connexion we must consider Hughes's findings (which we have confirmed by another method) that in something like 14% of cases, after inoculations into man with a Record syringe, some body fluid enters the needle and can be transferred to the syringe in the process of changing the needle.

The amount of body fluid gaining access to the needle may be very small, and in the vast majority of cases it would not be infected. The potential danger, however, remains, and clearly the method of changing needles should be discarded, especially if one equally convenient can be substituted.

We have shown that, without removing them from the syringe, needles can easily and quickly be sterilized by immersion for 10 seconds in liquid paraffin at a temperature of 130° C. or over. If the oil is to be used only to sterilize needles then it might be heated to about 150° C., which obviously gives a greater margin of safety, although we have shown that at 130° C. sterilization is complete in three seconds or less.

If hot oil is not obtainable, immersion of the needle for 10 seconds in boiling water is effective. Oil is preferable, as the needles remain sharp for a longer time and do not rust.

In mass inoculation there is a second problem to be considered—namely, the possibility of reflux of fluid from the body of the inoculated man beyond the needle into the syringe. When multiple doses are given from one filling of the syringe, it is very difficult to be certain that in the course of the series of inoculations some accidental movement of the piston of the syringe does not occur. Quite apart from the possibility that there might be sufficient pressure in the tissues to cause reflux of the fluid into the syringe, any movement of the piston might easily draw a small quantity of fluid back through the needle. There is therefore a possibility of danger in this method of taking up into the syringe a number of doses and giving them one after another to different people, however thoroughly the needle is sterilized.

We have provided evidence that if only one dose is taken up into the syringe, and after inoculation pressure is kept up on the piston until the needle is withdrawn, there is no reflux of fluid into the syringe even when the injection is made against pressures enormously greater than could possibly obtain in the body.

This method of inoculating, combined with sterilization of the needle by 10 seconds' immersion in oil at over 130° C., seems to offer a method which is perfectly safe and which is rapid enough for mass inoculation.

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According to a recent news report in the *New York Times*, Dr. J. E. Smith, of the United States Civil Aeronautics Administration, has invented an instrument which records the heart beat, quite literally. That is to say, it is a miniature seismograph which is strapped across the patient's shins and records the vibration in the body from each beat. The ballistocardiograph has been in diagnostic use for some time, but Dr. Smith's apparatus is apparently an improved version and works in conjunction with an electrocardiograph.

POST-GASTRECTOMY SYNDROMES

A STUDY IN APPLIED PHYSIOLOGY

BY

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Gastrectomy is a good and safe operation for the relief of peptic ulceration. The mortality is less than 2% in capable hands, and over 90% of the patients are well and lastingly satisfied. The results of various forms of gastrectomy for carcinoma of the stomach and oesophagus are steadily improving. We are concerned here, not with the vanishing problem of recurrent ulceration or with recurrence of growth, but with those other sequelae of operation which collectively are known as the "post-gastrectomy syndromes." We believe that they all result from, and can be understood in terms of, the alterations in alimentary physiology which follow operations on the stomach. The more we study these conditions the more we are able to see a coherent pattern into which the different pieces can be fitted. We have studied in detail some 300 patients who have undergone gastrectomies of various types, and many of them have been seen repeatedly and fully investigated. A fair proportion of them have been "problem patients" who have been referred to us. We have not studied in detail the effects of gastro-enterostomy or of vagotomy, but shall refer to them in so far as they throw light on the problems of gastrectomy.

Normal Physiology*

A knowledge of the normal physiology of the stomach is essential to an understanding of the effects of the various operations. It may be considered under four headings (Fig. 1).

1. *Reservoir Function of Stomach: Gastric Emptying.*—The main function of the stomach seems to be the preparation of food for the small intestine, in which most of the digestion and absorption occurs. Food is mixed, diluted with gastric juice, and converted by churning peristaltic movements of the pyloric end of the stomach into liquid chyme, which is passed at a suitable rate through the pylorus and into the duodenum. The rate at which the stomach empties depends on the difference in pressure on the gastric and duodenal sides of the pylorus, and this is regulated by various factors. Thus the presence of hydrochloric acid or of products of protein digestion in the duodenum delays the rate of gastric emptying by a reflex mechanism which involves the vagi. Fat and carbohydrate in the duodenum stimulate the production of enterogastrone, which delays gastric emptying by a humoral mechanism. Hypertonic solutions of sugar, protein digests, etc., which are formed from food, are rapidly diluted in the stomach, so that they are isotonic by the time they reach the jejunum. Gastric movements and tone are normally under the control of the vagus nerves.

2. *Secretion of Hydrochloric Acid.*—HCl is secreted by the oxyntic cells, most of which are found in the body of

*In the anatomical nomenclature we have followed the British Revision (1933) of the B.N.A. (Gray, 1935). Confusion exists because many authors still refer to the "body" of the stomach as the "fundus" and to the "fundus" of the stomach as the "cardia."

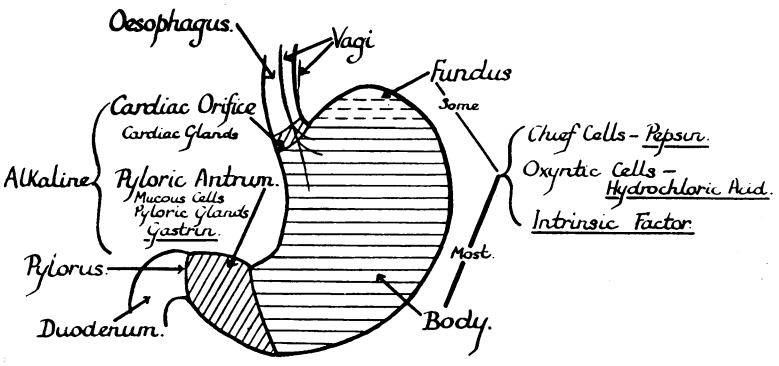

FIG. 1.—The stomach. Principal functions.

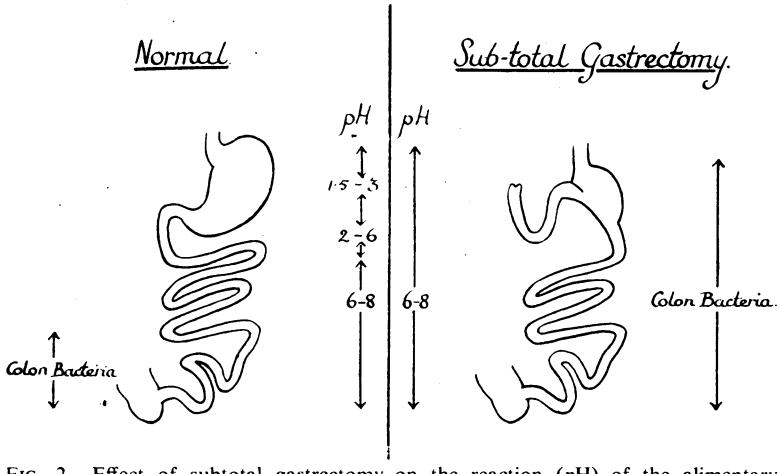

FIG. 2.—Effect of subtotal gastrectomy on the reaction (*p*H) of the alimentary tract and on bacterial growth.

the stomach, only a small number being present in the fundus. Two distinct mechanisms are involved. The *vagal flow* is stimulated by the thought, smell, sight, or taste of food, and also occurs during sleep. The *humoral flow* occurs under the influence of gastrin, which is secreted mainly by the mucous membrane of the pyloric antrum. The acidity of the full stomach is high (*p*H 1–3) (Fig. 2). As chyme passes through the duodenum it is diluted and the acid is partly neutralized. The *p*H rises rapidly, and the reaction of the jejunal and ileal contents is approximately neutral (*p*H 6–8). Strongly alkaline reactions are not found. The acid reaction of the stomach provides optimum conditions for the proteolytic action of pepsin. It encourages the solution and dissociation of iron salts so that they may be easily absorbed in the small intestine; it has a protective action on some of the vitamins; and it has a sterilizing effect on the food, so that bacteria are unable to grow in the upper reaches of the alimentary tract.

3. *Secretion of Pepsin.*—Pepsin is secreted by the chief cells, which have a distribution similar to that of the oxyntic cells. It is not essential to health, because the pancreatic and small-intestinal enzymes can digest proteins completely to amino-acids.

4. *Production of Intrinsic Factor.*—The "intrinsic factor" is produced by the body and, to a less extent, by the fundus of the stomach (Fox and Castle, 1942; Wilkinson, 1949). This factor activates or aids the absorption of the "extrinsic factor," which is present in food, and the resulting "liver factor" is essential for haemopoiesis. It is not yet known what relation these factors bear to vitamin B_{12} or to folic acid.

Types of Operation

Modern operations for the relief of peptic ulceration are designed to reduce the amount of hydrochloric acid. Partial gastrectomy has steadily gained favour in place of gastro-enterostomy, and the operation has become more and more radical. The standard procedure to-day is *subtotal gastrectomy* (Fig. 3, *2 and 1*), in which at least three-quarters of the stomach is removed, including the pyloric antrum (to remove gastrin) and the whole of the body (to remove HCl). The fundus only is left behind, and is joined to the duodenum or jejunum. There are several ways in which the anastomosis can be made: (1) End-to-side with jejunum (Fig. 3, *1*); retrocolic (Polya type) (*1a*); antecolic (Balfour type) (*1b*); left-to-right (Moynihan type) (*1c*); right-to-left without a valve (*1a, 1b*); right-to-left with a valve (Finisterer–Lake type) (1*d*). (2) End-to-end with duodenum (Billroth I type) (Fig. 3, *2*). It is better to describe the anastomosis anatomically than to use the debatable terms "isoperistaltic" and "antiperistaltic."

The same operations may be used in the treatment of carcinoma, but there are two other types of gastrectomy which are commonly used in this condition:

Oesophago-gastrectomy (Fig. 3, *3*), which is used for growths of the oesophagus and cardiac end of the stomach. A varying length of oesophagus is resected together with up to three-quarters of the stomach at its cardiac end. The remaining stomach is brought up into the chest and anastomosed to the proximal end of the oesophagus. A vagotomy is unavoidably performed at the same time.

Total gastrectomy (Fig. 3, *4*), which is used for more extensive growths of the stomach and sometimes for high peptic ulcers. As a rule the oesophagus is anastomosed to a loop of jejunum, and an entero-anastomosis is performed between the two limbs of the loop (*4a*). Alternatively, a Roux-en-Y form of anastomosis may be used (*4b*).

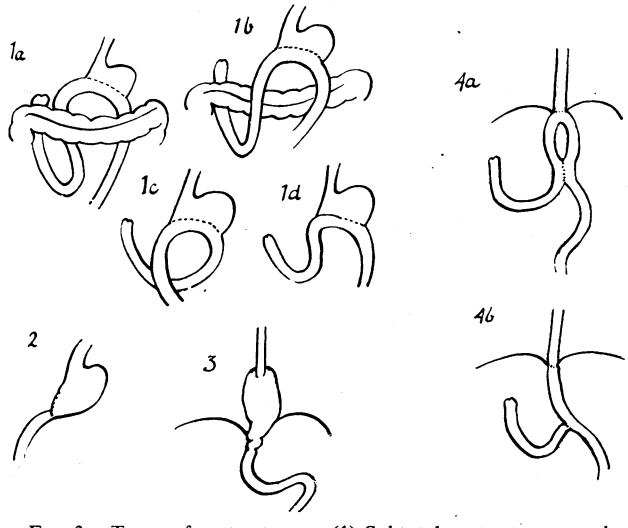

FIG. 3.—Types of gastrectomy. (1) Subtotal gastrectomy; end-to-side gastro-jejunal anastomosis: (*a*) retrocolic (Polya); (*b*) antecolic (Balfour); (*c*) left-to-right (Moynihan); (*d*) right-to-left with a valve (Finsterer–Lake). (2) Subtotal gastrectomy; end-to-end gastro-duodenal anastomosis (Billroth I). (3) Oesophago-gastrectomy. (4) Total gastrectomy with oesophago-jejunal anastomosis: (*a*) end-to-side with entero-anastomosis; (*b*) end-to-end (Roux-en-Y).

Physiological Effects of Gastrectomy

Rapid Emptying of the Gastric Remnant (Small-stomach Sequelae)

After subtotal gastrectomy the stomach is no longer able to perform its reservoir function adequately. The consequent disturbances may be regarded as the "small-stomach sequelae." After operations with a gastro-jejunal anastomosis the gastric stump behaves like a funnel, pouring food into the jejunum. The reflex and humoral mechanisms, which normally operate from the duodenum to control the rate of gastric emptying to two to three hours, are completely by-passed. Within 10 to 15 minutes of taking a barium meal several feet of small intestine are seen filled and the gastric stump is often virtually empty. When a large bulk of food enters the jejunum rapidly, it stimulates active peristalsis and is passed down the small bowel very fast. Foods which form hypertonic solutions are particularly apt to be treated in this way. It has been pointed out already that they are normally held in the stomach until they have been diluted. When they are introduced direct into the jejunum they are diluted there by an outpouring of fluid from the jejunal mucosa (Machella, 1949, 1950), and their bulk is increased very greatly.

The stimulus to peristalsis may be enormous, and in an extreme case (Welbourn and Glazebrook, 1951) a mixture of barium and hypertonic glucose reached the caecum in five minutes. In many cases, however, the increased activity affects the jejunum only, there being a compensatory slowing in the ileum. This passage of hypertonic solutions direct into the jejunum, with consequent rapid intestinal passage, is of fundamental importance in the understanding of several of the post-gastrectomy syndromes. After total gastrectomy this effect is exaggerated, while after the Billroth I operation (gastro-duodenal anastomosis) and oesophago-gastrectomy it is not nearly so much in evidence. The duodenum has a marked capacity for diluting hypertonic solutions, and probably exerts a protective action on the jejunum.

Stasis and Reflux

The altered anatomical arrangements which result from the various types of anastomosis may cause interference with the rate and direction of flow of both food and digestive juices, causing regurgitation or actual vomiting of bile or food or of both.

Reduction of Acid

The reduction of HCl is directly proportional to the amount of body of the stomach which is removed, and the pyloric antrum (because it secretes gastrin) is important out of proportion to its size. The fundus of the stomach usually produces no acid after the antrum has been removed, and complete achlorhydria is found in 85–90% of cases after subtotal gastrectomy. Complete achlorhydria is always present after total gastrectomy. Vagotomy alone reduces the acidity to about one-third of its pre-operative level (Beattie, 1948). Oesophago-gastrectomy (which includes vagotomy) also produces hypochlorhydria unless the gastric resection is extensive, when complete achlorhydria may result.

The effect of achlorhydria on the *p*H of the alimentary tract is shown in Fig. 2. There is no *p*H gradient as there is in the normal upper tract, and the contents of the gastric stump (and of the duodenum after the Billroth I type of operation) have the neutral reaction (*p*H 6–8) of the small intestine (Welbourn, 1950). There may be interference with the absorption of certain food substances, especially iron and vitamins, and bacteria which normally inhabit the colon grow freely all the way up the small intestine and in the gastric stump (Fig. 2) (Welbourn, Hughes, and Wells, 1951).

After gastro-enterostomy, and gastrectomies which fail to abolish the secretion of HCl, frankly acid reactions (*p*H 4) are found in the jejunum (Pantlitschko and Schmid, 1950) and ulcers may recur.

Removal of Intrinsic Factor

In subtotal gastrectomy most of the area which produces the intrinsic factor is removed (Fig. 1). On the other hand, a *p*H of 7 is optimum for the interaction of intrinsic and extrinsic factors, so that the small amount of intrinsic factor which is secreted by the fundus is usually adequate. The intrinsic factor *may* also be secreted by the duodenum or small intestine in man, although there is no direct evidence for this. The best *indirect* evidence is the rarity of megaloblastic anaemia after total gastrectomy.

Post-gastrectomy Syndromes

Serious defects in digestion and absorption might be expected to occur regularly as a result of these "physiological insults" to the alimentary tract. But the functional reserve is such that in most cases there is no disturbance. Indeed, many patients whose nutritional state was poor at the time of operation are restored to full health afterwards. On the other hand, such sequelae as do occur after the various forms of gastrectomy can be understood on the basis of these physiological effects.

The term "dumping syndrome" has been coined and used very loosely to describe almost any symptoms which may occur at any time after meals in patients who have

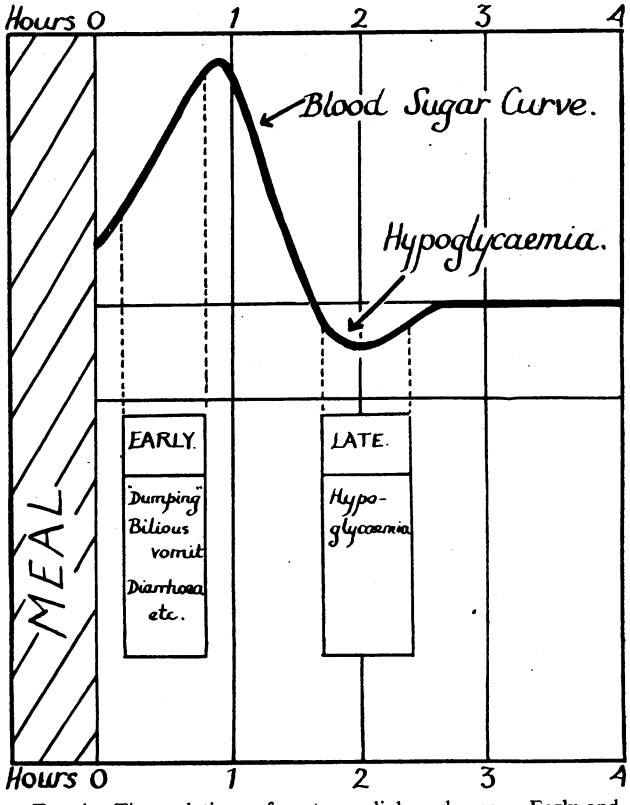

FIG. 4.—Time relations of post-prandial syndromes. Early and late syndromes are shown in relation to a meal and to the blood-sugar curve.

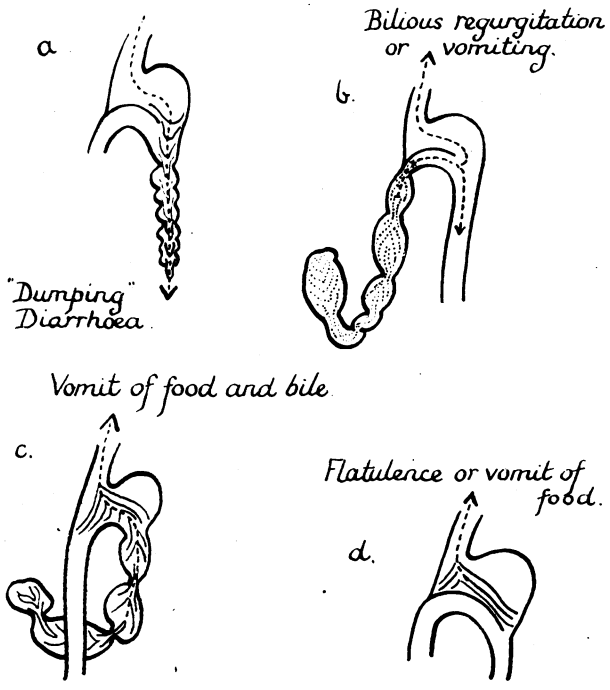

FIG. 5.—Mechanism of early post-prandial syndromes: (*a*) rapid emptying of gastric remnant; (*b*) afferent-loop stasis; (*c*) afferent-loop reflux; (*d*) gastric distension.

undergone gastrectomy. Many different syndromes can be distinguished, with different mechanisms and distinct symptoms and time relations (Figs. 4 and 5). The term "dumping" should be applied to one of these only (the efferent-loop syndrome), and more accurately descriptive names should be used for the others, including those associated with various deficiency states.

The accompanying scheme shows the main relations of the various post-gastrectomy syndromes to the physiological effects of operation. It is most important to bear in mind these aetiological relationships, but for descriptive purposes it is simpler to arrange the syndromes in clinical groups—namely, the early post-prandial, the late post-prandial, and the deficiency syndromes.

A. Rapid emptying of gastric remnant (Small-stomach sequelae) →	Efferent-loop dumping syndrome	(Early post-prandial syndromes)
	Post-prandial diarrhoea	
	Impaired digestion and absorption	
	Hypoglycaemic syndrome	(Late post-prandial syndrome)
B. Stasis and reflux →	Afferent-loop stasis and bilious regurgitation	(Early post-prandial syndromes)
	Afferent-loop reflux	
	Gastric distension	
C. Achlorhydria →	Iron-deficiency anaemia	(Deficiency syndromes)
	Vitamin-B deficiencies	
D. Removal of intrinsic factor →	Macrocytic anaemia	

To complete the scheme we must add:
Early post-prandial syndromes → Reduced calorie intake → Loss of weight (Deficiency syndrome)
Impaired digestion and absorption → Reduced calorie intake → Loss of weight (Deficiency syndrome)

Early Post-prandial Syndromes

This term was introduced by Adlersberg and Hammerschlag (1947) to distinguish the conditions which cause symptoms shortly after a meal from the hypoglycaemic syndrome, which causes symptoms after two to three hours—that is, late post-prandial (Fig. 4). There are several recognizable causes, and two groups of symptoms and signs may be distinguished:

1. Those which are peculiar to the individual syndromes. They include regurgitation or vomiting of bile or food, or of both, and diarrhoea.

2. Those which, when present, are common to all the early post-prandial syndromes. They result from afferent impulses in the splanchnic nerves, and may also be divided into two groups: (*a*) True visceral sensations, which are felt in the abdomen—namely, fullness in the epigastrium, which may amount to marked discomfort, but rarely to actual pain; consciousness of intestinal movements; and bowel colic. (*b*) Those which we believe to be due to "irradiation" of impulses in the nervous system: They are listlessness and fatigue; nausea; giddiness, headaches, etc.; and vasomotor phenomena, such as sweating, palpitations, pallor, a feeling of warmth, and an increased heart rate and blood pressure.

Many of the symptoms can be produced by distending a rubber balloon in the jejunum (Ray and Neill, 1947; Muir, 1949; Machella, 1949, 1950). All of them can be produced by introducing rapidly into the jejunum a large volume of isotonic solution (Alvarez, 1949) or a smaller volume of hypertonic solution (Machella, 1949, 1950). Welbourn and Glazebrook (1951) have shown by radiological and kymographic studies that hypertonic glucose stimulates active contraction and peristalsis in the jejunum while the symptoms are present. It is reasonable, therefore, to suppose that it is *increased tension* in the jejunum rather than distension (which has not been demonstrated) that is the adequate stimulus for the production of symptoms. Alvarez (1949) has observed that ice-cold water and specific allergens introduced into the jejunum will also reproduce the symptoms, but it is not clear whether these act by increasing tension or in some other specific manner.

Capper and Butler (1950) have produced the symptoms by placing a mercury-weighted balloon in the gastric stump in patients who have had gastrectomies with a gastro-jejunal anastomosis. They point out that the stomach is no longer supported by the lesser omentum, and that in the upright position food or mercury in the stomach will exert a considerable pull on the oesophago-gastric junction, with consequent splanchnic stimulation. This mechanism may well contribute to the production of symptoms after this type of operation, but can hardly operate in individuals with intact stomachs who occasionally experience similar symptoms. There must therefore be some other factor such as we have already described.

A neurogenic basis for the symptoms is suggested by the fact that interruption of the afferent nervous pathway will prevent their onset when the usual stimuli are applied. This can be achieved by bilateral thoracolumbar splanchnicectomy (Ray and Neill, 1947), by procaine injection of the splanchnic nerves and sympathetic trunks (Capper, 1950), and by the administration

of ganglion-blocking agents such as pentamethonium and hexamethonium salts (Welbourn and Glazebrook, 1951). These last also act by reducing bowel tone and activity.

The following are the varieties of early post-prandial syndromes.

1. Efferent-loop Dumping Syndrome (Fig. 5, a)

This is the commonest and most important of the post-gastrectomy syndromes.

Clinical Features.—Very shortly after the end of a meal (or even before the meal is finished) the patient feels full in the epigastrium and is overcome with a desire to sleep. Fullness alone cannot be regarded as a "syndrome." These symptoms may limit the size of the meal, and may cause the patient to sit in a chair or actually to lie down for 15 to 30 minutes or even longer, until they have passed off. Other symptoms—sweating, palpitations, nausea, giddiness, etc.—may be present, but in our experience are less constant. Occasionally fatigue or other symptoms are present without the feeling of fullness. In the most severe cases there may be violent peristalsis with borborygmi and actual colic, and the symptoms may last for one to two hours, ending in an attack of watery diarrhoea. All the symptoms tend to be eased by lying down, which reduces the rate of gastric emptying.

The symptoms usually appear as soon as the patient starts to take normal-sized meals after a gastrectomy. They may appear after every meal, but usually follow only the main meal of the day. Certain foods, especially milk, sweets, and rich puddings, may produce symptoms constantly, but more often the size of the meal is the determining factor.

The syndrome is commonest after operations with a gastro-jejunal anastomosis (Fig. 3, *1*), but opinion differs regarding the incidence. Muir (1949) reported it in 75% of cases (but this figure seems to include patients with fullness only), while Lake (1948) reported it in 3%. In our own series (of Finsterer-Lake type of subtotal gastrectomies) (Fig. 3, *1d*) we have met it in 35%, and in many it has passed off within one to two years. In only 2 to 3% does it persist in a severe form, and in these it is a serious handicap. It is uncommon after a gastro-duodenal anastomosis. We have met it after total gastrectomy and oesophago-gastrectomy.

Mechanism.—Alvarez (1949) has encountered the condition in otherwise normal people with rapidly emptying stomachs, and most people have experienced fullness, listlessness, and fatigue after a large meal. The essential feature is the rapid entry of a large bulk of food into the jejunum. After operation hypertonic solutions—for example, sweet foods—are particularly troublesome because their bulk is increased rapidly by the diluting fluid from the jejunum itself. It is important to remember that what occurs is heightened bowel activity, and not bowel distension. This is reflected in the intestinal movements and colic which are sometimes felt, and the diarrhoea which sometimes occurs in the most severe cases.

It is not clear why some people have dumping symptoms after gastrectomy and others do not. It must be supposed either that the jejunum reacts with different degrees of intensity in different people or that there is an individual variation in the threshold for the appreciation of symptoms. The disappearance of symptoms with the passage of time probably reflects an adaptation on the part of the jejunum to the new conditions created by the gastrectomy.

Management.—Various precautionary measures can be taken at the time of operation. The Billroth I operation is technically difficult in duodenal ulcer and ulceration is apt to recur (Wells and Brewer, 1948). O'Neill (1950) uses a small central stoma and Steinberg (1949) a "pantaloons" anastomosis; Capper and Butler (1950) reconstitute the lesser omentum to "hitch" up the lesser curve and lessen the drag on the oesophagus.

When a patient leaves hospital after a gastrectomy he should be told that at first he must take small meals at frequent intervals, and chew his food well. He must be assured that during the next few months he will be able to eat more and more until he is taking normal meals. If dumping symptoms are complained of he must be reassured and advised to reduce the bulk of his meals by taking them dry and drinking in between them. Any foods which are particularly troublesome should be avoided. Phenobarbitone, or even a placebo, is sometimes effective. We have found this regime quite satisfactory in most cases. Tanner (1950), however, advises taking large meals as soon as possible in the hope that adaptation will occur more rapidly.

In those who have not responded to these simple measures after a year or so, certain drugs which reduce bowel motility may be tried. Machella (1949) recommends atropine, but we have had little success with it clinically. Hexamethonium bromide has been helpful in investigations, and in some people it greatly reduces the symptoms, but its effect is too uncertain for it to be used routinely. Further trials are required with other drugs with similar pharmacological actions.

There is little agreement on operative procedures that may be used. Muir (1949) suggests that an entero-anastomosis between the afferent and efferent jejunal loops should be effective by increasing the space into which the stomach can "dump" its contents. We criticize this in a later paragraph, and prefer the operation illustrated in Fig. 6, *3*. Perman (1947) recommends converting a Polya into a Billroth I type of anastomosis, but technically this may be very difficult. Simple reduction in the size of the stoma may be effective (Machella, 1950). Capper (1950) has used his "hitching" operation successfully in a number of cases.

2. Post-prandial Diarrhoea (Fig. 5, a)

In this uncommon syndrome diarrhoea occurs within 15 to 20 minutes of the end of a meal, and in some cases food which has just been eaten appears in the stool. There are no other symptoms apart from a consciousness of bowel movement and sometimes colic. We regard this as an exaggerated form of the intestinal hurry already noted as resulting from rapid emptying of the gastric stump into the jejunum. The fact that no other symptoms are present suggests that in these people there is a high symptom threshold. We have, however, seen one patient who had this syndrome constantly for two years and then developed a typical efferent-loop dumping syndrome without diarrhoea. The diarrhoea tends to appear in episodes with exacerbations and remissions, and the tendency is for it to clear up completely

The stools are often offensive, which suggests that bacterial growth in the intestine may be a contributory factor, and we have had some success with giving calcium lactate, 10 gr. (0.65 g.), before meals with the object of precipitating the irritant fatty acids which may be produced by the bacteria (Frazer, 1949a). Drugs which reduce the rate of small-intestinal passage have also proved helpful.

3. Afferent-loop Stasis and Bilious Regurgitation (Fig. 5, b)

This syndrome results from the fact that in certain operations the duodenum and afferent jejunal loop are converted functionally into an extension of the common bile and pancreatic ducts.

Clinical Features.—Some time after the end of a meal (usually 10–45 minutes) the patient brings up a mouthful of bitter-tasting fluid (usually described as "acid"), which may be swallowed again or actually vomited. It is usually bile-stained, but may be colourless, and contains no food. The regurgitation may be produced constantly by certain foods, especially fats. Occasionally a large bilious vomit (½ to 2 pints—0.28 to 1.14 litres) occurs, especially in the evening or during the night. Most of the symptoms of splanchnic stimulation (but especially fullness and nausea) may precede the vomiting, but are not usually prominent.

They may steadily increase for two to three days before a large bilious vomit, and the stools may become pale. The symptoms are always relieved after the bile has been brought up.

Some bilious regurgitation is often troublesome for the first few weeks or months after a gastrectomy with gastro-jejunal anastomosis (Fig. 3, *1*). It then usually passes away completely. In only a small number of patients does it persist. Very rarely it appears for the first time some years after operation. It is much less common with a gastro-duodenal anastomosis (Fig. 3, *2*) and after total gastrectomy. Patients who have had a cholecystectomy as well as a gastrectomy, and who have consequently lost their power of concentrating bile, tend particularly to be troubled. An antecolic anastomosis gives no more trouble than a retrocolic anastomosis, provided the afferent loop is not unduly long. This syndrome is sometimes associated with the efferent-loop dumping syndrome, but we believe that the two conditions are essentially different.

Mechanism.—These phenomena are probably caused by bile and pancreatic juice accumulating in the afferent jejunal loop owing to a slight kink at the lesser curvature. When a certain volume of fluid has accumulated it stimulates active peristalsis, which overcomes the obstruction and empties the fluid rapidly into the stomach. All the fluid cannot be easily accommodated and a proportion is vomited. Food does not regurgitate, because it has already passed on down the efferent loop. The large vomits which occur occasionally result from an exaggeration of the same mechanism.

Management.—Reassurance that there is nothing seriously wrong and that the symptoms will soon pass is all that is usually required, but troublesome foods (especially fats) should be avoided. Operation is necessary if symptoms are serious after a year or so. Illingworth (Muir, 1949) claims excellent results from entero-anastomosis (Fig. 6, *2*), but this procedure cannot be without risk of recurrent ulceration, and simultaneous abdominal vagotomy should be considered. We have devised an operation (which

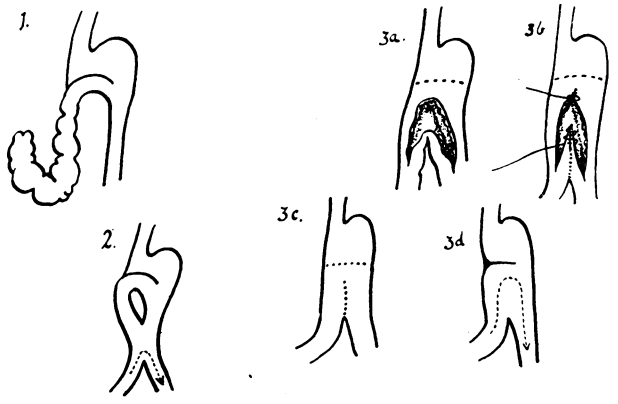

FIG. 6.—Operations for relief of bilious vomiting. (1) Afferent-loop stasis. (2) Entero-anastomosis. (3) Jejunoplasty: (*a*) line of incision; (*b*) insertion of sutures; (*c*) sutures completed (anterior view); (*d*) completed operation (in section).

is an adaptation of the "pantaloons anastomosis") for the relief of bilious vomiting. It consists essentially in performing a jejunoplasty between the two jejunal loops immediately adjacent to the stoma (Fig. 6, *3*). We have performed it in three cases with excellent results. The operative details and case histories will be reported when we have a larger series.

4. Afferent-loop Reflux (Fig. 5, c)

In this syndrome *food mixed with bile* is vomited shortly after a meal. Symptoms of splanchnic stimulation may be present, and are relieved by vomiting. It is caused by the passage of food into the *afferent* jejunal loop, and is commonest with *left-to-right* gastro-jejunal anastomosis (Ogilvie, 1947; Mimpriss and Birt, 1948) (Fig. 3, *1c*). For this reason such operations are now seldom performed. Pannett (1950), however, is a firm advocate of this technique for the very reason that it does allow food to mix with the bile and pancreatic juices before passing down the intestine. These symptoms are much less common after gastrectomy with a right-to-left anastomosis (Fig. 3, *1a, 1b*), and hardly ever occur when a valve (Fig. 3, *1d*) is made. When symptoms are troublesome entero-anastomosis or jejunoplasty should give relief.

5. Gastric Distension (Fig. 5, d)

Some patients who experience symptoms after meals, especially fullness and nausea, are shown by barium-meal examination to have gastric distension, a small stoma, and *slow* emptying of the stump. Their symptoms may be relieved by vomiting *food*. In these cases we believe the symptoms are produced by gastric distension. If small, well-chewed, semi-solid meals do not bring relief it is necessary to enlarge the stoma.

Other patients who have the same symptoms are wholly or partly relieved by bringing up wind. Aerophagy must be corrected, and carminatives, such as peppermint, often bring prompt relief.

The Hypoglycaemic (Late Post-prandial) Syndrome

One of the consequences of rapid emptying of the stomach is that glucose, which is absorbed from the jejunum, reaches the blood stream much more rapidly than normal (Fig. 7). Hyperglycaemia may occur before

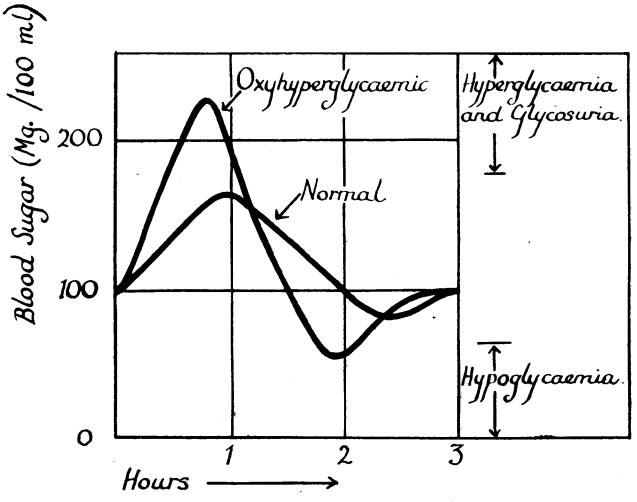

FIG. 7.—Glucose-tolerance curves—normal and post-gastrectomy (oxyhyperglycaemic).

enough insulin can be mobilized to bring about its storage in the liver. The hyperglycaemia does not cause any symptoms, but may produce glycosuria, which is of no serious significance. On the other hand, it may stimulate an overproduction of insulin, so that the blood sugar falls rapidly to *hypo*glycaemic levels. The hypoglycaemic phase *may* be accompanied by symptoms, which occur two to three hours after a meal—that is, late post-prandial (Fig. 4). A blood-sugar curve of this type is referred to as a "lag storage curve" by MacLean (1927) and as an "oxyhyperglycaemic curve" by Lawrence (1936, 1947) (ὀξύς = sharp). Curves of this type are found in about one-third of gastrectomized patients, but in only about 15% of these does the hypoglycaemic syndrome occur. It must be emphasized that the blood-sugar levels are in no way responsible for the *early* post-prandial syndromes. The whole subject is admirably reviewed by Muir (1949).

Deficiency Syndromes

Loss of Weight

It is only since gastric resections have become extensive—that is, subtotal and total—and since patients have been accepted earlier for operation that loss of weight after gastrectomy has become a serious problem. Gordon-Taylor *et al.* (1929), for example, who removed up to two-thirds of the stomach, found an average *increase* of 2 stones (12.7 kg.) in weight in their series. Recently various authors—for example, Muir, 1949—have touched on the problem, but we have seen no careful analysis of the details or of the mechanisms involved.

It is important to distinguish between patients who are in a good state of nutrition at the time of operation and those (usually with pyloric stenosis or carcinoma) who have lost weight before operation. The accompanying Table shows an analysis of the pre-illness, pre-operative, and final post-operative weights of 100 cases of subtotal gastrectomy, 5 of total gastrectomy, and 8 of oesophago-gastrectomy. The weight tends to become stabilized a year or so after operation, and the term "final" refers to the weight at the end of one or more years.

The significant facts are as follows. If all the subtotal gastrectomies are considered together, there was an average loss of 17 lb. (7.7 kg.) in weight before and a *gain* of 3 lb. (1.4 kg.) after operation. If only those who had maintained their weight up to the time of operation are considered (and they account for 58%), there was an average *loss* of 7 lb. (3.2 kg.) after operation. One-fifth of these lost more than 14 lb. (6.4 kg.). The 42% who had lost more than 14 lb. before operation *gained* an average of 18 lb. (8.2 kg.) *after* operation, but were still 19 lb. (8.6 kg.) below their pre-illness weight. The numbers of total gastrectomies and oesophago-gastrectomies are small, but the figures are interesting. The total gastrectomy cases lost, on the average, 14 lb. before and another 16 lb. (7.3 kg.) after operation. The oesophago-gastrectomy cases (all of whom had organic dysphagia) lost 31 lb. (14 kg.) before and only 5 lb. (2.3 kg.) after operation.

We believe that two important factors contribute to the loss of weight: a diminished calorie intake and impaired digestion and absorption of food.

Diminished Calorie Intake.—A careful analysis of the actual food intake of patients who are underweight shows that, although they are taking a varied diet, including first-class protein, fat, vitamins, etc., the total quantity is nearly always insufficient to supply their energy requirements. As a result they lose weight until they become stabilized at a lower level. Some patients take barely enough to satisfy normal basal requirements, but seem to manage very well without any real disability. The usual cause of the reduced intake is the presence of symptoms after food, which limit the size of the meal. Actual vomiting of food is a rare contributory factor.

Impaired Digestion and Absorption.—After gastrectomy the small intestine has to perform the normal functions of the stomach—that is, to mix, dilute, churn, and liquefy the food as it passes down—so that there is a much shorter length available for the normal intestinal phase of digestion and absorption. In addition, after operations with a gastro-jejunal anastomosis, the bile and pancreatic juice tend to follow food down the intestine instead of being intimately mixed with it. The subject has not, so far as we know, been thoroughly investigated, but there have been isolated reports of increased fat and poorly digested meat fibres and connective tissue in the faeces of patients who have undergone gastric operations (Spriggs and Marxer, 1922; Gordon-Taylor *et al.*, 1929; Rekers *et al.*, 1943; Wollaeger *et al.*, 1946; Muir, 1949). Welbourn and Glazebrook (1951) have found steatorrhoea in two patients who were markedly underweight after gastrectomy. One had 76% and the other 74% absorption. Hexamethonium bromide before each meal increased their absorption to 87% and 89% respectively and both put on weight, the second increasing by 20 lb. (9.1 kg.) in five weeks. Hexamethonium bromide, as we have already mentioned, reduces the rate of passage through the small bowel. This result therefore indicates that intestinal hurry is an important factor in the production of steatorrhoea and consequent loss of weight in these cases. Brain (1950a, 1950b) has studied steatorrhoea after total gastrectomy. He finds it very frequently, and has shown that absorption can be greatly increased by giving six meals a day, each of which contains a quantity of fat.

In most cases the steatorrhoea is symptomless and can be discovered only by a careful fat balance. After gastrectomy many patients have much freer bowel actions than previously, and the motions are often paler. The colour change is usually due to an alteration in the pigment and not to an increase in fat content.

Management.—Patients should be warned before operation that they may fail to gain weight and may even lose a little. This is important, as it obviates anxiety. Then, when serious causes of loss of weight have been excluded, reassurance is all that is usually necessary. Attention should be given to the post-prandial syndromes which may be limiting the food intake, and the patient should be encouraged to take six fat-containing meals in the day. Hexamethonium bromide seems worthy of further trial in patients with steatorrhoea.

Iron-deficiency Anaemia

Iron salts are absorbed mainly from the upper small intestine after being dissolved and dissociated by HCl in the stomach. A *p*H of 5 or less is necessary for dissociation, and after gastrectomy, when a *p*H of less than 6 is rarely found, absorption of iron is impaired. The increased rate of passage through the jejunum may also interfere with absorption. For these reasons iron-

Table Giving Weight Analysis of 100 Cases of Subtotal Gastrectomy, 5 Cases of Total Gastrectomy, and 8 Cases of Oesophago-gastrectomy

Type of Gastrectomy	No. of Cases	Average Weight			Final Post-operation Weight in Relation to Pre-operation Weight		
		Pre-illness	Pre-operation	Final Post-operation	Maintained or Gained	Lost up to 14 lb. (6·4 kg.)	Lost over 14 lb. (6·4 kg.)
		lb. kg.	lb. kg.	lb. kg.	No. (%)	No. (%)	No. (%)
Subtotal gastrectomy: all cases	100	148 (67·1)	131 (59·4)	134 (60·8)	59 (59)	28 (28)	13 (13)
Within 14 lb. of pre-illness weight at operation	58	146 (66·2)	144 (65·3)	137 (62·1)	21 (36)	24 (41)	13 (22)
Lost more than 14 lb. before operation	42	151 (68·5)	114 (51·7)	132 (59·9)	38 (90)	4 (10)	0 (0)
Total gastrectomy	5	142 (64·4)	128 (58·1)	112 (50·8)	1	2	2
Oesophago-gastrectomy	8	157 (71·2)	126 (57·2)	121 (54·9)	3	2	3

deficiency anaemia—that is, anaemia of the hypochromic microcytic type—is not uncommon after gastrectomy, and Watson (1947), reviewing the literature, concluded that the incidence was about 10%. The following points are important.

(*a*) It is necessary to define the standards which are regarded as normal. Whitby and Britton (1950) give 14 g. per 100 ml. (=94% Haldane) as the lower limit of normal for the blood haemoglobin in a man, and 12 g. per 100 ml. (=81% Haldane) as the lower limit in a woman. The appearance of the blood film is of more value than a red cell count in routine work.

(*b*) Although some people, anaemic as a result of haemorrhage, steadily improve after gastrectomy, many tend to remain anaemic or even to deteriorate. It is most important to make the blood normal with iron and blood transfusion before operation, and again before the patient leaves hospital.

(*c*) Women are more likely than men to become anaemic, because of the menstrual loss. Men lose about 5 mg. of iron a day in the bile pigments, and women lose in addition an average of about 10 mg. a day from the uterus. Since a normal diet contains only about 15 mg. of iron, it is important to advise plenty of foods rich in iron (meat, liver, eggs, etc.).

(*d*) The anaemia is rarely severe enough to cause symptoms, but the blood should always be examined if a patient is not doing as well as he should. Vague symptoms such as paraesthesiae and loss of energy are sometimes due to anaemia, and disappear when it is corrected.

(*e*) The modern radical operations cause more anaemia than the older ones because there is a greater reduction in acid and more tendency to intestinal hurry. In our own series we have found anaemia in about 15% of men and 30% of women at periods from 1 to 10 years after subtotal gastrectomy. In some cases there is a steady deterioration over the years if the condition is not treated.

(*f*) In most cases the anaemia responds readily to iron by mouth. Ferrous sulphate, 6 gr. (0.4 g.) three times a day, is usually adequate. Intravenous iron is effective in the rare cases that do not respond.

Macrocytic Anaemia

At one time pernicious anaemia was regarded as a real danger after gastrectomy, and it was a common practice to give liver injections as a routine. In fact, very few cases have been reported. We have, however, seen two cases after subtotal and one case after total gastrectomy. Macrocytosis occurs commonly after total gastrectomy (Brain, 1950b), but its significance is not clear. Our patient with pernicious anaemia after total gastrectomy took four years to develop a megaloblastic marrow, although he had had macrocytosis for some time before. It may be that, even if all the intrinsic factor is removed, an appreciable time is necessary for the full picture of pernicious anaemia to develop. On no account should liver or vitamin B_{12} be given until the patient has had a full haematological investigation.

Vitamin-B Deficiencies

Very little attention has been paid to these syndromes, but we have seen some evidence of avitaminosis B in about 10% of our own series of subtotal gastrectomies (Welbourn, Hughes, and Wells, 1951). In most cases the patients are taking adequate quantities of the B vitamins in their food, even though the total calorie intake may be low. Achlorhydria is probably the most important cause, though intestinal hurry may play a part. Some of the B vitamins are stable only in an acid medium and are partly destroyed at the neutral reaction of the stomach and small intestine after gastrectomy. The bacteria, which grow all the way up the alimentary tract, require the B vitamins and may take appreciable quantities for their own use, thus depriving their host (Frazer, 1949b). The common syndromes are:

Ariboflavinosis.—The features of this condition are cheilosis and angular stomatitis, and superficial glossitis. They may occur alone or in association with other deficiency states.

Incipient Peripheral Neuritis.—This is due to aneurin deficiency, and is seen quite often. The features are: paraesthesiae (pins-and-needles, etc.) in the hands and feet; muscle cramps; and lack of energy. Sensation and tendon reflexes may be quite normal. It is important to exclude other conditions, such as anaemia, which may cause similar symptoms. The lack of energy usually accompanies the other symptoms, but does not always respond to specific therapy.

Severe Deficiency States.—Rarely, severe peripheral neuritis with oedema develops, but it should not do so if it is treated in the incipient stage. Pellagra and Wernicke's encephalopathy are other rare vitamin-B deficiencies which we have seen after gastrectomy.

The first two syndromes should be treated with vitamin-B complex—for example, capsulae vitaminorum (*N.F.*) 4 t.d.s. They usually clear up rapidly, but resistant cases may require larger doses of the specific vitamins as well. Some source of the whole B complex should be taken regularly, and after gastrectomy everyone would be well advised to do the same as a prophylactic measure.

Conclusion

It must be stressed that, although a considerable proportion of patients do have a disordered economy after gastrectomy for peptic ulcer, nearly all of them are well satisfied with the results of their operations. It is clear that most of the disorders which occur result from, or are aggravated by, the modern radical operations which are necessary to prevent recurrence of ulceration, and we await with great interest the results of less radical types of gastrectomy combined with vagotomy.

So long as surgery is essential for the relief of peptic ulceration and carcinoma it is important to study the effects of the operations on the normal physiology. Attention must be directed to stasis and reflux, to alterations in *p*H, and especially to exaggerated peristalsis and jejunal hurry.

Summary

The normal physiology of the stomach is described, and its importance in preparing food for the small intestine is emphasized.

The different types of gastrectomy are enumerated.

The physiological effects of these operations are described under four headings: rapid emptying of the gastric remnant; stasis and reflux; reduction of acid; and removal of intrinsic factor.

These effects give rise to two groups of post-gastrectomy syndromes: post-prandial syndromes and deficiency syndromes. These are described and their management is discussed.

[Since this article was submitted for publication we have found that the operation of jejunoplasty, which we have used for afferent loop stasis and bilious regurgitation, was previously described by C. L. Hoag and J. B. Saunders (*Arch. Surg.*, 1941, **42**, 259).]

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Static electricity is a well-known risk in the operating theatre, where its sudden discharge as a spark may cause an ether explosion. It is a risk also in many industrial processes, wherever paper, plastic, rubber, or other insulating substance is passed at high speed between rollers or some other surface on which there is friction—for example, in both flat-bed and rotary printing presses, and in the rubber-coating of fabrics. A spark may fire an inflammable solvent, but apart from this the static may interfere with the manufacturing process, making sheets of paper fly apart or stick together instead of stacking evenly, and causing powders to clump and stick to surfaces. One of the commonest methods of preventing static is to ionize the neighbouring air so that it becomes electrically conducting and the charge can leak away as it is formed, and radioactive substances are used to do this, most commonly radium itself. This creates a danger to the workers, however, from possible over-exposure to alpha, beta, and gamma radiation and inhalation of radon, the radioactive gas liberated during radium decay. The *Monthly Review* of the New York State Department of Labor for November and December last discusses this as an industrial hygiene and safety problem. It advocates study of each individual instance of the use of a radioactive static eliminator to make sure that it is in the least dangerous position as judged by measuring instruments and by film badges worn by the workers. Gloves should be worn when cleaning the machine, or those parts of it containing the radioactive source. Ordinary ventilation should blow away the radon produced. Periodical medical examination, with a blood count, is also advised, and the skin of the fingers should be examined for flattening of the ridges, which is one of the signs of excessive radiation.

EFFECT OF STREPTOMYCIN ON VESTIBULAR FUNCTION

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During the early period of the use of streptomycin in pulmonary tuberculosis, when 2 g. was given daily, disturbances of vestibular function were frequent. Indeed, this threatened to limit its usefulness almost as seriously as did the emergence of bacterial drug resistance. After it had been shown that the daily dose could be halved without reducing the therapeutic effect, the importance of this toxic manifestation diminished. But evidence of vestibular dysfunction is still found in about 15% of patients being treated with the usual 1-g. dose daily, and the toxic action of streptomycin on the vestibular apparatus therefore remains of some importance to the clinician.

The following observations are based on a study of 76 patients with pulmonary tuberculosis receiving streptomycin treatment at the Brompton Hospital during the Medical Research Council clinical trials of this antibiotic.

All patients were treated for at least 12 weeks and some for as long as 32 weeks. Sixty-five were given streptomycin alone, 23 receiving 2 g. daily in doses of 0.5 g. six-hourly, 13 receiving 1 g. daily in doses of 0.25 g. six-hourly, and 19 having 1 g. daily in a single injection. Four patients received 2 g. daily in doses of 0.5 g six-hourly during alternate weeks and 6 were given the same dosage during alternate months. Eleven patients were treated with 1 g. streptomycin daily in a single injection and from 5 to 20 g. of the sodium salt of *para*-aminosalicylic acid (P.A.S.) daily. The patients were examined at weekly intervals and more frequently if toxic symptoms were present. Tests of vestibular function were carried out in 50 patients.

Giddiness

In several patients the earliest symptom of vestibular dysfunction was a complaint of mistiness of vision. This was noticed mainly during the evening when changing quickly from near to distant vision. There might also be difficulty in reading, probably due to incoordination of eye movements. Some patients had no other evidence of toxicity, the disturbance of vision disappearing after a few days. In others it was followed by giddiness. This was only rarely a definite sense of rotation; more commonly there was merely a sense of

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