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.