viscus, innervated by the corresponding nerve roots, which results in inflammatory lesions and spasm in the case of the hollow organs. This suggests that, in addition to the sympathetic and parasympathetic pathways, there exists a third nerve pathway by which visceral activity may be altered.

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In a booklet entitled *Health Services in Britain* (H.M.S.O., 3s. net) the Central Office of Information has added another useful publication to its series of "Reference Pamphlets." Starting with water supply and sewerage, the booklet proceeds to describe the other public health services, the National Health Service, and industrial health. There are additional sections on the health services in Scotland and Northern Ireland, on the Medical Research Council, and on professional training in Britain; a bibliography of Government publications; and a useful list of the names and addresses of health departments and organizations. Some statistics are given of the National Health Service. About 97% of the population of Great Britain uses the Service; 98% of general practitioners, the "great majority" of specialists, 94% of dentists, and almost all chemists are taking part in it. The cost of the Service amounts to about 3½% of the total national resources, and four-fifths of the gross cost (£570m.) falls on the Exchequer to be met from general taxation.

INFLUENCE OF HYDROCHLORIC ACID ON GASTRIC SECRETION AND EMPTYING IN PATIENTS WITH DUODENAL ULCER

BY

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There are at least three possible ways of accounting for gastric hyperfunction in patients with duodenal ulcer. They are: (1) an increased number of cells which secrete acid (Meyers, 1948; Guiss and Stewart, 1948; Hunt, 1950; Cox, 1952; Card, 1952; Hunt and Kay, 1954); (2) an increased vagal drive to the gastric mucosa (Dragstedt, 1956); and (3) a failure of the duodenal mechanisms which inhibit gastric activity; this hypothesis embraces both gastric secretion and emptying (Shay, 1944). The present paper is concerned with the last of these possibilities.

Methods

The main purpose of the present experiments was to examine the influence on gastric secretion of low concentrations of acid added to test meals. Such acid might be expected to have three effects: it should stimulate duodenal receptors which slow emptying and thus prolong gastric distension by the meal. Since gastric distension is a stimulus to secretion in man (Hunt and Macdonald, 1952; Macdonald and Spurrell, 1953) such slowing of emptying should increase gastric secretion; on the other hand, acid should depress secretion via the duodenal inhibitory mechanism or possibly by inhibiting the release of gastrin (Woodward, Lyon, Landor, and Dragstedt, 1954). Thus whether added acid increases or decreases gastric secretion will depend upon the balance between the increased stimulus of distension and the inhibition of secretion from the duodenum. It has been found that in most normal subjects adding sucrose to test meals delays emptying and reduces secretion, but in two the addition of sucrose caused an increase of secretion. On further examination it was found that in these two subjects increasing gastric distension produced an unduly large increase in gastric secretion (Hunt, 1954).

For the present experiments, which were mainly concerned with inhibition of secretion, it was decided to use a solution of glucose as the control meal to which acid could be added as a variable. By slowing the emptying of the meals with glucose it was hoped to minimize the effect of the added acid on emptying. A further advantage of adding glucose to both meals is that it minimizes (Bandes, Hollander, and Glickstein, 1940) the absorption of water from the stomach which occurs to some extent in man (Hunt, 1949), so that phenol red added to the meal may be used as a reliable marker to determine the volume of original meal remaining.

The methods used have been described (Hunt, 1954). In essence the technique involves giving a test meal containing a dye which is not absorbed in the stomach. After 30 minutes the gastric contents are recovered, and from the amount of dye remaining the volume of the original test meal in the stomach at the time of recovery may be calculated. Further, the volume of the gastric contents which has passed into the duodenum may be assessed, together with the total amounts of acid, chloride, and pepsin secreted. From the amounts of acid and chloride secreted the outputs of hypothetical parietal component and non-parietal secretions may be found. Twenty-seven volunteer students were studied and 18 tests were made in 16 patients with a diagnosis of duodenal ulcer without pyloric stenosis. The patients were under treatment in the medical and surgical wards, and the

majority were not having pain during the period of the tests, nor did any of them have pain during their test. The control meal was 750 ml. of solution containing 100 g. of glucose per litre; the experimental meal was similar except that 20 mEq HCl per litre of meal was added, a level of acidity which may be exceeded for 12 hours out of 24 in the gastric contents of normal subjects (Atkinson and Henley, 1955). By giving the meal down the tube and by not taking up the tube during the digestive period the slowing of emptying resulting from the stimulation by acid of receptors lying between the lips and the cardia was avoided (Hunt, 1956). Saliva was aspirated during the tests.

Results

Gastric Emptying.—Table I shows that no marked difference between the rates of gastric emptying in normal subjects and patients with duodenal ulcer was found. Moreover, the slowing of the emptying of the meal by added acid was equal in the two groups.

TABLE I.—Influence of 20 mEq HCl/litre of Test Meal on Gastric Emptying of a Solution of Glucose (100 g./litre)

	27 Normal Subjects		18 Tests in 16 Patients with Duodenal Ulcer	
	Glucose Solution	Mean Change with Acid	Glucose Solution	Mean Change with Acid
Mean volume of meal remaining in stomach at 30 min. (ml.)	489	+36.8	439	+37.7
S.E. of mean	±9	±9	±23	±13
Mean volume of gastric contents at 30 min. (ml.)	566	+10.8	542	+23
S.E. of mean	±9	±9	±21	±9

Gastric Secretion.—Table II shows that the patients with duodenal ulcer as a group secreted twice as much acid, chloride, and pepsin as did the normal subjects. Adding acid to the test meal reduced the secretion of acid in the

TABLE II.—Influence of 20 mEq HCl/litre of Test Meal on Gastric Secretion in Response to a Solution of Glucose (100 g./litre). All Data Refer to a Digestive Period of 30 Minutes

	27 Normal Subjects		18 Tests in 16 Patients with Duodenal Ulcer	
	Glucose Solution	Mean Change with Acid	Glucose Solution	Mean Change with Acid
Mean amount of acid (mEq)	5.1	-1.1	12.5	-0.7
S.E. of mean	±0.6	±0.3	±1.7	±0.8
Mean amount of chloride (mEq)	8.4	+0.1	18.5	-0.7
S.E. of mean	±0.7	..	±2.0	±1.0
Mean amount of pepsin (units)	6,300	No change*	11,300	No change*
S.E. of mean	±870		±1,900	
Mean volume parietal component (ml.)	36.7	-5.1	87.8	-3.9
S.E. of mean	±3.8	±2.1	±11.7	±4.1
Mean volume non-parietal secretions (ml.)	18.5	+6.5	35.0	-1.0
S.E. of mean	±1.8	±2.1	±3.4	..

* Because of very large changes occurring in some subjects the effect of acid was determined by comparisons of amount secreted with added acid
log amount secreted with control meal.

This gives an equal weight to halving and doubling the outputs of pepsin in each subject.

normal subjects by an average of one-fifth but produced no other significant mean change in the measurements of these quantities in either group.

Because the chloride of the gastric secretion has its origin in at least two types of cell it is often helpful to describe the gastric secretion of electrolytes in terms of the hypothetical acid or parietal component and the non-parietal secretions (Hollander, 1938; Fisher and Hunt, 1950), although with some reservations (Hunt, 1957). The data expressed in these terms show that the hypersecretion in the patients with duodenal ulcers involves not only the acid-secreting parietal cells but also the sources of the non-

parietal secretions. The addition of acid to the test meal decreased the mean secretion of parietal component and increased the output of non-parietal secretions in the normal subjects, but produced no significant mean changes in the patients with duodenal ulcers. It will be seen from Fig. 1,

FIG. 1.—Relation between changes in outputs of acid and chloride on adding 20 mEq H⁺/l. of test meal (glucose 100 g./l.).

in which the change in the output of acid has been plotted against the change in the output of chloride for each subject, that the changes in the outputs of these two electrolytes on adding acid were considerable and correlated with each other in much the same way in both groups. This relationship is masked when only the mean data are considered.

Discussion

Gastric Emptying

It has so far proved impossible in this laboratory, using various test meals, to provide any convincing evidence that as a group patients with duodenal ulcers have stomachs which empty more rapidly than normal subjects. This could be the result of our normal subjects being younger or more

FIG. 2.—Relation between slowing of emptying of meal in response to 20 mEq HCl/litre of test meal (glucose 100 g./l.) and amount of acid secreted in response to meal in 30 minutes.

physically active than the patients, but it does not seem a probable explanation, since these factors have not masked the hypersecretion of acid and pepsin which is associated with duodenal ulcer.

In the data based on these experiments the magnitude of the influence of added acid on gastric emptying depends upon the gastric secretory response to the control meal, a finding which confirms that of Shay and Gershon-Cohen (1934).

In Fig. 2 the change in the volume of the meal remaining after adding acid to the test meal has been plotted against the secretion of acid in response to the control meal for each subject. For the normal subjects there is a tendency, significant at the 1 in 20 level, for the added acid to be more effective in slowing emptying in those subjects who secreted little acid in response to the control meal. Moreover, the subjects were unselected, whereas Shay and Gershon-Cohen (1934) chose subjects with hyperchlorhydria and achlorhydria in arriving at their conclusion. It is clear that the mechanism for the slowing of emptying responds effectively to such low concentrations of acid as 20 mEq/l. even under conditions chosen to minimize the influence of acid by adding glucose to the control meal.

The Shay (1944) hypothesis also suggests that in patients with duodenal ulcer the threshold of the mechanism which delays emptying is raised during the activity of the ulcer and returns to normal during remission. From what has been said above it is clear that comparisons of the reactivity of the mechanism which slows gastric emptying in response to acid must take into account the amount of acid secreted in response to the control meal. The line fitted to the data for normal subjects in Fig. 2 shows that a group of patients with a mean secretion of 6 mEq H^+ would not be expected to show any slowing of emptying by added acid. When the points are considered individually it may be seen that the normal subject who secreted 12.5 mEq H^+ showed no slowing of emptying by added acid. Since 12.5 mEq is the mean amount of acid secreted by the patients with duodenal ulcer, no slowing of emptying by added acid would be expected in these patients as a group if the mechanism were normal. In fact, the slowing of emptying was equal in the normal subjects and the patients with duodenal ulcer, a finding which implies that the mechanism which slows emptying in response to acid is overactive in patients with duodenal ulcers.

It is remarkable that the single-withdrawal method used here, which confirms that acid is more effective in slowing emptying in normal anacid subjects than it is in those secreting much acid, fails to confirm Shay's findings in patients with duodenal ulcers. It is possible that the discrepancy arises because Shay was principally recording "emptying time," which depends not only on the rate of emptying in the main part of the digestive period but also upon the final rapid emptying demonstrated by Hunt and Macdonald (1954), whereas the present data refer only to the first 30 minutes of the digestive period.

Gastric Secretion of Acid

The second part of the Shay hypothesis postulates that the gastric hypersecretion of patients with duodenal ulcer results from the failure of the duodenal inhibitory mechanism to slow gastric secretion as the gastric contents become more acid. In confirmation of this it will be seen from Table II that adding 20 mEq HCl/l. of test meal did depress the secretion of acid by about one-fifth in the normal subjects but that the mean depression of secretion in patients with duodenal ulcer was negligible.

A more detailed examination of the data on secretion, set out in Fig. 3, gives a somewhat different impression from that obtained by studying the mean data of Table II. It will be seen that for the normal subjects the greater the amount of acid secreted in response to the control meal the greater was the reduction in the secretory response when acid was added to the test meal (see also Hunt, Macdonald, and Spurrell, 1951). The data for 11 out of 18 tests in patients

with duodenal ulcer conform to this normal pattern, even though in one patient the secretion was five times greater than the mean for normal subjects. In 6 patients out of 16 with duodenal ulcers there was either a very small fall or an increase in secretion on adding acid to the test meal. No explanation of this increase in secretion is offered. It was not,

FIG. 3.—Relation between amount of acid secreted in response to a test meal of glucose solution (100 g./l.) and inhibition of secretion by acid added to test meal (20 mEq/l.). For explanation of A, B, and C, see text.

apparently, the result of increased distension by the meal containing acid, for acid did not especially delay emptying in these six patients.

One patient, noted in Fig. 3 by a special symbol, was tested three times. After six weeks of strict medical treatment his gastric secretion of acid was slightly augmented, although he felt extremely well at the end of this period and was entirely free from pain. In tests at the beginning (A) and end (B) of this six-weeks period, adding acid to the test meal increased secretion. Six months later he was readmitted to hospital and did not respond to strict medical treatment, although at this time his gastric secretion of acid was less and adding acid to the test meal did reduce secretion (C).

Gastric Secretion of Pepsin

The finding that the mean output of pepsin was unaffected in normal subjects when the mean output of acid fell was unexpected, for in previous experiments in which sucrose was used to inhibit secretion (Hunt *et al.*, 1951) the secretion of acid and pepsin fell together. Even when the present data for individual subjects were examined there was no correlation between the changes in the secretion of acid and pepsin. It is possible that the experimental conditions were responsible for concealing a change. Pepsin is partly inactivated at pH 5, so that with the control meal there was probably some inactivation of pepsin during the initial part of the digestive period until the pH fell. When acid was added to the meal this putative inactivation would not occur, so that a small fall in output of pepsin might be masked. In addition the secretion of pepsin may have been inhibited by the glucose in the control meal leaving very little possibility for further inhibition.

Significance of Failure of Inhibitory Mechanism

It is clear that the failure of the duodenal inhibitory mechanism is only an inconstant and minor factor in causing gastric hypersecretion in these patients with duodenal ulcer, for marked hypersecretion was found in some patients in whom the inhibitory mechanism was obviously effective. In addition it is known that, as compared with normal subjects, patients with duodenal ulcers have about 75% greater maximal parietal secretory capacity (Hunt and Kay, 1954) and about 75% more parietal cells (Cox, 1952), findings which can account for the majority of the gastric hypersecretion of acid in such patients.

Summary

The influence on gastric activity of adding 20 mEq HCl/litre of test meal containing 100 g. of glucose per litre has been investigated in 27 normal subjects and 16 patients with duodenal ulcer. The test meal contained a marker which allowed the volume of meal remaining and amounts of acid, chloride, and pepsin secreted to be calculated.

These experiments have not revealed any convincing evidence of abnormally rapid gastric emptying in patients with duodenal ulcers.

In normal subjects the less the secretion of acid in response to the control meal the more effective was the added acid in slowing emptying. The mean slowing of emptying by added acid in patients with duodenal ulcer was equal to that in the normal subjects. When the increased secretory response of the patients with duodenal ulcer is borne in mind this finding indicates that the mechanism which slows emptying in response to acid in patients with duodenal ulcer is more reactive than it is in normal subjects.

In response to the control meal the group of patients with duodenal ulcer had about twice the mean normal outputs of acid, chloride, and pepsin.

The added acid caused a mean fall in the output of acid in the normal subjects. In the patients with duodenal ulcer there were 10 out of 16 in whom the addition of acid reduced the output of acid. In the six others there was some augmentation of the output of acid.

The significance of these findings in the genesis of gastric hyperfunction in patients with duodenal ulcer is discussed.

It is a pleasure to thank many clinical colleagues for permission to investigate their patients with peptic ulcers, the patients themselves, and the students who have co-operated in these studies. I am also indebted to the Society of Apothecaries for the award of the Gilson Scholarship, out of which part of the expenses of this research was defrayed.

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"The Australian Department of Health has issued a permit for the importation of the European rabbit flea, which it is hoped will intensify the spread of myxomatosis. The fleas are being collected from Europe this month, the time of the year when they are most plentiful. They will be bred under strict quarantine conditions. Tests will be carried out to ensure that the flea will not be harmful to Australian native animals; experts in wild life will determine whether the flea represents a hazard to such animals as the koala and other marsupials or to certain domestic animals and birds."
 —*New Scientist*, March 14.

POST-PARTUM BLOOD PICTURE IN SINGAPORE

BY

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Previous investigations carried out by Wadsworth (1952a, 1952b, 1954) in Singapore showed that healthy people of all races living in a tropical climate had normal packed red-cell volumes, red-cell counts, and haemoglobin levels. The poorer classes of the population were not included in those investigations, but Millis (1954) found only one case of anaemia among 106 infants of the poorest and worst-fed section of the Singapore population, and it was of interest to find out whether there was any great degree of anaemia among those most likely to be affected—mothers in the lower-income groups.

Material and Methods

A total of 212 mothers were investigated in the free post-natal wards of the Kandang Kerbau Hospital, Singapore, where the inmates are all from the lower income groups. All patients had normal full-time deliveries without any haemorrhage before or after delivery, and in each case the puerperium was proceeding normally. They were unselected in respect of race, age, or number of pregnancies, and they represented a typical cross-section of non-paying patients in the hospital. There were 174 Chinese, 36 Indians, and 2 Malays. Their ages ranged from 16 to 46 years, and the number of previous pregnancies from 0 to 11. The climate of Singapore is uniformly warm and humid (Wadsworth, 1954); there is no malaria, very little hookworm, and less poverty than almost anywhere else in Asia and Africa.

All samples of blood were taken from an antecubital vein, using minimal stasis, on the second, third, or fourth day after delivery. The blood was placed in a dry stoppered bottle containing an ammonium-potassium oxalate mixture (Heller and Paul, 1934), and examination of the blood was made within two hours of collection. Haemoglobin estimations were made in all cases and haematocrit estimations in 192, there being slight haemolysis in 20 samples.

Estimations of the haemoglobin concentration were made with an M.R.C. photometer (King *et al.*, 1948a, 1948b). Pipettes used were of N.P.L. standard, and special care was taken to ensure that glass cells were scrupulously clean. Blood was thoroughly mixed before dilution, and errors were eliminated so far as possible by making frequent checks with the calibrated neutral glass standard. The packed red-cell volume was estimated by Wintrobe's method, and the samples were centrifuged for 45 minutes at 3,000 r.p.m.

Results

The mean haemoglobin level was 11.85 g./100 ml. (S.D. 1.85), and the mean packed red-cell volume was 35.3% (S.D. 4.77). The distribution of haemoglobin values is shown in Fig. 1 and the distribution of packed red-cell volumes in Fig. 2. Of the haemoglobin values observed, 95% were above 9 g./100 ml., and 86% of the packed red-cell volumes were over 30%. There were only a few isolated cases of moderately severe anaemia. The lowest levels of haemoglobin were 4 g./100 ml. in one patient, 5 g./100 ml. in two patients, and 6 g./100 ml. in one patient. Two patients had packed cell volumes of less than 20%.

No difference was observed between different age groups, nor was any difference found between primiparae and multiparae, and there was no correlation between the results observed and the number of pregnancies. There was, however, a gradual decrease in haemoglobin levels between the second,