II and aVF.

26 mins. Heart rate 70: P:- No change: QRS:- No change: RT:- return to control level: T:- control voltage: U:- still present II.

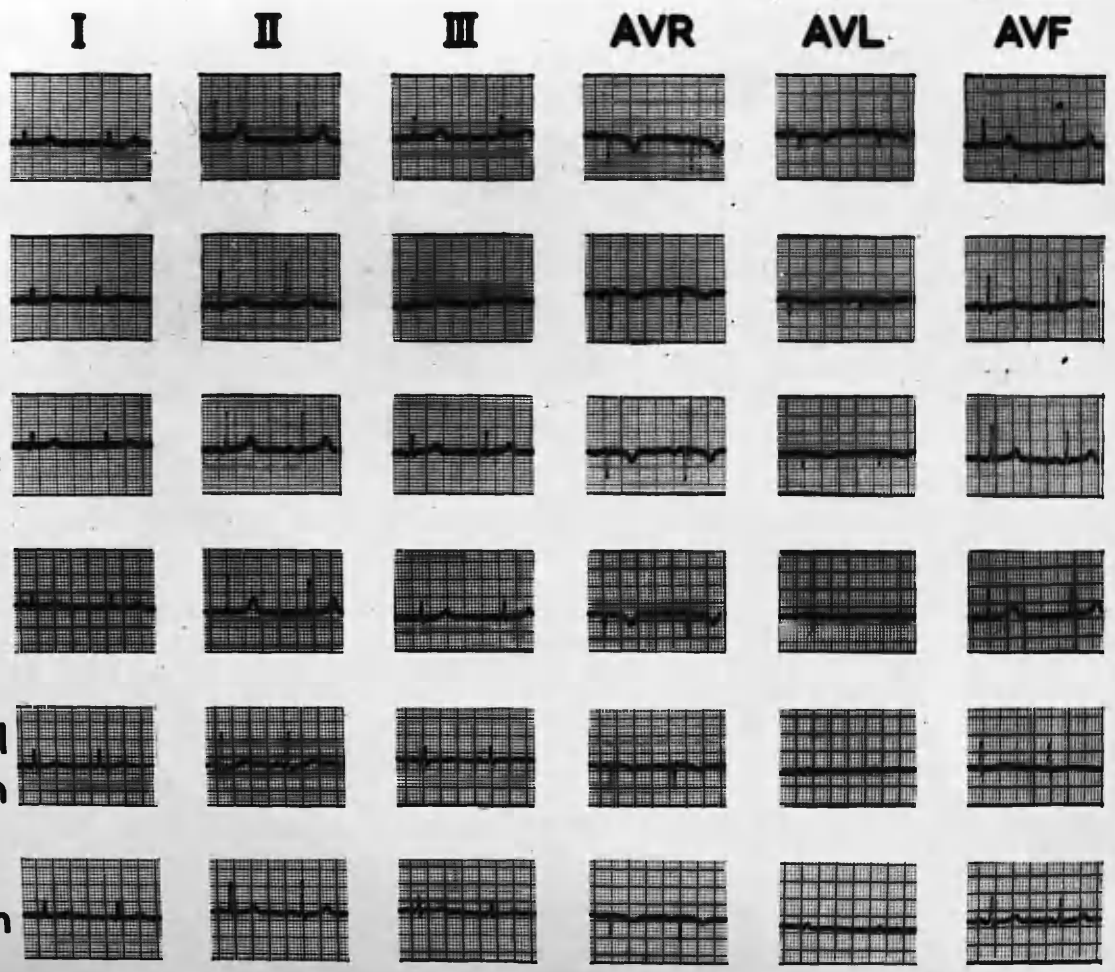
2. GLUCOSE AND NO ERGOTAMINE.

25 mins. Heart rate increased from 56 to 65: P:- No change: QRS:- No change: RT:- No change: T:- flatter II, III and aVF and less negative aVR: U:- increased II.

60 mins. Heart rate 67: P:- No change: QRS:- No change: RT:- No change: T:- returning to control voltage: U:- back to control.

EFFECT OF ERGOTAMINE I.V.

No: 5



EFFECT OF ERGOTAMINE - EXAMPLE NO. 5.

PATIENT 43.

1. GLUCOSE AND ERGOTAMINE.

20 mins. Heart rate increased from 65 to 76: P:- No change: QRS:- No change: RT:- No change: T:- flatter I, II, III and aVF and less negative aVR and aVL: U:- increased II, III and aVF.

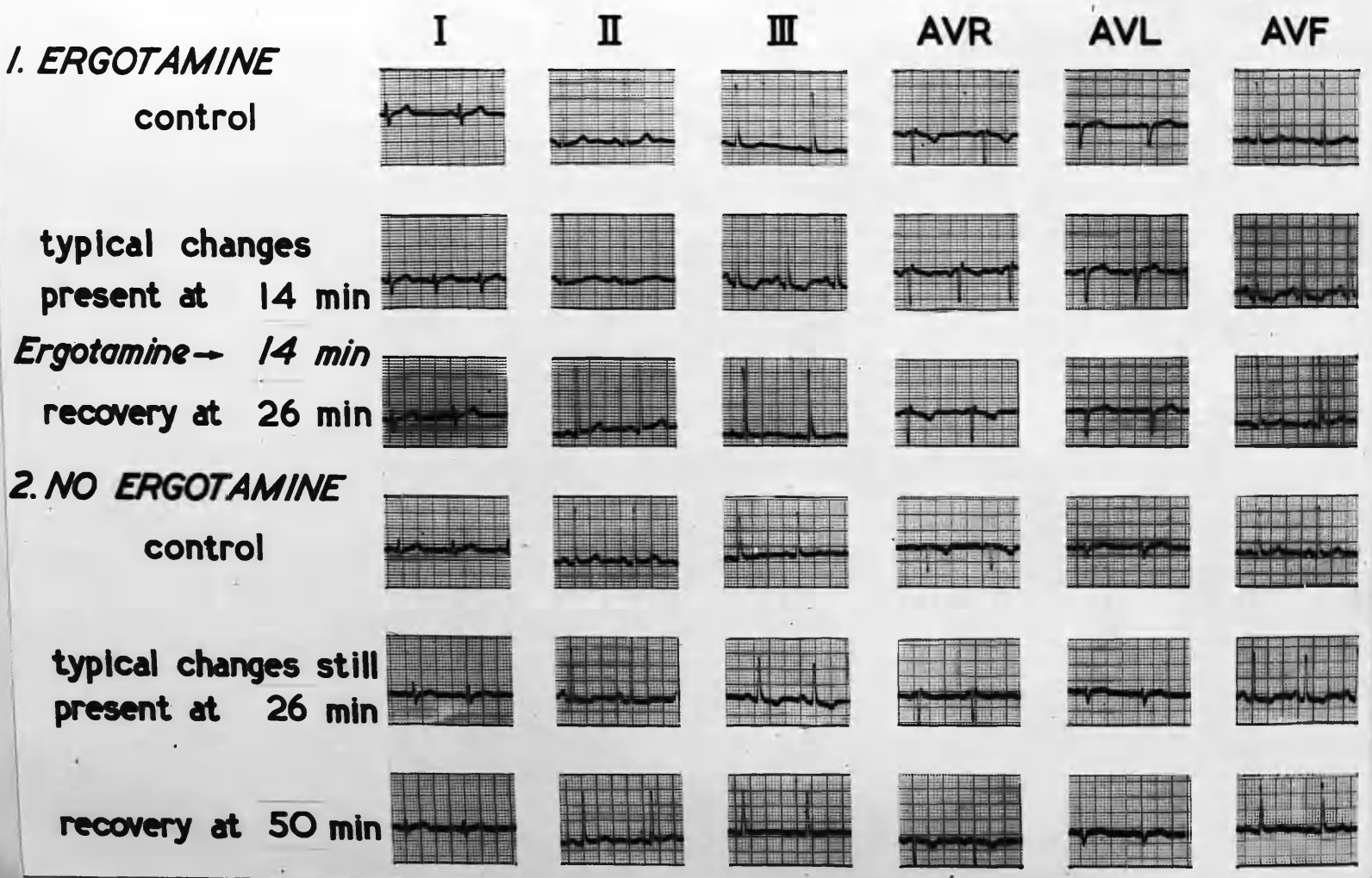
34 mins. Heart rate 72: P:- No change: QRS:- No change: RT:- No change: T:- return to control voltage except II: U:- back to control.

2. GLUCOSE AND NO ERGOTAMINE.

31 mins. Heart rate increased from 62 to 75: P:- No change: QRS:- No change: RT:- No change: T:- flatter I, II, III and aVF and less negative aVR: P:- increased II and aVF.

60 mins. Heart rate 75: P:- No change: QRS:- No change: RT:- No change: T:- still flatter I, II, III and aVF and less negative aVR: U:- still increased II and aVF.

EFFECT OF ERGOTAMINE I.V. No: 6



EFFECT OF ERGOTAMINE - EXAMPLE NO. 6.

PATIENT 60.

1. GLUCOSE AND ERGOTAMINE.

14 mins. Heart rate increased from 65 to 94: P:- peaked II and aVF: QRS:- R II, III and aVF increased: RT:- No change: T:- flatter I, biphasic II, inverted III and aVF, less negative aVR and more positive aVL: U:- No change.

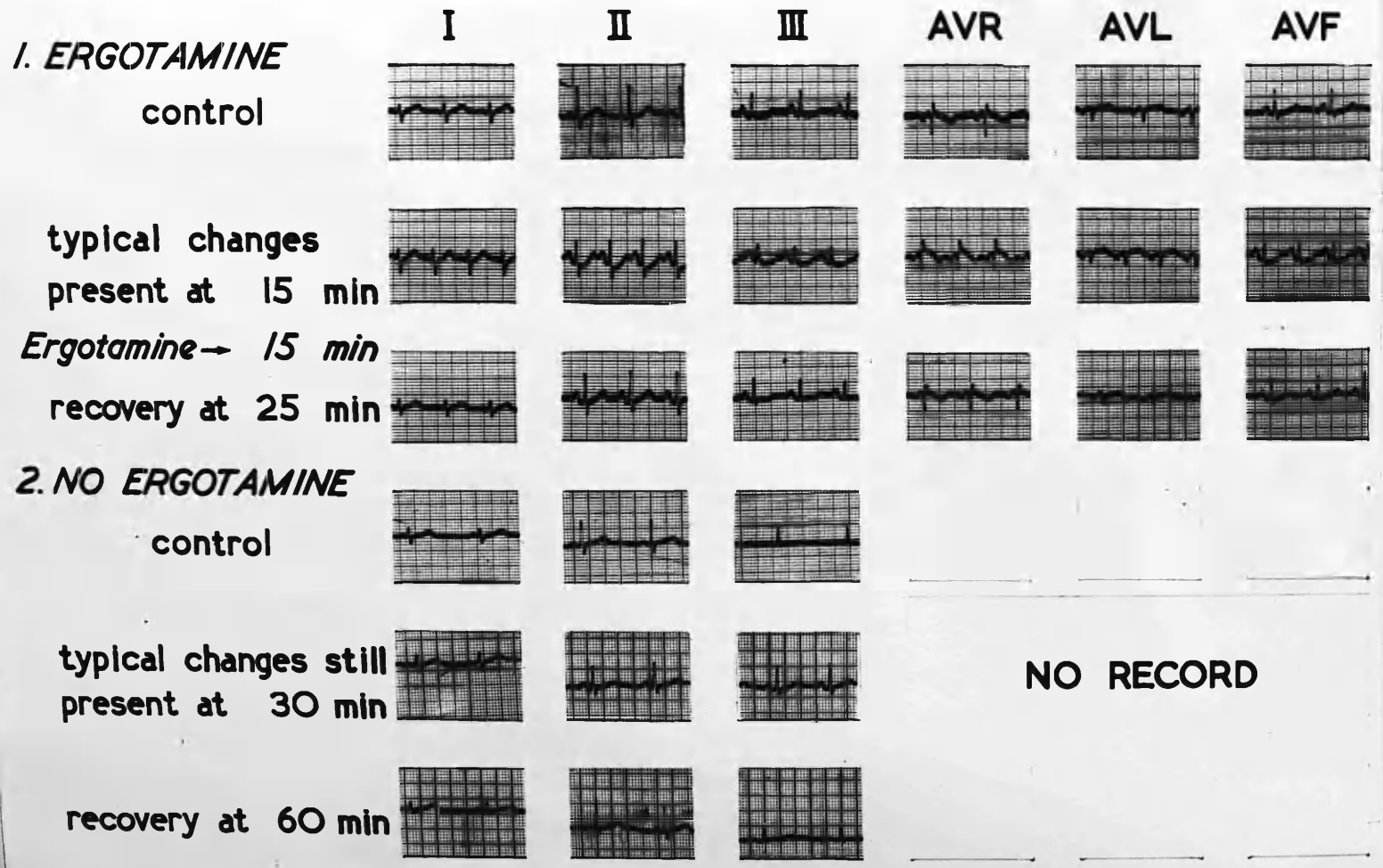
26 mins. Heart rate 75: P:- control: QRS:- R III and aVF still increased. T:- back to control voltage: U:- No change.

2. GLUCOSE AND NO ERGOTAMINE.

26 mins. Heart rate increased from 74 to 84: P:- peaked II and aVF: QRS:- No change: RT:- No change: T:- flatter I, biphasic II and aVR, inverted II and aVF: U:- No change.

50 mins. Heart rate 75: P:- as control: QRS:- No change: RT:- No change: T:- return to control voltage: U:- No change.

EFFECT OF ERGOTAMINE I.V. *No: 7*



EFFECT OF ERGOTAMINE - EXAMPLE NO. 7.

PATIENT 51.

1. GLUCOSE AND ERGOTAMINE.

15 mins. Heart rate increased from 90 to 134: P:- peaked II, III and aVF. QRS:- No change: RT:- ST low take off II and sagging aVF: T:- flatter II, III and aVF and less negative aCR: U:- No change.

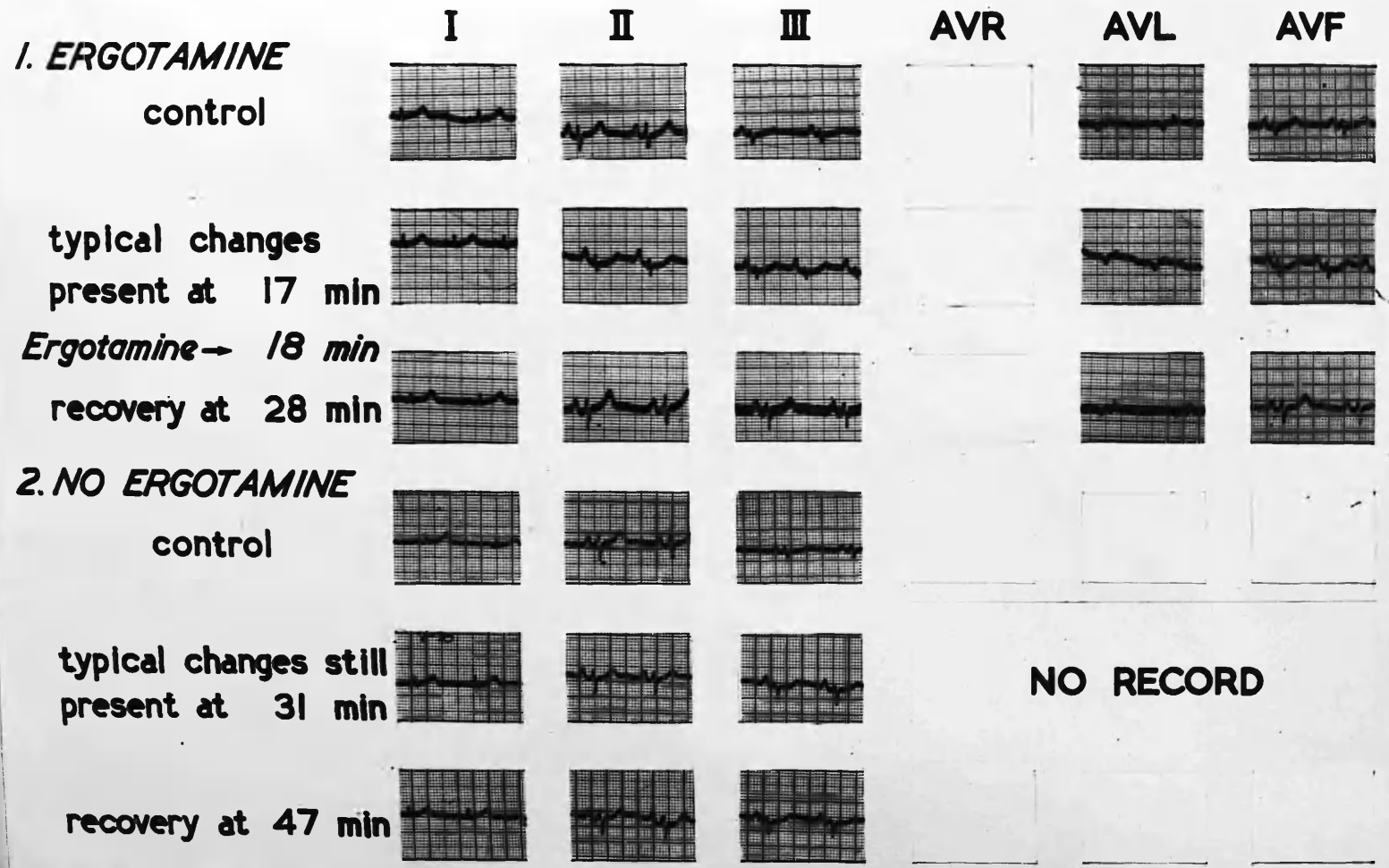
25 mins. Heart rate 100: P:- still peaked in II: QRS:- No change: RT:- No change: T:- as control voltage: U:- No change.

2. GLUCOSE AND NO ERGOTAMINE.

30 mins. Heart rate increased from 65 to 77: P:- peaked in II and III: QRS:- No change: RT:- No change: T:- II flatter: U:- increased II.

60 mins. Heart rate 64: P:- as control: QRS:- No change: RT:- No change: T:- as control: U:- II still increased.

EFFECT OF ERGOTAMINE I.V. *No: 8*



EFFECT OF ERGOTAMINE - EXAMPLE NO. 8.

PATIENT 10.

1. GLUCOSE AND ERGOTAMINE.

17 mins. Heart rate increased from 72 to 88: P:- peaked III and aVF: QRS:- reduced in II: RT:- ST sagging II and aVF: R:- flatter II, III and aVF: U:- increased II and III.

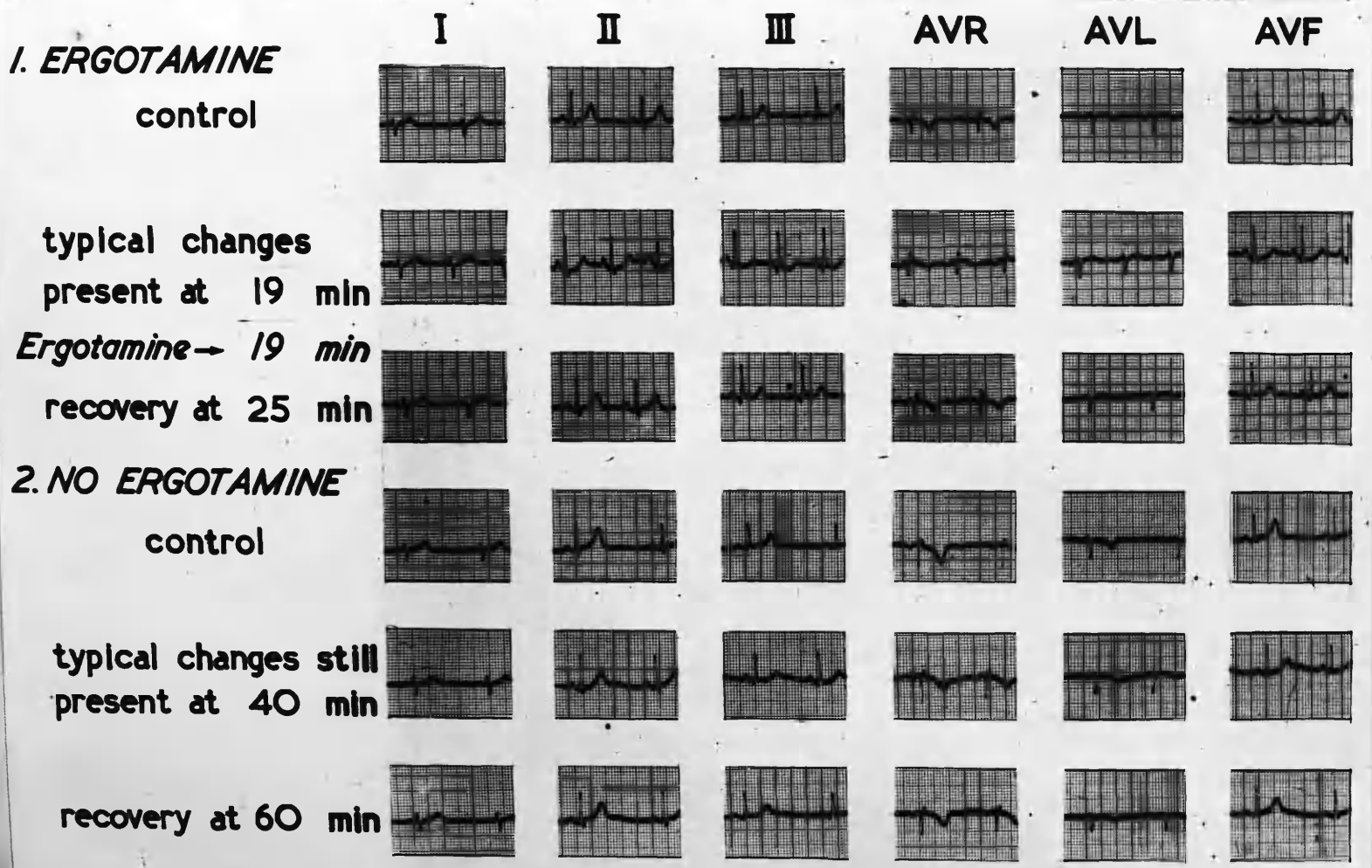
28 mins. Heart rate 62: P:- as control: QRS:- as control: RT:- ST isoelectric: T:- control voltage: U:- still increased in II, III and aVF.

2. GLUCOSE AND NO ERGOTAMINE.

31 mins. Heart rate increased from 66 to 75: P:- peaked in III: QRS:- increased S III: RT:- no change: T:- flatter II, biphasic III: U:- increased II and III.

47 mins. Heart rate 71: P:- still peaked III: QRS:- No change: RT:- No change: T:- as control voltage: U:- still increased II and III.

EFFECT OF ERGOTAMINE I.V. *No: 9*



EFFECT OF ERGOTAMINE - EXAMPLE NO. 9.

PATIENT 25.

1. GLUCOSE AND ERGOTAMINE.

19 mins. Heart rate increased from 65 to 97: P:- peaked in II and III and aVF: QRS:- R III increased: RT:- ST sagging III: T:- flatter I, II, III and aVF, less negative aVR: U:- No change.

25 mins. Heart rate 80: P:- as control: QRS:- as control: RT:- as control: T:- voltage as control: U:- No change.

2. GLUCOSE AND NO ERGOTAMINE.

40 mins. Heart rate increased from 56 to 72: P:- peaked in II: QRS:- No change: RT:- No change III: T:- flatter II, III, and aVF and less negative aVR: U:- increased II, III, aVR and aVF.

60 mins. Heart rate 60: P:- as control: QRS:- No change: RT:- No change: T:- control voltage: U:- still increased II, III and aVF.

PATIENT	SERUM POTASSIUM Mg. per 100 ml.				
	CONTROLS		MIN. AFTER GLUCOSE 15	30	45
2	17.5	18.1	18.1	18.1	18.7
5	-	15.0	17.5	15.0	16.3
10	18.0	16.5	18.0	18.0	17.0
12	21.9	21.9	19.4	19.4	21.3
14	16.0	16.2	15.7	16.0	15.0
15	15.0	15.7	14.7	14.5	14.5
22	17.0	17.0	18.5	17.0	-
23	17.0	19.0	19.0	18.0	18.0
24	16.0	19.5	15.5	14.9	14.5
27	18.0	17.1	17.0	17.0	17.0
29	18.5	18.0	18.0	18.5	18.5
31	15.0	17.5	16.3	16.3	16.3
32	16.0	15.2	16.0	15.8	16.0
34	17.5	18.0	17.5	15.0	18.5
40	15.5	16.5	16.5	19.5	20.5
42	17.5	-	17.0	16.0	-
47	-	14.0	15.1	16.0	16.0
50	-	18.0	18.3	18.0	18.5
51	17.0	16.5	16.5	16.5	20.0
59	18.5	18.0	18.0	16.7	16.0
MEAN	17.2	17.2	17.1	16.8	17.4

SERUM POTASSIUM LEVELS IN 20 PATIENTS WHO HAD ELECTROCARDIOGRAPHIC CHANGES FOLLOWING A MEAL OF 100 ML. 50 PER CENT GLUCOSE.

A: PATIENTS WITH ECG CHANGES				
PATIENT	BLOOD SUGAR MG. PER CENT			
	CONTROL	AFTER GLUCOSE MIN.		
		15	30	45
2	110	140	160	180
3	85	158	197	165
5	95	140	142	130
6	90	120	90	90
12	100	168	195	223
13	95	140	210	220
14	65	-	135	112
18	78	110	180	190
23	85	75	100	110
24	65	105	150	185
25	93	160	230	250
26	103	113	175	195
29	85	105	140	160
30	90	130	223	240
31	95	150	148	100
37	65	100	150	150
38	75	95	140	150
39	100	140	180	200
40	95	125	150	200
42	95	150	175	175
43	95	105	150	205
57	95	170	160	155
MEAN	89	129	163	172

B: PATIENTS WITHOUT ECG CHANGES				
PATIENT	BLOOD SUGAR MG. PER CENT			
	CONTROL	AFTER GLUCOSE MIN.		
		15	30	45
4	100	130	150	130
11	105	120	135	160
16	75	135	225	214
17	120	130	175	140
83	85	92	142	205
MEAN	97	122	165	170

BLOOD SUGAR LEVELS FOLLOWING THE INGESTION OF 100 ML. 50 PER CENT GLUCOSE IN PATIENTS AFTER GASTRECTOMY COMPARING 22 PATIENTS WHO HAD ELECTROCARDIOGRAPHIC CHANGES WITH 5 PATIENTS WHO HAD NO ELECTROCARDIOGRAPHIC CHANGES.

PATIENT	BLOOD SUGAR MG. PER CENT							
	ORAL GLUCOSE				INTRAVENOUS GLUCOSE			
	CONTROL	AFTER GLUCOSE MIN.			CONTROL	AFTER GLUCOSE MIN.		
		15	30	45		15	30	45
13	95	140	210	220	105	325	260	248
30	90	130	223	240	100	185	195	225
31	95	150	148	100	95	170	205	160
32	90	130	185	215	85	158	165	150
MEAN	92	137	191	193	96	209	206	195

BLOOD SUGAR LEVELS IN 4 PATIENTS AFTER GASTRECTOMY COMPARING THE EFFECT OF 100 ml. 50 PER CENT GLUCOSE ORALLY AND 0.5 g. PER Kg. GLUCOSE INTRAVENOUSLY. THE PATIENTS ALL HAD ELECTROCARDIOGRAPHIC CHANGES WITH THE ORAL GLUCOSE BUT NONE HAD CHANGES WITH THE INTRAVENOUS TEST.

PATIENT	GLUCOSE		ATROPINE		POSTURAL CHANGE	
	PULSE	T_{II} MM.	PULSE	T_{II} MM.	PULSE	T_{II} MM.
3	69	5.7	59	5.0	67	3.9
	75	5.3	62	5.0	72	3.2
	82	4.4	56	4.9	97	2.6
	101	3.0	95	3.7	99	2.5
	107	2.3	96	3.7	106	2.5
			97	3.6	111	2.4
			101	3.5		
15	82	2.7	80	2.9	63	2.5
	104	3.0	102	2.7	65	2.2
	107	2.5	104	2.5	78	2.3
	115	2.6	107	2.6	83	2.0
	115	2.7	111	2.5	86	2.1
29	56	4.7	59	5.7	53	5.5
	60	4.5	61	5.4	56	5.2
	67	2.2	69	4.5	60	3.9
	68	3.0	72	4.5	64	4.2
	71	2.5	93	2.5	75	2.5
	73	1.9	95	2.5	75	2.2
					78	1.8

THE HEIGHT OF T WAVE IN LEAD II AND THE CORRESPONDING PULSE RATE IN 3 PATIENTS AFTER GASTRECTOMY SHOWING THE EFFECT OF INGESTION OF 100 ML. 50 PER CENT GLUCOSE, OF THE SUBCUTANEOUS INJECTION OF 1 MGM. ADRENALINE HYDROCHLORIDE AND OF CHANGE OF POSTURE FROM LYING HORIZONTAL TO 75° TO THE HORIZONTAL.

PATIENT	GLUCOSE		ATROPINE		POSTURAL CHANGE	
	PULSE	T_{II} MM.	PULSE	T_{II} MM.	PULSE	T_{II} MM.
41	52	1.8	65	1.2	71	1.8
	56	1.5	65	1.5	74	2.0
	58	1.2	70	1.1	77	2.1
	59	1.5	73	1.0	85	1.0
	59	1.0	84	0.7	87	1.0
	60	0.8	85	0.8	88	1.0
			86	0.6	89	1.0
			87	0.7		
57	77	3.7	65	3.5	51	3.6
	88	3.1	77	2.7	56	3.5
	89	3.0	79	3.0	61	3.5
	89	2.8	88	2.4	62	3.4
	90	2.4	89	2.4	65	3.2
			90	2.3	67	3.2
			95	2.2		2.9

THE HEIGHT OF T WAVE IN LEAD II AND THE CORRESPONDING PULSE RATE IN 2 PATIENTS AFTER GASTRECTOMY SHOWING THE EFFECT OF INGESTION OF 100 ML. 50 PER CENT GLUCOSE, OF THE SUBCUTANEOUS INJECTION OF 1 MGM. ADRENALINE HYDROCHLORIDE AND OF CHANGE OF POSTURE FROM LYING HORIZONTAL TO 75° TO THE HORIZONTAL.

BLOOD SUGAR RESULTS.

PATIENT	BLOOD SUGAR MG. PER CENT			
	CONTROL	MIN. AFTER GLUCOSE 15	30	45
1	85	110	123	110
2	110	115	170	180
3	100	125	160	130
4	80	105	115	105
5	90	110	130	125
6	105	113	147	150
7	103	-	113	115
9	105	105	145	143
10	63	-	103	155
11	70	-	105	160
61	80	100	240	245
62	103	113	195	210
63	75	95	100	95
MEAN	90	108	142	148

BLOOD SUGAR LEVELS FOLLOWING A MEAL OF 100 ML. 50 PER CENT GLUCOSE IN 13 PATIENTS BEFORE OPERATION.

PATIENT	BLOOD SUGAR MGMS. PER CENT			
	CONTROL	MIN. AFTER GLUCOSE 15	30	45
5	95	140	142	130
10	60	100	140	145
12	100	168	195	223
13	95	140	210	220
14	65	-	135	112
15	65	150	165	160
16	75	135	225	214
17	120	130	175	240
18	78	110	180	190
19	93	120	120	235
20	85	125	175	200
21	95	130	190	205
22	120	120	160	200
23	85	75	100	110
24	65	105	150	185
25	93	160	230	250
26	103	113	175	195
27	90	125	145	180
28	113	150	170	160
29	85	105	140	160
MEAN	89	126	166	186
S.E.MEAN	±3.85	±5.51	±7.43	±4.85
"DUMPING" SYMPTOMS				

PATIENT	BLOOD SUGAR MGMS. PER CENT			
	CONTROL	MIN. AFTER GLUCOSE 15	30	45
2	110	140	160	180
3	85	158	197	165
4	100	130	150	130
6	90	120	90	90
7	105	137	120	120
11	105	120	135	180
30	90	130	223	240
31	95	150	148	140
32	90	130	185	215
33	105	140	170	195
34	75	270	235	230
37	65	100	150	150
38	75	95	140	150
39	100	140	180	200
40	95	125	150	200
41	70	140	180	175
42	95	150	175	175
43	95	105	150	205
44	90	-	130	205
45	88	100	155	185
MEAN	91	136	161	176
S.E.MEAN	±3.22	±8.11	±7.53	±4.26
NO "DUMPING" SYMPTOMS				

BLOOD SUGAR LEVELS FOLLOWING A MEAL OF 100 ml. 50 PER CENT GLUCOSE IN 40 PATIENTS AFTER POLYA GASTRECTOMY, COMPARING THOSE WITH AND WITHOUT "DUMPING" SYMPTOMS.

PATIENTS	BLOOD SUGAR MG. PER CENT			
	CONTROL	MIN. AFTER GLUCOSE 15	30	45
1	95	150	137	125
8	80	100	155	150
9	90	130	210	190
47	88	90	105	125
48	85	105	160	198
49	90	138	190	194
50	105	145	165	145
MEAN	90	123	160	161

BLOOD SUGAR LEVELS FOLLOWING A MEAL OF 100 ML. 50 PER CENT GLUCOSE IN 7 PATIENTS AFTER GASTROJEJUNOSTOMY WITH VAGOTOMY.

PATIENT	BLOOD SUGAR MG. PER CENT							
	BILLROTH CONTROL	MIN. AFTER GLUCOSE 15	30	45	POLYA CONTROL	MIN. AFTER GLUCOSE 15	30	45
14	138	145	190	196	65	-	135	112
15	105	145	195	140	65	150	165	160
22	83	113	165	170	120	120	160	200
34	100	140	195	230	75	270	235	230
41	90	120	170	155	70	140	180	175
44	80	103	130	160	88	100	155	185
MEAN	97	124	164	165	81	156	172	177

BLOOD SUGAR LEVELS FOLLOWING A MEAL OF 100 ML. 50 PER CENT GLUCOSE IN 6 PATIENTS WHO HAD BOTH A POLYA AND A BILLROTH I PARTIAL GASTRECTOMY.

PATIENT	BLOOD SUGAR MG. PER CENT							
	LYING CONTROL	MIN. AFTER GLUCOSE 15	30	45	SITTING CONTROL	MIN. AFTER GLUCOSE 15	30	45
16	83	180	195	200	75	135	225	214
25	80	148	158	288	93	160	230	250
34	90	170	180	210	75	270	235	230
38	80	130	180	205	75	95	140	150
43	75	160	170	200	95	105	150	205
MEAN	82	158	177	221	83	153	196	219

BLOOD SUGAR LEVELS FOLLOWING 100 ml. 50 PER CENT GLUCOSE IN 5 PATIENTS AFTER GASTRECTOMY COMPARING INTRAJEJUNAL ADMINISTRATION WHEN LYING, AND ORAL INTAKE WHEN SITTING.

IRON ABSORPTION RESULTS.

PATIENT	OPN.	Hb. g %	PCV %	MCHC %	SERUM IRON Mg.%	59 FE % DOSE		FREE HCl IN GASTRIC JUICE	F.O.B.	MARROW IRON
						BLOOD	FAECES			
A	P	17.2	56.5	30	125	44	56	+	-	NOT DONE
	B I	17.7	57.5	31	220	43	54	+	-	"
B	P	11.3	36.5	31	75	4	87	+	-	"
	B I	11.3	36.0	31	80	7	78	+	-	NORMAL
C	P	16.0	48.5	33	85	14	82	+	-	NORMAL
	B I	16.3	50.0	32.5	110	4	102	+	-	NOT DONE
D	P	12.7	41	31	80	11	89	+	-	NOT DONE
	B I	13.9	44	31.5	90	17	79	+	++	"

FINDINGS BEFORE AND AFTER CHANGE OF SITE OF ANASTOMOSIS FOLLOWING PARTIAL GASTRECTOMY IN PATIENTS A AND B FROM POLYA (P:GASTROJEJUNAL) TO BILLROTH I (B I GASTRODUODENAL): IN PATIENTS C AND D VICE VERSA.