To the reader it might appear that Dr. Doll was intent on seeing that a straight line should go through the datal points; but if we proceed on the usual lines by joining up points in sequence we get the graph to the right of Fig. 2, from which it will be seen that there is no simple relation between the death rate from lung cancer and the degree of cigarette-smoking. The people of the U.S.A. are the largest per person cigarette-smoking people in the world, yet their lung-cancer death rate is about the same as that of the people of Switzerland and Denmark, who smoke about one-half of the cigarette quota of Americans. It is to be accepted as a fact that people in England and Wales with lung cancer do on the whole smoke more cigarettes than control people of the same age group, but the fact that as we go from one country to another no relationship exists between cancer death rates and the degree of cigarette-smoking seems to point to something quite significant; surely there must be some other factor or factors at work. This is not a matter of academic interest but one of high practical importance. If cigarette-smoking is responsible for the majority of deaths from lung cancer, how is it that people in the U.S.A., who smoke about 30% more cigarettes than we do, show a death rate from lung cancer (1950) less than half that of ours? The respective rates are 120 and 280 per million inhabitants.

Whether smoking is, or is not, one of the causes of lung cancer in our midst, there will be few people to dissent from the warning given by the Minister of Health (February, 1954) to young people on the dangers of excessive smoking. All excess is bad.

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Fatal Case of Toxic Hepatitis Implicating Chlorpromazine

Chlorpromazine has been used on 500 psychiatric cases (to be reported subsequently), of which six developed jaundice. The one ending fatally is here described.

The issue is clouded by the fact that three cases diagnosed as infective hepatitis have occurred concurrently in patients who had never received chlorpromazine or other liver toxin. These cases have been clinically and biochemically indistinguishable from the chlorpromazine cases. The incidence of jaundice in the latter, however, is significantly higher than among the rest of the hospital population.

CASE REPORT

The patient, a woman aged 45, with a recent history, was admitted in a severely disturbed, excited, restless, overactive state with a paranoid psychosis. She showed a mitral stenosis, normally asymptomatic, which at her current level of activity led to dyspnoea and cyanosis. Her blood pressure was 130/80. As sedation was not consistently successful, she was given oral chlorpromazine, the dosage being quickly stepped up to 500 mg. a day. Aided by amylobarbitone sodium in doses not exceeding 36 gr. (2.4 g.) a day, the excitement and overactivity abated, the mental content becoming progressively more rational and coherent. After three weeks of this regime, when 10 g. of chlorpromazine had been consumed, jaundice appeared, so the drug was withheld. A 24-hour pyrexia of 101° F. (38.3° C.) had preceded the jaundice by four days.

Throughout the ensuing 13 weeks the serum proteins, thymol turbidity, and colloidal gold were normal. Alkaline phosphatase remained between 20 and 30 Armstrong units, apart from a terminal climb to 70. Serum bilirubin rose to a peak of 23 mg. per 100 ml. by the end of the third week, thereupon declining to a level of 16–18 mg. per 100 ml. Throughout, the "direct" component of the serum bilirubin was two to two and a half times as great as the "indirect." Blood counts showed no significant change.

The liver was enlarged from the onset, but its margin rose and fell several times for no apparent reason. It was regular and never tender. Stools were pale or near-white; the urine was heavily laden with bilirubin. Her appetite was good until the last week. Itching was present during the earlier weeks. Prior to death in stupor, auricular fibrillation supervened.

Necropsy (Dr. David Haler) showed an enlarged nutmeg liver, double mitral and aortic lesions, purulent terminal pericarditis, and congestive heart failure. Microscopy of the liver supported a diagnosis of toxic hepatitis.

Comment.—It is now generally accepted that chlorpromazine is toxic to some apparently healthy livers, and even more so to those already diseased. It would appear from this case that passive congestion of the liver renders it vulnerable. No mechanism explaining this toxic effect can be offered, nor any prophylaxis suggested other than the orthodox dietary measures.

My thanks are due to Dr. H. C. Beccle, medical superintendent, to Dr. David Haler for conducting the necropsy and to Dr. R. Forgan, of May and Baker Ltd.

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Removal of Rubber Teat from Intestine of an 8-weeks-old Baby

A child born on October 20, 1952, by normal delivery, birth weight 7 lb. (3.2 kg.), was admitted to hospital at 11.30 a.m. on December 17. The history was that two days before admission it had started vomiting and had vomited every feed since then. It had been breast-fed for six weeks, and a week before admission had begun feeding on full-cream National dried milk, and had become constipated and then had diarrhoea; it then again became constipated for two days before admission. The vomit was projectile, consisted of milk, and usually occurred during the feed.

On examination the child looked marasmic and was in a very dirty condition. It was dehydrated and had depressed fontanelles of the skull. When fed on admission it had projectile vomiting, but there had been no visible peristalsis, and no tumour was felt. After every feed that was now given there was projectile vomiting, and the vomit contained bile. On December 23 the child was screened after a barium meal and was found to have an obstruction in the jejunum, 1–2 ft. (30–60 cm.) from the duodeno-jejunal flexure.

The patient was referred to me by the paediatrician for a surgical opinion, with a diagnosis of intussusception. On examination there seemed to be a mass in the left hypochondrium which came up to meet the palpating fingers but which disappeared on deep palpation.

Operation was carried out on December 23 through a right paramedian incision. A distended loop of small gut presented in the wound. On palpation the obstruction was found to be due to a rubber teat in the jejunum, about 2 ft. (60 cm.) from the duodeno-jejunal flexure. A longitudinal incision was made over the jejunum and the teat removed. The bowel was sewn up transversely with interrupted silk sutures, and the wound closed in layers. The child made an uninterrupted recovery and was discharged from hospital on January 12, 1953.

The only reference which I could find to a previous case was a case of Lawson's (1899) in which a 6-months-old baby was playing with a "comforter," and the parents knew that the baby must have swallowed it. On this occasion the patient was treated by observation, and four weeks and four days later the teat was found lodged in the baby's anus.

An interesting point is the fact that if, on examination of a case of intussusception, a mass is found on light palpation which disappears on deep palpation, it may be due to the presence of a teat in the small bowel as in the present case.

My thanks are due to Dr. J. G. Dathan for referring this case to me, and to Dr. C. Hayes for the anaesthesia.

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REFERENCE
Lawson, G. L. (1899). *Aust. med. Gaz.*, **18**, 225.