have been tested for sterility as they were received, and the results of these tests have been reported back to the depots. Some of the bottles have been found to be contaminated, and the number has steadily decreased as the depots have gained experience in the separation of the serum. As in no case was there any evidence of gross infection, and as the bottles are held at 4° C., at which temperature bacterial growth is slight, the infected bottles were pooled with the sterile ones during filtration. The majority of the contaminants were spore-bearing aerobes of the *subtilis* type, and Gram-positive cocci—either micrococci or sarcinae; pseudomonas was also isolated from serum and from stored citrated plasma. The necessity for filtration is evident, and from experimental results it is also evident that, although the contaminants of the *subtilis* type would be susceptible to the action of disinfectant, the Gram-positive cocci and pseudomonas would be resistant to its action, and the value of adding disinfectant to serum is doubtful.

While it is obvious that the correct procedure is to maintain conditions of absolute sterility during both the preparation and the solution of dried serum, and that the reconstituted serum should be used immediately, it is possible that in wartime reconstitution might take place under difficult conditions and it might not be possible to use the serum immediately. In such circumstances results would indicate that proflavine 1/100,000 and phenyl mercuric nitrate 1/50,000 would prevent bacterial growth for three days.

Summary

The effect of various disinfectants in controlling the growth of bacteria in serum, dried and reconstituted serum, and citrate plasma has been studied.

Growth of *B. subtilis* and *Staph. aureus* was prevented for six days by merthiolate 1/10,000, metaphen 1/10,000, phenyl mercuric nitrate 1/50,000, and proflavine 1/100,000. Growth occurred in sulphanilamide 1/5,000 after twenty-four hours.

Enterococcus was inhibited for three days by phenyl mercuric acetate 1/100,000, proflavine 1/100,000, and merthiolate 1/10,000. Growth in three days could be demonstrated in the presence of metaphen 1/10,000, phenyl mercuric nitrate 1/50,000, and sulphanilamide 1/5,000.

Ps. pyocyanea was not inhibited by any of these disinfectants.

Thymol and *p*-chlor-*m*-cresol were ineffective as bacteriostatics; and these and phenyl mercuric acetate precipitated proteins in serum.

Freezing at -20° C. and drying in serum did not affect the bacteriostatic power of metaphen, sulphanilamide, proflavine, and phenyl mercuric nitrate; nor did the presence of the disinfectants affect the solubility of the dried serum.

Moulds were inhibited by 1/50,000 phenyl mercuric nitrate but not by 1/10,000 proflavine.

Sulphanilamide will prevent the growth of *B. subtilis*, *M. tetragenus*, and pseudomonas in whole blood for thirty days at 4° C. but not at 22° C.

REFERENCES

- Aitken, R. S., Barling, B., and Miles, A. A. (1936). *Lancet*, **2**, 780.
 Albert, A., Francis, A. E., Garrod, L. P., and Linnell, W. H. (1938). *Brit. J. exp. Path.*, **19**, 43.
 Bernstine, J. B. (1929). *Amer. J. Obstet. Gynec.*, **18**, 220.
 Birkhaug, K. E. (1930). *J. Amer. med. Ass.*, **95**, 917.
 — (1934). *Ibid.*, **102**, 1224.
 Eggerth, A. H. (1926). *J. infect. Dis.*, **38**, 440.
 Fildes, P. (1940). *Brit. J. exp. Path.*, **21**, 67.
 Hirschfelder, A. D., and Wright, H. N. (1930). *J. Pharmacol.*, **39**, 13.
 Hunwicke, R. F. (1940). *British Medical Journal*, **2**, 380.
 Novak, M. (1939). *J. Amer. med. Ass.*, **113**, 2227.
 Powell, H. M., and Jamieson, W. A. (1931). *Amer. J. Hyg.*, **13**, 296.

The Foreign Department of the Reich Students' Administration announces (*Klin. Wsch.*, 1940, **19**, 1296) that more than 3,300 foreign students of both sexes are now enrolled in German universities. It is stated that no fewer than 65 countries of the world have sent delegates from their student youth to study in Germany.

LACTOSE FOR PREVENTION OF ODOUR IN THE CLOSED-CAST TREATMENT OF COMPOUND FRACTURES

BY

ALLAN D. WALLIS, M.D.

AND

MARGARET J. DILWORTH, B.S.

(From the Germantown Dispensary and Hospital,
Philadelphia, U.S.A.)

The increasing use of the closed-cast method of treating compound fractures, particularly under wartime conditions, induces us to propose a means of preventing the foul odour that is one of its principal disadvantages. Our procedure, of which a more detailed account will be published elsewhere, has been devised in connexion with the treatment of osteomyelitis by the Winnett Orr technique. The application of that method to compound fractures has been well described by Trueta (1939).

Protein-sparing Effect of Carbohydrate

Briefly stated, it is our hypothesis that the foul odour which develops in these casts is the result of putrefaction, defined by Rettger (1928) as "anaerobic decomposition of protein with the formation of foul-smelling products characteristic of cadaveric decomposition." Autolysis of devitalized tissue probably helps to initiate this proteolysis by providing preliminary breakdown products which are more readily attacked by micro-organisms than are native tissue proteins. It seems likely that anaerobic bacteria play an important part in putrefaction. Trueta (1939) has noted the presence of anaerobes in the wounds under consideration. Whether these organisms arrive through the blood stream or by accidental implantation remains to be determined. On the basis of these assumptions, by implanting a sugar in the wound one may take advantage of the fact that most bacteria will attack carbohydrate in preference to protein when both are simultaneously available. As Kendall *et al.* (1913) have expressed it: "Fermentation takes precedence over putrefaction." It is when bacteria are utilizing protein for fuel rather than for merely structural requirements that foul products are formed. This principle is commonly referred to as the "protein-sparing effect of carbohydrate."

In selecting a sugar for use in wounds lactose was chosen instead of glucose because, since its molecule is larger (Kendall and others, 1913), a solution of it isotonic with the blood contains more sugar, and (Kendall, 1923) loss by absorption from the wound should be less. Since the first stage in the fermentation of lactose is the production of galactose and glucose, the latter becomes available for the relatively few organisms which cannot utilize lactose.

Method of Using Lactose

For clinical use we have arbitrarily chosen a 12% solution. Lactose of U.S.P. grade is dissolved in distilled water and autoclaved in quantities of about 50 c.cm. Sterile gauze dressings which have been dipped in this solution are placed in the wound instead of the usual vaselined gauze packs. A covering of dry gauze is employed to keep the sugar from contact with the plaster lest crumbling occur. In débridement the solution should be made and autoclaved within two hours of the time the water is distilled, as a precaution against the presence of spores. At subsequent re-dressings this precaution is unnecessary, since the granulation tissue is an adequate barrier against toxins (White, 1931).

We have employed this method in Winnett Orr casts for periods up to four weeks without the development of

a definite odour of putrefaction. For longer periods it is possible that larger amounts of sugar may be needed. These could be provided by increasing the concentration of the solution or by adding crystalline lactose to the dressings. With the use of lactose both the amount of exudate and the tendency to excoriation of the surrounding skin appear to be diminished, probably because bacterial production of proteolytic enzymes is decreased in the presence of utilizable carbohydrate (Kendall, 1923). The usual healing process does not seem to be impaired by the addition of sugar. The dressings do not become adherent to the wound as is sometimes the case with vaselined gauze. Since *B. pyocyaneus* has little or no ability to utilize sugar this method does not affect its odour, which, however, is not nearly so offensive and penetrating as that of true putrefaction.

It is to be hoped that the use of lactose will prove as satisfactory in compound fractures as it has in osteomyelitis.

REFERENCES

- Kendall, A. I. (1923). *Physiol. Rev.*, **3**, 438.
 — Day, A. A., and Walker, A. W. (1913). *J. infect. Dis.*, **13**, 425.
 Rettger, L. F. (1928). *Newer Knowledge of Bacteriology and Immunology*, edited by Jordan and Falk, p. 222, Chicago.
 Trueta, J. (1939). *Treatment of War Wounds and Fractures*, Hamish Hamilton, London.
 White, M. (1931). *Lancet*, **1**, 1293.

ACUTE DILATATION OF THE STOMACH

BY

MAURICE LEE, F.R.C.S.

AND

EDGAR SOMERVILLE, M.B., B.Ch.

It is our intention, as the result of an unusual case of acute dilatation of the stomach, to discuss briefly the current views on this condition. Acute dilatation was described first by Kunderdt in 1871, and was soon recognized and commented upon by other people (Fagge, 1873; Rokitsky, 1861). Rokitsky suggested that it was caused by the dragging of the mesentery of the small intestine, which pressed upon and occluded the transverse portion of the duodenum. This theory has persisted till recently, but it is surprising to find it still in modern textbooks. It is difficult to conceive how the occlusion of the duodenum can bring about acute dilatation of the stomach when the case described below is considered. Most of the reported cases follow operations, but there are many accounts of the disease arising apart from operation—e.g., during a severe wasting disease (Herrick, 1906)—and the condition has been known to occur in healthy individuals as the result of overeating (Gillette, 1930). It has also been reported following childbirth and severe blows to the abdomen.

Dragstedt and others (1931) state that acute dilatation is rare after operations upon the stomach itself, and we have considered it noteworthy to report a case following an operation of partial gastrectomy, as no such case could be found in the literature.

There are many peculiarities in respect of acute dilatation. The first striking feature is the enormous amount of fluid from the stomach: where does it come from? One opinion is that the stomach is able to hold all this fluid because it has become paralysed as the result of reflex sympathetic stimulation. The suggestion has been put forward (Morris, 1882-3) that the enormous amount of fluid was the result of excessive secretion of the stomach mucosa—in other words, a gastrorrhoea. Certain factors discountenance this theory. For instance, in our own case, in which a large part of the stomach had been removed, it

is difficult to understand how the remaining portion could secrete 8½ pints in twenty-four hours. Again, if the fluid aspirated or regurgitated came from the stomach alone it would contain hydrochloric acid, but examinations of the fluid show it to contain little or no free HCl. It always contains bile (Herrick, 1906; Dragstedt and Dragstedt, 1922).

The present-day view considers that the dilatation is neurogenic and is the result of paresis of the muscles of the stomach. We suggest that the enormous amounts of fluid collecting in these cases arise from the alimentary canal below. As the result of the stomach becoming paralysed and dilated a negative pressure is produced in its interior. The intra-alimentary pressure where the muscular tonus is normal is higher than the intragastric pressure, and fluid flows backwards into the stomach—in other words, there is a backwash. The fluid keeps on collecting, and when enough is present it wells up into the oesophagus, rises into the pharynx, and is eventually expelled. This theory is supported by the fact that in these cases there is actually no true vomiting. The fluid which comes away just pours out from the patient's mouth. There is no sign of its being discharged in an expulsive manner. Once the condition has been started it is easy to understand why it should continue and how quickly the patient loses fluid. Not only fluid is lost but also valuable substances which are absorbed naturally lower down in the intestine and which are essential to retain the normal equilibrium of the blood. It is the loss of these valuable substances from the blood stream that is the real cause of death in these cases. There is no doubt that after operations acute dilatation of the stomach is of frequent occurrence in minor degrees, just as paralytic ileus is. It is difficult to understand why some cases should develop acute dilatation and others paralytic ileus. Fortunately these minor cases tend to right themselves. Factors which may make one suspicious that acute dilatation (or paralytic ileus) is developing are that the pulse rate is not lessening as expected after an operation, or that it is rising after having been lower for a short while. Or, again, the patient may complain of being uncomfortable under the bandages or show some uneasiness in his epigastric region. These cases should be met by passing a duodenal tube and leaving it in. This process of "sounding the stomach" is of the greatest assistance.

Case Report

The patient, a thin man aged 64, gave a history of abdominal pain in relation to meals for the last twenty years. He had noticed recently that his stools were often tarry. For the past six to twelve months vomiting had become more frequent and of greater volume; it relieved pain. A barium meal revealed great delay in emptying time. Gastroscopic examination showed chronic gastritis and pyloric stenosis, but no ulcer was seen. Occult blood ++ was found in the stools. A test meal disclosed a very high content of free acid.

The patient has had extensive medical treatment at other places, but without improvement. On January 31, 1941, a partial gastrectomy under spinal anaesthesia and gas-and-oxygen was carried out. A large-sized duodenal ulcer was seen. At the end of the operation the patient's condition was satisfactory: the pulse had fallen from 120 to 96. Twenty hours after the operation the pulse rate began to rise slowly, though the patient was comfortable and did not complain of symptoms. Thirty hours after operation the pulse rate had risen to 120 and he had a feeling of distension in the epigastrium. At this time he spat out 2 oz. of dark brown fluid which he said "just came up into my mouth." Acute dilatation was suspected, and the stomach was sounded by means of a Ryle tube. 30 oz. of fluid was aspirated. A continuous gastric drainage apparatus was set up and an intravenous drip was started. Morphine grain 1/4 four-hourly was administered, and the bed was raised two feet at the bottom. Within twenty-four hours from beginning the aspiration 8½ pints of fluid were withdrawn and the pulse came down to 86.