aegypti) 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

or hydrocyanic acid were present. No mercury, lead, copper, arsenic, or tin were present. No alkaloidal poison could be detected."

In view of the assertion by Pinner as to the relative harmlessness of cresylic acid, and of the statement of the makers of the weed-killer that the toxic action of this substance would be similar to that of carbolic acid and lysol, it seems that in this case, at all events, the toxic effects were not those usually seen in a case of carbolic or lysol poisoning. The symptoms were those of a poison acting on the nervous system—the so-called narcotic action of carbolic acid and its homologues—and this action was probably due to the paracresol. It is evident that emulsified preparations of the tar acids (carbolic homologues) are not necessarily corrosive; if a 75 per cent. emulsion is tested on the lip or tongue the effect is simply a temporary smarting, whereas carbolic or lysol produces burns on even hard skin.

Conclusions.

My object in reporting this case is threefold:

1. To show that cresol or cresylic acid is not the innocuous substance described by Pinner, at all events when taken in a concentrated form as an emulsion on an empty stomach. Under these conditions, when the toxic cresols are present, the rapid absorption of the poison by the stomach has a profound and fatal effect on the nervous system.
2. To suggest that a substance which can have so rapid and fatal a result should be included in either Part 1 or Part 2 of the Schedule of Poisons. At present it can be sold over the counter by any person, provided it is contained in a closed vessel distinctly labelled poisonous, with the name and address of the seller and a notice of the special purpose for which it is intended.
3. To urge that more attention should be paid in textbooks to the toxic action of the cresols on the nervous system. Attention seems to be focused mainly on the corrosive action. This is true of carbolic acid, and of lysol to a lesser degree, but when we deal with the cresols (cresylic acid), the corrosive action is negligible, while the toxic effects on the nervous system are profound.

ERYTHEMA NODOSUM AND ACUTE ENDOCARDITIS FOLLOWING TONSILLITIS:

RECOVERY AFTER TREATMENT WITH ANTISTREPTOCOCCUS SERUM.

BY

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THE following case seems of sufficient clinical interest to record.

On January 20th I was called to see a girl, aged 22, suffering from acute follicular tonsillitis. A swab taken for diphtheria proved negative, and the patient was given a sodium salicylate mixture. Defervescence with amelioration of symptoms occurred, and by the fifth day the temperature was normal and the throat much better. The next morning, however, the patient was worse, and the temperature had risen abruptly to 102° F. The face was flushed, she was sweating profusely, and she complained of painful spots on the legs, pains in her joints, dyspnoea, and palpitation on moving. Examination of the legs revealed a crop of large, roundish, raised, tense swellings, of the whole thickness of the skin, along the anterior borders of both tibiae. Bright red, glossy, and extremely tender to the touch, they presented the typical eruption of erythema nodosum. The right knee, right ankle, and left wrist were painful, but not swollen. The precordial impulse was increased, the first sound at the apex was roughened, and the second pulmonic sound accentuated. I recalled the association of erythema nodosum with true rheumatism, and accordingly increased the salicylate dose to 20 grains every three hours, combined with sodium bicarbonate. A sedative lotion was also prescribed for the legs. The urine was febrile, and contained a little albumin. During the next few days the temperature ranged between 101° and 103.6° F., and fresh crops of erythematous swellings kept appearing on the legs and thighs. There was now at the apex a soft, blowing, systolic murmur, conducted into the axilla, the second pulmonic sound remained definitely accentuated; and an appreciable increase in the transverse area of cardiac dullness could be detected. There were no rigors, the spleen was not enlarged, and the lungs were clear. Severe pain continued in the joints, and now affected also those of the fingers of both hands, but no arthritis developed. The

salicylates, however, did not influence the temperature nor relieve the joint pains.

On the fifth day following the onset of the erythema nodosum and the endocarditis the patient was still very ill, with no sign of improvement. The temperature was 103° F., and the pulse 120; she was still dyspnoeic, and a fresh crop of swellings had appeared that morning on the right leg. That afternoon I injected 25 c.cm. of polyvalent antistreptococcus serum well under the skin of the flank. The result was almost dramatic. By the next morning the temperature had dropped to 99.6° F., and the pulse to 90; the joint pains had gone, the tension in the erythematous swellings was relieved, and the dyspnoea was much better. From that day no fresh crops of erythema nodosum appeared, and those already present, even the new ones of the previous day, disappeared in five days. On the third day after the injection some peritonsillar inflammation developed (until then the throat had been much better), and the temperature rose from 98.8° to 100° F. I injected a further 10 c.cm. of the serum, and twelve hours later the throat had improved and the temperature was normal. The temperature and pulse have now remained normal for a fortnight, no acute symptoms have recurred, and the girl feels much better. At the time of writing she is being kept in bed a little longer for her mitral condition.

I am aware that the connexion of erythema nodosum with rheumatism, although long debated, is not proved, but there were justifiable grounds in this case for associating the two conditions—namely, the initial tonsillitis, the joint pains, and the endocarditis. In spite of this, however, large doses of the salicylate had no effect. It is well known that tonsillitis can be an etiological factor in acute endocarditis, and, judging from the remarkable effect of the antistreptococcus serum in this case, it is possible that the tonsillitis ushered in a train of streptococcal developments, producing not only the endocarditis, but also the erythema nodosum. The etiology of the latter is still not decided, although Rosenow claims to have isolated a bacillus with coccoid forms. I am convinced that the serum had a decided beneficial effect on the erythema nodosum in my case, and, this being so, there may be here a point of etiological significance in connexion with his theory.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

DIAGNOSIS OF "PLEURAL SHOCK" FROM COCAINE POISONING.

THE differential diagnosis of acute cocaine poisoning and "pleural shock" may be a very difficult matter, as the following report of a case shows.

A dental mechanic was readmitted to Winsley Sanatorium on March 16th, 1928, and, owing to constant bleeding, it was decided to perform artificial pneumothorax, though this was not otherwise required. I ordered a 1/4 grain of morphine to be injected half an hour before the operation, and the patient seemed unduly anxious to know what drug had been employed. When I came for the operation he asked me what local anaesthetic we used, and I told him epinephrine, each cubic centimetre of which contains epinephrine 0.0003 gram, and cocaine hydrochloride 0.02 gram. I thought the question a professional one and treated it as such. He made no comment. The injection of 1 c.cm. was given with the usual small syringe with its corresponding needle, and I "felt" the pleura; since the patient was nervous I paid particular notice to the effect of this, but he did not feel the prick. Wishing to wait for five minutes and to take his mind off the artificial pneumothorax, I talked about the war, in which he had been gassed and had suffered from neurasthenia. He suddenly became faint, and cried out "I am going"; he was at first quite limp and looked like a corpse, then there followed a spasm, the face was drawn to one side with marked stiffening of the left sterno-cleido-mastoid muscle, reminding me of the classical picture of torticollis in the textbooks. The teeth were clenched and grinding, the respirations rapid, the pulse could not be felt at the wrists, and there were only very feeble indistinct heart sounds. I thought the condition was delayed pleural shock. I administered strychnine grain 1/60 on empirical grounds, and after half an hour the whole attack was over. In the afternoon he was much better and told me he had an idiosyncrasy to cocaine; this I verified subsequently.

In his introduction to *Forensic Medicine* Dr. Burridge gives the symptoms of cocaine poisoning as—

"If a poisonous dose has been absorbed the person becomes pale, feels faint, and may fall to the ground. Convulsions followed by unconsciousness may occur. The pupils are dilated and insensible to light. The pulse may be uncountable at first. On recovery the patient complains of diminished sensibility of the hands, etc. In several cases epileptiform convulsions have occurred, followed by death in a few minutes";