into attacks of abdominal colic coinciding with constipation, followed by relief with a copious stool and the passage of a large quantity of flatus. Any one of these attacks may terminate in an acute volvulus, and it seems probable that the determining factor is the circulation in the affected loop. So long as the twist is not sufficient to interfere with this there is a chance of spontaneous untwisting. The importance of this factor is illustrated by the varying way in which an acute attack supervenes. In most cases the onset is quick, but in some it is much more gradual.

Many years ago I had under my care in the Booth Hall Infirmary a mentally deficient man, aged 55 years. My attention was called to him by the attendant because he was complaining of abdominal pain. I found him not urgently ill, with a moderately distended abdomen and very evident visible peristalsis of the large bowel type all over the abdomen. His condition was not serious, and I watched him for some days before I operated. At operation there was an enormous volvulus of the sigmoid without any strangulation, and I successfully excised the sigmoid loop in two stages by Mikulicz's method. Visible peristalsis was also a well-marked feature in the case I mentioned previously.

That the progress of the final attack may be slow when strangulation is absent is also supported by the case of a mentally deficient male, aged 22 years, reported by Edward Milton Foote.² The first symptom was abdominal distension, noticed by an orderly, and operation was undertaken three days later because of increasing distension and visible peristalsis.

A much more striking example was recorded by Maurice Richardson.³

The patient, a man aged 47, congenitally deaf and dumb, had suffered from severe attacks of abdominal pain with constipation for five years. The attack which led to operation was precipitated by a dose of castor oil, and for a month after the onset there was no passage of motion. The abdominal distension gradually increased, until at the time of operation it was very great, and was accompanied by visible peristalsis. There was no vomiting. Although the attack had lasted so long there was no gangrene, but the affected loop was dark in colour. The bowel was aspirated, untwisted, and fixed in position. The volvulus recurred five months later, and was then resected successfully.

X-ray examination in patients suffering from symptoms of chronic volvulus may be of great value. A barium enema will show the greatly enlarged sigmoid loop. On two occasions I have operated in cases of this type and have successfully removed the sigmoid loop. In one this resulted in a complete cure of the symptoms, but the other patient still suffers from constipation and abdominal pain.

TREATMENT.

The treatment of acute volvulus is entirely surgical. Little is to be gained by wasting time with enemata, and purgatives only make conditions worse. When the loop is viable it is often possible to evacuate it by a tube pushed up from the anus guided by a hand in the abdomen. The twist is then easily undone, and, so far as that attack is concerned, the patient is cured. Many attempts have been made to prevent future twists by fixing the sigmoid to the abdominal wall or by plecting the mesentery. All of these are liable to be followed by recurrence. Indeed, one might almost say that recurrence is the rule.

There can be no doubt that the ideal treatment is resection of the sigmoid loop. This is always essential when the bowel is too damaged to recover, and I believe it is the right treatment in all cases. I have employed it in all four acute cases I have had to deal with. One patient, with a gangrenous sigmoid, died from prolapse of the small gut alongside the double-ended colostomy I had made. This happened on the fifth day, and up to that time the patient had been very well. The accident was certainly due to an error in technique. The other three patients, in all of whom gangrene was absent, recovered. The method I adopted in all four cases was immediate resection of the affected loop with a double-ended colostomy. Later an end-to-end anastomosis was made in two of these patients. The third saved me the trouble by re-establishing her intestinal canal spontaneously. This two-stage operation has seemed to me to be safer than an immediate end-to-end union. There is no doubt that immediate anastomosis gives a much cleaner and pleasanter result, and it may be, as Mr. Morley points out, that there is not the same accumulation and distension in the colon

above the volvulus as there is in cases of carcinomatous obstruction, and therefore not the same risk with immediate anastomosis as there is in these latter cases.

Finally, it is sometimes possible to recognize the presence of intermittent symptoms from chronic volvulus. When this is so, and the diagnosis is confirmed by the x-ray appearances, it seems justifiable to excise the sigmoid loop, as I have done on two occasions.

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THE PRESERVATION OF YELLOW FEVER VIRUS

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THE study of yellow fever has recently been greatly facilitated by the important discovery (Stokes, Bauer, and Hudson, 1928) that the common rhesus monkey (*Macacus rhesus*) is very susceptible to this infection. Hitherto the necessity of using human volunteers for any experiments has prevented a very systematic study of the disease, and therefore the value of this discovery cannot be overestimated.

Unfortunately the cost of transporting monkeys from India to the West Coast of Africa, necessitating transshipment in Europe, is very considerable, as the double journey involves a very high mortality. One of us (A. W. S.) found the mortality of monkeys sent to West Africa was such that their average cost came to nearly four times that in London. Apart from financial considerations, the study of the yellow fever virus can be conducted far more readily in countries where the natural carrier of the infection is absent. Experiments in localities where *Aedes aegypti* may be present involve keeping all infected animals under mosquito netting to prevent the chance of their being bitten by any mosquito and possibly starting an epidemic. Consequently infected monkeys in such localities are always a potential source of danger to the community and involve continual attention. In temperate countries these difficulties do not arise, as even in the presence of *Aedes* the temperature conditions are such that the mosquitos could never become infective. The development of the virus in the intermediate host is extremely slow and uncertain at a temperature of 22° C., and the optimum temperature seems to be in the neighbourhood of 28° C. Consequently the disease is restricted definitely to countries where such conditions prevail, and has never become endemic in temperate zones.

Hitherto the study of the disease has been confined to the regions in which it occurs, unlike the majority of human infections, which can now be investigated in the main laboratories of Europe and America, for although the French Yellow Fever Commission (Marchoux, Salimbeni, and Simond), 1903, carried infected mosquitos back to Paris from Brazil, no method of maintaining the virus was then known.

Considering the evidence in support of the view that yellow fever is caused by a filterable virus, it seemed of interest to see whether the virus could maintain its vitality when frozen, in the same way as many other filterable viruses, and accordingly an attempt was made to transport infected material in this manner.

The strain of virus employed was isolated from a case of yellow fever in a young Syrian at Dakar (Mathis, Sellards, and Laigret, 1928). This patient suffered from a mild attack, characterized by two febrile periods separated by a short intermission. The conjunctivae were slightly jaundiced and congested, and the urine contained a trace of albumin. On the seventh day of the disease, just before convalescence, the patient showed signs of collapse, with marked cardiac symptoms. Twenty hours after the commencement of the fever sixteen mosquitos (*Aedes*

egypti) were fed on the patient, and at the same time 4 c.cm. of blood were collected and inoculated into a rhesus monkey. Two days later this animal had a slight fever and was found dead on the eighth day after the inoculation. At the necropsy a slight jaundice was observed, and also lesions resembling those of yellow fever. The blood and a suspension of liver were inoculated into two young guinea-pigs, which remained healthy, thus excluding the presence of *Leptospira icteroides*.

Twenty-four days after feeding on the patient the mosquitos were allowed to feed on a rhesus monkey, which showed a sharp rise in temperature after an incubation period of three days, and succumbed to a typical attack of yellow fever two and a half days later. Another monkey was also infected by this same batch of mosquitos.

This strain was maintained in monkeys, either by the bites of infected mosquitos or by direct passage, for a period of nearly three months, all the animals showing the characteristic symptoms of the disease. Shortly before leaving Africa one of these monkeys was killed at the height of the infection, and some of its blood, and also pieces of liver, were collected in sterile tubes, which were then carefully sealed to exclude air, and at once frozen in a mixture of ice and salt. The sealed tubes, in metal containers, were kept frozen during the journey to London—a period of twelve days—and on arrival were inoculated into two rhesus monkeys.

The first monkey, inoculated with an emulsion of the liver, showed a rise of temperature after an incubation period of four days, and was found dead on the morning of the seventh day. Its temperature chart (Fig. 1) is reproduced herewith, and

FIG. 1.—Temperature chart of rhesus monkey inoculated with a suspension of frozen liver from a case of yellow fever. The animal was found dead on the morning of the eighth day.

it will be noticed that on the day preceding death the temperature was subnormal, a common feature of the disease in monkeys. On the last day of the disease the animal showed symptoms of jaundice, which were particularly well marked in the ears and face. At necropsy the subcutaneous fat, and especially that around the pericardium, was intensely yellow. The liver was mottled and pale, and showed marked signs of fatty degeneration. The stomach contained a quantity of the characteristic coffee ground material.

The second monkey was inoculated intraperitoneally with the frozen blood, and as it showed no rise of temperature on the fourth day it was reinoculated with infected liver material from another monkey. The following day it showed a rise in temperature, but in view of the subsequent course of the disease, as shown in the accompanying chart (Fig. 2), it is

FIG. 2.—Temperature chart of rhesus monkey inoculated with frozen blood from a case of yellow fever, and reinoculated on the fifth day with infected liver material. The animal was killed on the tenth day.

somewhat doubtful whether this first rise was the result of the blood inoculation or not. Four days after being inoculated with the infected liver emulsion the temperature showed a distinct rise, which continued the following day, when the animal was killed. At necropsy the only obvious features of the disease were slight jaundice in the subcutaneous fat, fatty degeneration of the liver, and slight inflammation of the lining of the stomach. Subinoculation into another monkey, however, produced a typical and fatal attack of yellow fever. Cultures for *Leptospira* on Noguchi-Wenyon medium, blood agar slopes, and Fletcher's medium, all gave negative results.

It is evident, therefore, that the yellow fever virus can maintain its virulence when frozen for at least twelve days, and possibly much longer, a property which will be of considerable assistance in preserving the virus and further studies on the nature of the infection.

We should like to express our thanks to Dr. C. M. Wenyon, F.R.S., for providing us with accommodation, and also for much valuable assistance.

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A FATAL CASE OF POISONING BY A NON-ARSENICAL WEED-KILLER.

BY

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IN the following case of poisoning by a weed-killer the effects were so profound and so rapidly fatal that an account of the clinical course would seem to be of general interest. The weed-killer taken consisted of 75 per cent. tar acids emulsified with soap; it was dark in colour, had a strong smell of tar, and formed a milky emulsion in water. The makers reported that both lysol and this weed-killer consisted of carbolic acid, soap, and water, lysol containing 50 per cent. and the weed-killer 75 per cent. carbolic acid, while the proportion of soap in lysol was larger. They also stated that the symptoms of poisoning produced by the weed-killer would be similar to those of carbolic acid or lysol poisoning.

Commercial cresylic acid, so-called liquid carbolic acid, consists of 35 per cent. orthocresol, 40 per cent. metacresol, and 25 per cent. paracresol. The effects produced by the cresols are identical with those of phenol; metacresol is the least poisonous, and paracresol the most poisonous (see *British Pharmaceutical Codex*, p. 369). In cases of poisoning by cresol the treatment for carbolic acid should be employed (*ibid.*, p. 370). Taylor, in *Medical Jurisprudence*, describes as comparatively innocuous a substance consisting of 20 per cent. cresylic acid with neutral hydrocarbon oil and resin soap; it is a dark-coloured liquid, mildly antiseptic. One instance of poisoning by it is reported in detail by Dr. Pinner, who states that the effects are not fatal.

The clinical details of the case are as follows.

The maximum amount which could have been swallowed was just over 2 fluid ounces (56.763 grams). To test the accuracy of this statement the average contents of a series of unopened tins were compared with the contents of the tin from which the poison had been taken. The patient, a well-built man, aged 32, had always enjoyed fairly good health, but had recently been greatly worried by the death of his wife. Somewhere about 10 a.m. on August 21st, 1927, he took the poison, and about 10.40 a neighbour found him lying in bed; he was groaning, but quite conscious, and refused a cup of tea. He did not complain of pain and had not vomited. When I saw him, at 11 a.m., he was comatose; there was no corneal reflex and his breathing was stertorous. He was markedly cyanosed, and his pulse was fast and almost imperceptible. His skin was cold and clammy, and he was sweating profusely. In his breath a strong smell of tar was detected, similar to that of a liquid contained in a glass by his side and in a tin of weed-killer also in the room. Careful examination revealed no sign of corrosion of the lips, tongue, mouth, or pharynx.

He was removed to hospital, and about 11.20 was receiving treatment. The stomach was washed out, and the returning fluid, milky in appearance, had a strong smell of tar; it contained no foodstuffs. After repeated wash-outs white of egg was put into the stomach, and in view of his cyanotic condition oxygen was given; atropine sulphate was also injected hypodermically. He rapidly went downhill and died about 11.40 without regaining consciousness. There were no signs of corrosion of the lips, mouth, tongue, or pharynx.

The necropsy revealed no signs of any abnormality save an old pleurisy in the left side. The stomach and oesophagus were removed and the contents sent for analytical examination. Careful examination of both stomach and oesophagus showed not the slightest sign of corrosion or excoriation. The stomach contained no food, only some turbid fluid with a strong smell of tar.

The report of the analyst stated:

"The contents of the stomach smelt of phenols or tar acids and the distillate gave a strong reaction for them. No cyanides