the aim is to provide a vocational training flexible enough to meet the requirements of a choice of careers, including general practice. The first year after registration would be in general training in a senior house officer (S.H.O.) grade. After that two to four years would be spent in a medical officer (M.O.) grade (replacing the present registrar grade), and this would lead either to a chief assistant or assistant post or to a career outside the hospital service. Tenure of chief assistant and medical officer posts would be subject to review after one year. The value of these new grades would greatly depend on the effectiveness of the "assessments" and "reviews" which are essential to planning, and much of the responsibility for these will rest on the proposed regional advisory committees.

What the report has to say about the medical assistant grade is likely to arouse controversy. There is a need for the grade, and all will endorse the panel's belief that it should be made more attractive. Status and pay are at the heart of the matter. If the proposals for limiting the tenure of medical officer and chief assistant posts are adopted there will be no lack of potential medical assistants. But will they be willing recruits, and will they be content to soldier on in the grade? It offers a good opening for general practitioners and others to work part time in hospitals, but here again problems of comparative pay may well arise.

The panel's proposals are a brave attempt to right current wrongs in hospital medical staffing, especially junior staffing. Its report is put forward for debate, and it is to be hoped that it receives the constructive comment that it merits.

Screening Tests for Phenylketonuria

The detection and treatment of phenylketonuria early in infancy can prevent severe and permanent intellectual impairment. Diagnosis by mass screening is now possible.

Phenylketonuria is inherited as a recessive disease. It is due to a deficiency of the enzyme phenylalanine hydroxylase, which normally converts phenylalanine into tyrosine. When this enzyme is deficient, abnormally high levels of phenylalanine accumulate in the blood and other tissue fluids, and it is the excess of phenylalanine which is held to be responsible, directly or indirectly, for the progressive cerebral damage in the untreated patient. Some of the phenylalanine is excreted unchanged in the urine and the remainder as a variety of breakdown products. Among them is phenylpyruvic acid, and this causes the characteristic greenish-blue colour reaction when a few drops of a solution of ferric chloride are added to the urine.

The accepted view is that the infant with phenylketonuria has a normal brain at birth and that, if the serum level of phenylalanine can be maintained within normal, or perhaps very slightly above normal, limits by a diet low in phenylalanine, intellectual development will proceed normally. The evidence for the efficacy of dietary treatment has been challenged by S. P. Bessman and by H. G. Birch and J. Tizard, but their arguments have recently been contested. The first step in the application of knowledge about phenylketonuria was the early detection and treatment of affected infants born to parents with an older child with phenylketonuria. The next step was the attempt to detect all affected infants in the population. Surveys have shown that phenylketonuria in communities of predominantly European stock is commoner than was originally supposed and probably has an incidence of 1 per 10,000 population. This makes mass screening worthwhile.

The Phenistix test paper, a modification of the ferric chloride test, has been generally used until recently, but it gives a relatively low incidence of positive tests. Cases of phenylketonuria have been missed either because of false negative results or because of the difficulty in obtaining for the test a sufficiently fresh specimen of urine or a recently wetted napkin. Moreover, the Phenistix test is usually done at the age of 6 weeks, when contact with some of the babies may have been lost.

A number of alternative methods of testing are available, the most widely used being the Guthrie test. A drop of capillary blood is dried on special filter paper and is taken or posted to a central laboratory for assay of the level of phenylalanine. The Guthrie test has the advantages that it can be done early in life and that a drop of blood is often obtained with less difficulty from an infant than a fresh specimen of urine. In addition the blood sample can be tested for other metabolic disorders such as galactosaemia.

At page 674 of the B.M.J. this week a group of paediatricians and medical officers of health in South-east Scotland present their experience with the Guthrie test as a screening procedure in a population of 1½ million. By doing the test on the 6th day of life it was considered that a positive test would be missed once in every five years in the whole of Scotland, and that an attempt to improve on this by using a second later test would probably defeat its object owing to decreased co-operation from all concerned. This paper shows that the necessary organization and co-operation between the various branches of the National Health Service can be achieved.

It is necessary to confirm the diagnosis in those infants with a positive test. This should be done in a hospital where accurate determination of the serum levels of phenylalanine can be made and where a diet low in phenylalanine can be arranged when required. Experience has shown that the serum phenylalanine is slightly and transiently raised in 1 out of 4 premature and 1 out of 500 full-term infants. In these cases the level will be found to return to normal within a few days without dietary restriction. More difficult is the problem of "atypical phenylketonuria," described by L. I. Woolf and his colleagues in 1961. In this condition, in

10 Berry, H. K., and Wright, S., J. Pediat., 1967, 70, 142.
12 Castells, S., and Brandt, I. K., J. Pediat., 1968, 72, 34.
contrast to "true" phenylketonuria, relatively large amounts of phenylalanine can be tolerated without abnormally high levels appearing in the serum, yet the infants also require dietetic treatment.

The duration of treatment of phenylketonuria is a matter of great importance. There is general agreement that unless it is started before the age of 6 months (preferably under 3 months) the results will be poor. It is more difficult to know at what age the diet can be relaxed or stopped. Certainly a strict diet should be maintained well into the school age, and relaxation of control should be a planned operation, with frequent psychometric testing to detect any intellectual deterioration as soon as possible.

Enough evidence has now accumulated to justify replacing the Phenistix test by the Guthrie or a similar test. One difficulty which must be faced is that infants born outside the N.H.S. organization may escape testing unless special provision is made. Recently the State of Connecticut made the testing of infants a legal requirement.12

Predicting the Dumper

Removal, bypass, or destruction of the pylorus leads to sequelae which vary greatly in severity even in patients undergoing the same operation. One of the chief complaints (early postcibal dumping) consists of weakness, dizziness, sweating, and a feeling of fullness beginning 10 to 20 minutes after food and lasting up to one hour. Some patients are forced to rest after eating and a minority are severely disabled.

The normal intact stomach is an osmotic shield. Isosmolarity of the gastric contents with the body fluids is established gradually while gastric emptying proceeds in a controlled fashion. If, as a result of operation, pyloric regulation is lost, large volumes of hyperosmotic nutrients are passed rapidly into the small bowel. Owing to the large areas of mucosal surface, iso-osmolarity is established by a rapid flow of fluid from the plasma into the lumen of the gut; this rapid expansion of the gut contents distends the bowel.

The symptoms of postcibal dumping were described in 1913,1 and since then many investigations have been reported, sometimes with conflicting results. It would appear that symptoms result from the individual patient's response to three main factors—firstly, distension of the small bowel; secondly, reduction in plasma volume; and, thirdly, release of vasoactive substances into the circulation. The symptoms were initially attributed to jejunal distension1 and were in part reproduced by inflating balloons in the jejunum.2 Increased motility of the small bowel was later observed,3,4 and stretching of the unsupported gastric remnant has also been shown to produce symptoms.5-6

A reduction in plasma volume occurs, after eating, in most patients who have undergone partial gastrectomy7 whether they have severe postcibal symptoms or not. Similar changes are observed in normal persons when hypertonic glucose is instilled into the duodenum.8 This reduction in plasma volume almost certainly results from the passage of fluid into the gut lumen to establish iso-osmolarity. Some observers reported that the rapid infusion of plasma expanders controlled postcibal symptoms,9 but others could not confirm this.10 Cuffing of the legs to produce a fall in effective plasma volume has been recommended as a preoperative method of recognizing those patients who are intolerant of reduction of the plasma volume and are therefore liable to develop severe postcibal symptoms after gastrectomy.11

The rapid entry of food into the small bowel may also impair circulatory homeostatic reflexes by causing inappropriate vasodilatation in the splanchic area,12 forearm,13 and kidne14.

Although postcibal hypoglycaemia15 and hypokalaemia15 may be observed in patients after gastrectomy, the changes do not coincide with dumping symptoms. Delay in the absorption of carbohydrates leading to a prolonged osmotic "drag" in the gut has been postulated.16 Though symptomatic relief was reported when insulin was given before a meal, there is no good evidence that insulin influences carbohydrate absorption, nor has any abnormality in insulin response to hyperglycaemia been found in patients with severe dumping.17 It is possible that this symptomatic relief was a placebo effect, for it is now apparent that the assessment of any innovation in clinical treatment for dumping requires a rigorously controlled clinical trial, since these patients will often improve when a sympathetic clinician takes interest in them.

The introduction of hypertonic glucose into the jejunum of animals causes a release of vasoactive substances,18 but no specific circulating agent has been identified in the blood of patients during severe dumping attacks.

Recently a new approach to recognizing patients who are likely to experience dumping after operation has been reported.19 The standard threshold of alveolar carbon dioxide tension (sCO2) required to stimulate pulmonary ventilation was determined by studying patients who were rebreathing carbon dioxide in increasing concentrations up to 5%. Fifty-five patients were examined before and one year after gastric surgery. In those patients with severe postcibal symptoms the mean sCO2 was significantly lower than the normal 38.5 mm. Hg CO2. The authors postulate that the low threshold value represents an abnormally sensitive autonomic nervous system, and they state that there is a correlation between sCO2 and a series of psychological tests performed on this group of patients—details of this study are to be published. The test is not applicable to patients with emphysaemia because of poor gas mixing. Though all patients with severe dumping had a low threshold, so also did a minority of those with no postcibal symptoms.

The test requires further experience and evaluation to determine, for example, whether those patients who do badly after vagotomy and drainage also show a low threshold. But it may provide a little more information to guide the clinician in deciding whether or not a patient should undergo elective

2 Machella, T. E., Gastroenterology, 1950, 14, 237.