percentage of deaths as the mice injected with phenolized saline.

Having excluded the possibility of the production of non-specific immunity by bacteria, we found in the second experiment that there was no substantial difference between the resistance of animals in the four groups to subsequent infection with influenza virus.

These experiments indicate that the simultaneous application of heterogeneous substances had no influence on the production of immunity against influenza virus.

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THE CONTROL OF GASTRIC HYPERACIDITY IN PEPTIC ULCER

BY

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In spite of all attempts to discover the aetiology of chronic peptic ulcer it must be admitted that the essential cause still eludes us. Analysis of the gastric juice frequently shows a high acidity, less commonly a moderate or low one, but a total absence of hydrochloric acid is quite unknown in this condition. While we have often concluded that hyperacidity is a cause of gastric ulcer, we have no evidence in favour of this at all. There is more evidence that the presence of HCl tends to continue the activity of an ulcer, once it has formed, and that healing is prevented. For this reason, chiefly, our main attempts at therapy in peptic ulcer have hitherto centred on the continuous neutralization of HCl by diet, drugs, and other methods. Quite probably the acid factor is not the sole cause of an ulcer's not healing, but at present the neutralization of HCl is our main therapeutic weapon. Furthermore, it is already clear that ulcers do heal even when HCl is present in the stomach during part of each day.

Diet as a means of neutralization of acid takes first place, and is given at hourly or at most two-hourly intervals; but it is already known that this cannot bring about continuous neutralization, and, moreover, during the hours of sleep feeding is impossible. Numerous drugs (alkalis and others) have therefore been used, but they are in many ways unsatisfactory. We want to find, if possible, foods or simple drugs that will ensure continuous neutralization throughout the twenty-four hours, or will keep the secretion of HCl permanently as low as possible.

The series of experiments now to be described were undertaken with a view to discovering the most suitable foods or simple drugs to maintain neutralization for as long as possible during the twenty-four hours. The substances under investigation were given two-hourly, and the stomach contents in all cases were examined repeatedly during a continuous period of twelve hours, the Ryle tube

being kept in position; samples were removed for hourly examination shortly before and one hour after feeds. All patients included in this investigation presented clinical and radiological evidence of peptic ulcer, and fractional test meals were first given in the usual way, with oatmeal gruel as the stimulant of gastric juice. Every test showed hyperacidity, and the curves obtained over two and a half hours were in general similar to those described by Ryle (1921).

Methods of Titration

The amounts of fasting juice and residue were measured, and all specimens were titrated for free and total acidities with decinormal sodium hydroxide, using methyl orange and phenolphthalein as indicators respectively. Results are expressed as units in terms of c.cm. N/10 NaOH required to neutralize 100 c.cm. of fluid. Only the free-acid readings are here recorded.

Group I (9 Cases)

For purposes of comparison feeds of milk, 10 oz. by mouth, were used in each case before other treatment. Parallel experiments with whole milk and citrated milk indicated no appreciable difference between the two in the resultant acid level, and as the thick clots of whole milk were found to block the tube the work was continued with citrated milk, to which alone results refer. Considering the great importance attached to milk in the treatment of peptic ulcer, one would expect to find that it caused a substantial reduction of acidity. But Nicol (1939) could not demonstrate this either by two-hourly or by hourly feeds; indeed, he recorded a higher acidity with milk two-hourly than with a diet consisting of three full meals. By quarter-hour readings he showed that a fall occurred immediately following each feed, but only of short duration: before the next feeding-time the level had risen to its previous height. The results of the present experiments are in complete agreement with those of Nicol. Surprisingly high levels were obtained: it was common to find figures of total acidity of 70 to 80 units, and few were below 30. The charts, with hourly readings, did not show the fluctuations indicated by Nicol, but the reference is the same—namely, that milk given at two-hourly intervals has little if any continuous antacid value.

Gelatin (feeds of 10 oz.) was next used, prepared according to the method of La Due (1939)—gelatin 1 part, lactose 3, water 32 parts. Andresen (1927, 1939) used this mixture with success in the treatment of haematemesis. I had already tried it as a secretory stimulus in the fractional test meals of 7 patients and had compared the results with those of gruel meals in the same cases. Apart from the fact that gelatin specimens were homogeneous and easier to titrate, no significant differences were noted in the acid curves; similarly, in the twelve-hour tests gelatin appeared to have approximately the same neutralizing power as milk, although the former is supposed to be a powerful stimulant of gastric juice.

The rational use of olive oil in the treatment of peptic ulcer is demonstrated in this series; both olive oil and peach-kernel oil were found to cause a substantial reduction of acidity, an effect already reported by Roberts (1931). In 4 cases three feeds of milk (10 oz.) were given two-hourly followed by three of oil (2 oz.) two-hourly; and in the second half of each test there occurred a fall of 20 to 60 units. The experiments were repeated several times in the reverse order, and a similar depression with oil was followed by a rise to over 40 units under the influence of milk. Chart I represents such curves, obtained from one patient. Peach-kernel oil and an emulsion of olive oil, as will be observed from Table I, are not quite so effective as undiluted olive oil. A criticism was offered that the low acid values were due to the nauseating taste of oil; but this was disproved by the fact that when oil was syringed down the Ryle tube the values were even lower than when it was given orally.

As a deliberate contrast tests were made with two substances which are supposed to be strongly acid-producing—bovril and strained meat stock. The latter has always been excluded from the dietary regimes of dyspeptic patients; but, as Table I shows, from the point of view of acidity this is an

unnecessary restriction, as the acid levels were in no case higher than those resulting from milk.

Table I indicates the average readings of the experiments in this group. It would appear that so far as two-hourly feedings are concerned the actual food given is of no account; milk, as a supposedly acid-lowering food, and meat soup, as acid-raising, are shown to be of little value in either respect. Olive oil, however, is a remarkable exception, being a real depressant of acid secretion, and further experiments were designed to estimate its effect in conjunction with milk.

CHART I.—Hourly free acidity of gastric contents over 12-hour periods in one case, with 2-hourly feeds of (a) milk 10 oz. and olive oil 2 oz., and (b) olive oil 2 oz. and milk 10 oz.

TABLE I.—Average Free Acidities of Gastric Contents over Periods of 12 Hours, in Tests with Various Foods given 2-hourly

Case	F.T.M. Max.	Milk 10 oz.	Gelatin Mixture 10 oz.	Bovril 1 dr. in Water 10 oz.	Strained Meat Stock 10 oz.	Olive Oil 2 oz.	Peach-kernel Oil 1 oz.	Olive Oil Emul. 2 oz.
1 D.U.	60	53	—	—	—	31	—	53
2 D.U.	104	53	—	—	—	36	40	—
3 G.U.	80	47	—	50	43	11	—	—
4 G.U.	30	26	—	12	22	—	—	—
5 D.U.	100	97	52	64	90	—	—	—
6 D.U.	85	67	47	—	—	32	—	—
7 J.U.	90	60	50	50	—	—	—	—
8 G.U.	24	38	—	35	—	—	—	—
9 D.U.	62	33	44	—	—	—	—	—
Mean		52	48	42	51	27	40	53

Results are expressed as units in terms of c.cm. decinormal NaOH required to neutralize 100 c.cm. of fluid. G.U. = Gastric ulcer, D.U. = Duodenal ulcer, J.U. = Jejunal ulcer. F.T.M. max. = Maximum reading of fractional test meal.

Group II

Confirmation of the acid-depressing power of oil was demonstrated by this series, in which six feeds were given two-hourly by mouth, and 1 oz. of olive oil was syringed down the aspirating tube half an hour before each of the last three feeds. Reference to Table II will show that of the 9

TABLE II.—Average Free Acidities of Gastric Contents in 12-hour Tests of Six 2-hourly Feeds of Milk 10 oz., with Olive Oil 1 oz. Half an Hour before the Last Three

Case	F.T.M. Max.	First Half of Test: Milk Alone	Second Half: Milk and Oil
1 D.U.	45	3	15
2 D.U.	70	54	17
3 D.U.	31	31	31
4 G.U.	85	37	28
5 G.U.	60	16	5
6 D.U.	80	40	29
7 G.U.	70	14	30
8 D.U.	60	53	37
9 D.U.	—	73	68
10 D.U.	75	32	3
		43*	14*
Mean		36	25

* Test repeated with peach-kernel oil.

patients examined by this method 4 showed no significant difference between the two halves of the test; in the other 5 the addition of olive oil induced a reduction of acidity of 20 to 40 units. Peach-kernel oil in one case resulted in a depressant effect similar to that of olive oil. The preliminary fractional test meals of all 9 cases gave similar curves (Chart II), and no reason could be discovered for the apparent absence in the 4 cases of a depression by the vegetable oils, an effect which when present in the other 5 was considerable.

CHART II.—Hourly free acidity of gastric contents over 12 hours in one case, with six 2-hourly feeds of milk 10 oz. and olive oil 1 oz. half an hour before the last three.

Group III

The preceding data refer to results obtained with methods of treatment of a purely experimental nature, and it was felt desirable to attempt to evaluate the changes in acidity brought about by methods more applicable to routine treatment. Three patients admitted for peptic ulcer complicated by haematemesis were the subjects of this investigation, and three parallel experiments were performed: (1) Meulengracht - Witts dietary regime alone (Meulengracht, 1935; Witts, 1937); (2) that diet plus olive oil; (3) that diet plus olive oil and alkali.

The procedure was as follows: (1) On the first day gastric specimens on diet alone were analysed hourly; (2) on the second day the experiment was repeated with the addition of 1/2 oz. of olive oil half an hour before meals; (3) on the third day oil was given before meals and 1 drachm of an alkaline mixture (equal parts of mag. carb. pond., bismuth carb., sod. bicarb.) after meals. The results are summarized in Table III. With the diet alone readings are fairly high; in

TABLE III.—Average Free Acidities of Gastric Contents over 12-hour Periods in Patients on the Meulengracht-Witts Diet, with and without Olive Oil and Alkali

Case	F.T.M. Max.	Diet Alone	Diet + Olive Oil 1/2 oz. a.c.	Diet + Olive Oil 1/2 oz. a.c. + Alkali 1 dr. p.c.
1 G.U.	45	48	31	—
2 D.U.	55	49	40	31
3 D.U.	90	61	50	33
Mean		53	40	32

the series they varied from 30 to 80 units, and a fall of 10 to 20 units followed each feed. The use of oil depressed the acid level considerably, although in no case did it entirely obliterate free acid. With oil and alkali the reduction was more pronounced, several specimens being neutral in reaction; maximal values, however, were still high.

These experiments show that a dietary scheme which was designed to promote healing of an ulcer does nothing to lower acidity. Olive oil, as in the previous groups, plays its part in reducing acid levels, and a still greater fall is obtained by a combination of oil and alkali.

Group IV

The continuous intragastric drip apparatus has recently been much used experimentally to obtain a constant reduction of acidity. The substances in most common use have been milk (Winkelstein, 1933; Nicol, 1939) and aluminium hydroxide (Woldman, 1937; Woldman and Rowland, 1936; Woldman and Polan, 1939; Browne and McHardy, 1939; Eads, 1940). In the present series other simple foods and drugs have also been investigated. All 7 patients of this group received a standard "ulcer diet" consisting of three meals of easily digested food, not necessarily fluid, and three intermediate feeds, mainly of biscuits and milk. The acid levels with this diet alone were

similar to those in patients treated by the Meulengracht - Witts regime, described above, although the ulcer diet was fuller and more solid: it was thus again demonstrated that such dietary schemes are in themselves of little value in the control of hyperchlorhydria. The substances used flowed at the rate of 10 to 15 drops a minute into the stomach, through either a pharyngeal or a nasal tube with the tip at the cardia, from a container hung above the bed. At hourly intervals the tube was disconnected and swallowed further until the tip was near the pylorus; after air had been injected to clear it, gastric samples were removed, and the tube was withdrawn to its former position and reconnected to the apparatus.

TABLE IV.—Average Free Acidities of Gastric Contents over 12-hour Periods in Patients on the "Ulcer Diet" and a Continuous Intragastic Drip of Various Substances

Case	F.T.M. Max.	Diet Alone	Diet and Drip of				
			Milk	Aluminium Hydroxide	Olive Oil	Bovril	Water
1 D.U. ..	70	44	24	30	—	—	—
2 G.U. ..	37	65	10	4	29	41	—
3 D.U. ..	104	81	27	21	67	—	—
4 D.U. ..	48	60	18	2	41	—	—
5 G.U. ..	60	42	41	26	—	—	—
6 G.U. ..	63	46	5	4	29*	—	34
7 D.U. ..	68	63	17	—	—	—	—
Mean ..		57	20	14	41	41	34

* Drip of olive-oil emulsion.

Table IV summarizes the findings by this method. A drip of water was taken as a control, and, as was to be expected, it caused no significant alteration to the curve resulting from diet alone; bovril was similarly inactive. The acid-depressant action of olive oil was again in evidence: both undiluted and in emulsion form it reduced the general level to a considerable degree, although the minimal readings were still about 20 units. With both milk and a suspension of aluminium hydroxide (aludrox) the results were remarkably constant and effective; they regularly obliterated free acid, and one case was noteworthy in that no readings after the first were above the base-line. Chart III illustrates curves from one patient, and shows the effects of milk and aluminium hydroxide.

CHART III.—Hourly free acidity of gastric contents over 12-hour periods in one case on the "ulcer diet" with continuous intragastric drip. (a) i,....., diet alone; ii,....., diet plus aluminium hydroxide drip. (b) iii,....., diet plus olive oil drip; iv,....., diet plus milk drip.

It can be concluded from this part of the study that with chosen substances, used by a continuous-drip method—for example, milk and aluminium hydroxide—an effective control of hyperchlorhydria is obtained. An explanation is required, however, for the comparative failure of olive oil to do so alone, and some possible reasons are set out below.

Discussion

An investigator reporting alterations to gastric acidity in peptic ulcer must consider (a) the mechanisms involved in such alterations, and (b) their relation to healing. The factors concerned in the former are: (1) the motility of the

stomach; (2) the diminution of acid secretion; and (3) the neutralization of acid juice already produced.

Specimens taken an hour after the introduction into the stomach of any substance (especially in fluid form) do not show the changes in acidity which occur immediately, and by the end of an hour a rise in acid level may have taken place, whereas there had been immediate depression or neutralization. This may well explain the results with bovril and meat soup (Group I) and the initial fall in acidity with milk reported by Nicol (1939).

The present work was concerned, however, not with the immediate reaction to any food, but with the general effect over periods of prolonged feeding. The experiments indicate that there is no continued lowering of acidity when feeds are given two-hourly, in which circumstances a meal passes out of the stomach too rapidly for efficient neutralization because of enhanced motility, fresh secretion accumulating to raise the acidity of the emptying stomach to its previous high level. The initial fall after food may also be partly accounted for by dilution.

The continuous drip of milk induces a constant neutralization, enough milk being left to control the constant acid secretion, and this process can be maintained indefinitely. The effect is also obtained by the continuous drip of aluminium hydroxide, the properties of which, and of its pharmacological ally magnesium trisilicate, have been investigated very fully by many workers: to mention a few, Einsel and others (1932, 1934), Adams and others (1936), Emery and Rutherford (1938), Adams (1939a, 1939b), Bennett and Gill (1939), McIntosh and Sutherland (1940), in the case of the former; and, in the latter, Mutch (1936), Mann (1937), Kraemer (1938), Kraemer and Aaron (1940), Reid (1939). Experiments show that these agents neutralize HCl more slowly than do other alkalis, and from clinical experience their antacid effect is known to be spread over several hours.

Why should olive oil reduce acidity to a greater extent when given as a food (Group I) or added to the diet (Groups II and III) than when supplied continuously (Group IV)? If the hypothesis mentioned above holds good the explanation must be that oil has little acid-neutralizing power, and that its effect is in the main due to control of the other factors involved in the maintenance of acid level—namely, reduction of actual secretion and diminution of gastric motility. It is believed by many that the problem of motility is an important one, and that it throws light on the inconsistent results often obtained.

It is evident that undue stress has been laid on reduction of HCl alone as a necessary provision for the healing of gastric ulcers. Brown and Dolkart (1937, 1939) find that fluctuations in gastric acidity bear no relation to the onset of recurrence. Bloomfield and French (1938) obtain no correlation between healing and acidity, as measured by the "basal secretion" method. Wosika and Emery (1936) report that although the Sippy regime is effective in controlling acidity, they could demonstrate no relation between symptomatic relief and effectiveness of neutralization; while Steigmann and Fantus (1940), in their investigations of various antacids, showed that relief of pain could easily be produced, but that lowering of acidity occurred in less than one-quarter of their cases. Their conclusion is that ulcer therapy should not be based solely on methods of decreasing gastric acidity. The majority of these investigators, however, refer to acid levels before and after some particular application of treatment, and it is well known that hyperchlorhydria cannot be permanently cured by medical means. The matter is summarized by Mann (1939), who affirms that the important factors in the development of a peptic ulcer are chemical and mechanical. Healing always begins immediately after the surface is

protected from the acid gastric contents. The attainment of a low acid level is only one method of realizing this; such an effect is most completely attained by neutralization of the gastric juice as soon as it is secreted, a process which can only be carried out effectively at present by means of the continuous intragastric drip apparatus. This, however, is not a practicable method for the vast majority of cases, and should be reserved for those of especial severity.

The question of gastric motility in cases of ulcer would appear to be the problem of the future, and the next step ahead seems to be the discovery of some practical means of ensuring continuous diminution of gastric motility. Olive oil does not supply this want, and the continuous intragastric drip, as has been indicated, will not fulfil the condition of practicability. The purpose of further research must be to find some more effective method.

Summary

An investigation into the effects of various foods and simple drugs on the gastric hyperacidity of peptic ulcer is recorded, particularly with a view to prolonged acid reduction.

Certain foods, notably milk, when given two-hourly, have little persistent neutralizing power; these foods, although diverse in nature, produce similar acid levels.

Olive oil is strongly acid-depressant when supplied as a food or given before meals.

Considerable reduction of acidity results from a combination of oil and alkali with a suitable diet which by itself is ineffective.

The continuous intragastric drip apparatus provides at present the only means of constantly maintaining a low acid level. Milk and aluminium hydroxide are most effective in this method; oil is less so.

The mechanisms of these alterations in acidity are discussed.

Some considerations of the relation of acid control to healing of an ulcer are examined.

Investigation of alterations in gastric motility—i.e., some method of controlling the emptying of the stomach so that remedies are retained to ensure continuous neutralization of acid as it is formed—is suggested as perhaps the most important problem requiring further study in the treatment of peptic ulcer.

This work was performed in the Gardiner Institute of Medicine, Glasgow University and Western Infirmary, during the tenure of a Carnegie Medical Research Scholarship, which was interrupted owing to war service. I wish to express my gratitude to Prof. J. W. McNee for his constant advice and helpful criticism. I have to thank Messrs. John Wyeth and Brother, Ltd., for a supply of aludrox, and for the continuous intragastric drip apparatus.

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Medical Memoranda

Interstitial Subcutaneous Emphysema complicating Pulmonary Tuberculosis

After reading the case report by Dr. T. B. L. Bryan in the *Journal* of December 21, 1940 (p. 868), I looked up the records of a case which was under my care in the Sanatorium Pavilions, City Hospital, Walker Gate, Newcastle-upon-Tyne.

CASE RECORD

The patient, a man aged 27, was first seen by me in consultation in his home on April 21, 1936. He gave a history of not being well for about four months, the last five weeks of which had been spent in bed. There was no history of tuberculosis, but three years previously he had had an operation for a rectal abscess. The family history with regard to tuberculosis was bad. A sister was a sputum-positive case of pulmonary tuberculosis, a brother who had been a sputum-positive case of pulmonary tuberculosis died in 1934, and another brother who had been a sputum-negative case of tuberculosis died in 1919. The home conditions were poor; the family of three lived in a two-roomed tenement.

On examination the patient was pale and ill and his general condition was poor. He had impaired percussion over both sides of the chest, with crepitations more pronounced on the right. He was admitted to hospital on April 22, 1936. While there his temperature varied between 99° in the mornings and 103° in the evenings, and his pulse was rapid, usually about 120. A radiograph showed very widespread scattered densities in both lungs, with cavitation in each upper zone below the clavicles. His sputum was negative for tubercle bacilli. On April 25 he suddenly developed emphysema of the tissues of the neck and upper part of the chest and back. This caused some dyspnoea. A radiograph taken the same day showed a small left lateral spontaneous pneumothorax and also the air in the tissues of the neck. He was then transferred to the Newcastle General Hospital to see if surgical intervention could assist him and perhaps lessen the dyspnoea and distress. It was, however, decided that he would not be benefited by any operation, and he was brought back to the City Hospital on May 1. He continued to retrogress, and he died on May 31, 1936. The surgical emphysema which persisted until his death was limited to the neck and chest in its upper half. Permission for a post-mortem examination was not obtained.

COMMENTARY

This patient, who had extensive acute pulmonary tuberculosis, developed a left spontaneous pneumothorax and, at the same time, subcutaneous emphysema of the tissues of the neck and upper half of the chest. Spontaneous pneumothorax as a complication of pulmonary tuberculosis is common, but subcutaneous emphysema is a much rarer condition. Since I became associated with the Sanatorium Pavilions in 1923 there have been 6,266 admissions of cases of pulmonary tuberculosis and 1,512 deaths. The case recorded is the only one that has developed subcutaneous emphysema.

Fishberg (1932) states that interstitial subcutaneous emphysema is not unknown in acute pulmonary tuberculosis and is very rare in chronic tuberculosis. Meade and Stafford (1930) review the condition and describe some cases, but it is not often recorded in medical literature.

I am indebted to Dr. J. A. Charles, medical officer of health for Newcastle-upon-Tyne, for permission to publish this case.

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The wisdom of subsidizing private growers of cinchona in India is being urged by the Indian Chemical Manufacturers' Association, which points out that if efficient anti-malarial treatment is to be provided in India, at least 6 lakh lb. of quinine will be required annually. The present consumption is about 2 lakh lb., of which India produces about 70,000 lb., the remainder being imported from abroad.