which beset pioneers, to establish an organization designed to control tuberculosis by treating it as an infectious disease, a concept which, even after the announcement of Koch's discovery, was slow in gaining general acceptance. The functions of Philip's tuberculosis dispensary included examination of patients at the dispensary and the keeping of records, including those of the home surroundings; instruction of patients, especially in ways of minimizing the risk of infection to others; the treatment of patients in their own homes, and the selection of those likely to respond to sanatorium treatment and those needing hospital treatment; and general education of patients, their friends, and relatives. It is interesting to find Philip discussing, in 1904, the home treatment of pulmonary tuberculosis,[4] and to reflect how the urgent needs which developed during a major world war forty years later led to a revival of interest in this matter, though the vogue for polysyllabic pretentiousness had debased his simple word "home" into "domiciliary."

The basis of Philip's concept was to treat tuberculosis as a unitary problem. Recent developments in what are now called "the chest services" have militated both for and against the application of this concept. On the one hand, the tendency for the clinic physician to have control of beds, in either a hospital or sanatorium where he remains responsible for the treatment of patients from his clinic, is favourable to this concept. On the other hand, the tendency, of which the change of title from "tuberculosis dispensary" to "chest clinic" is a sign, towards the widening of the interests of the clinic physician to fields outside tuberculosis, while in many respects admirable, may well in the long run make it difficult to maintain the unity of treatment and prevention in tuberculosis. A situation may well arise at some time in the future in which the present organization becomes unworkable. Even after tuberculosis has become a rare disease, an integrated scheme for its prevention will still be required. If the existing organization remains unchanged, a physician who will deal clinically with a wide range of respiratory diseases, including only very few cases of tuberculosis, nevertheless will retain responsibility for a complex scheme of preventive measures. At this stage the question whether a physician whose main interests are clinical can be expected to be responsible for preventive measures directed towards a disease which he rarely sees will have to be faced. Although the principles underlying methods of control of tuberculosis remain unchanged, the organization which was efficient in applying them in Philip's day may well require modification as the epidemic situation alters.

As Dr. Clayson points out, we may be said at present to have controlled tuberculosis, but the eradication of tuberculosis is going to be much more difficult. The proper application of B.C.G. vaccination as the epidemiological situation changes, the use of chemoprophylaxis for those found by tuberculin testing to have been recently infected, thorough case-finding by all methods, especially by mass radiography, the routine radiography both of those whose occupation or environment places them at special risk and of those in whom, by virtue of their occupation, unsuspected tuberculous infection would be a special risk to others—all these have their part to play, and all of them were adumbrated, at least in principle, by Sir Robert Philip.

PROTECTION AGAINST IONIZING RADIATIONS

The need to protect hospital workers from the harmful effects of radiations has become more pressing with the growing use of radioactive substances in medicine. The problem is not a new one: recommendations for the protection of people using radium and radon were put forward by the National Radium Commission in 1929–30. In 1949 this advice was extended to include artificially produced radioactive isotopes, and further recommendations[1] for the maximum permissible amounts of many of these substances in the body were made in 1955. The surge of interest in the effects of atomic radiation has recently culminated in the formulation by the Radioactive Substances Standing Advisory Committee of a "Code of Practice for the Protection of Persons Exposed to Ionizing Radiations."[2] According to the preface the code is intended primarily for those who work in National Health Service hospitals. It is to be brought to the attention of the staffs of radiological, radiotherapy, and physics departments, and all employees concerned must read the relevant sections and sign a statement that they have understood them. The booklet consists of two sections, one on the more familiar problem of protection against *x* rays, whether used for diagnosis or therapy, the other on radioactive isotopes. The technical facts necessary for the interpretation of the various rules set out in the code are given in its supplement. All who work with radiation will do well to note the extension of the definition of "tolerance dose" (maximum 0.3 röntgen/week) to include a cumulative dose of 50 rem per decade: this allows an average dose of only 0.1 r/week. The amount of radiation received "should be systematically checked to ensure that maximum permissible doses . . . are not exceeded." Responsibility for this will be taken by the hospital physicists working in the major hospitals or by the Radiological Protection Service at Clifton Avenue, Belmont, Sutton, Surrey.

[1] "Recommendations of the International Commission on Radiological Protection," *Brit. J. Radiol.*, 1955, Supplement No. 6.
[2] Published by H.M.S.O., London. Price 8s. net.

No radiation worker can fail to benefit from a careful study of this code, which contains far more than a set of rules and should do much to ensure the safety of hospital staff exposed to radiation. For example, it gives useful information for assessing how much of a given radioisotope may be dangerous and for designing laboratories in which radioisotopes are to be handled. It also contains practical hints and tips such as a cleaning procedure for decontamination of the skin. It is perhaps a little disappointing to find that rules for the disposal of radioactive waste are not given; however, these are under consideration and will be issued shortly as an addition to the code. Expert advice will certainly be needed to put the code into practice in individual hospitals. The head of the department is, quite rightly, to be made responsible for working conditions and for the instruction of new personnel, but the code also designates a safety officer who is to be "specifically responsible for radiological protection measures." It would seem desirable to employ the services of the hospital physicist of the group or region for this duty; such an officer would be able to ensure, by means of periodic visits, that the spirit of the code was being applied in all the hospitals of the group. Many of the usual methods of protection are effective only if applied by the people concerned at all times: even in the larger centres it is no uncommon sight to see the most elementary mistakes being made, such as the pipetting of radioactive solutions by mouth. It is the responsibility of the head of the department and the safety officer to see that these things do not happen.

The radioisotope laboratory that a hospital requires will largely depend on the type and size of the hospital, but the code lays down a few general rules in terms of the quantity and radiotoxicity of the isotopes and the manipulations which are to be carried out with them. Where only very simple manipulations are made with solutions containing quantities of less than a millicurie of the more common isotopes, such as ^{131}I, ^{32}P, ^{59}Fe, ^{24}Na, and ^{51}Cr, a simple laboratory (grade C) will suffice. On the other hand, for those hospitals where ^{131}I, ^{32}P, and ^{198}Au are used for therapy, a rather more elaborate laboratory (grade B) is needed. However, even those facilities which are regarded as necessary for grade C laboratories often do not exist at the moment; and if they do exist they are often not used. In these laboratories only simple precautions are needed, such as the provision of benches with disposable covers, a good fume-hood, plenty of laboratory tongs and trays, and some lead to provide γ-ray shielding. It seems likely that the smaller hospitals would be well served with one isotope laboratory of the B or C type, while the larger ones might need a central grade B laboratory as a "hot lab.," which it would be sensible to site in a separate part of the hospital premises, and a series of grade C laboratories in those departments where tracer doses were being used. For the protection of those working in these laboratories the level of radioactivity is limited, and this ensures that the counting background will not be high. A simple instrument for detecting radioactive contamination in laboratories would be a valuable safeguard.

PSYCHOPATHS

One feature of the report of the Royal Commission on mental illness[1] which has stirred controversy is the use of the term "psychopathic." The Commission proposed to recognize three main groups of patients: (*a*) those suffering from mental illness, (*b*) "psychopathic patients or patients with psychopathic personality," and (*c*) "those of severely subnormal personality," which would include mainly idiots and imbeciles. Group (*b*) was to include any type of aggressive and inadequate personality not falling in either of the two other groups and requiring care and treatment. All patients at present classified as feeble-minded or moral defectives would also fall into this division. There is general agreement about the need for a category such as this, which would also comprise alcoholics and other addicts in need of care and treatment, but the proposed terminology has caused uneasiness, which has been ventilated in the correspondence columns of this *Journal*. Members attending a recent quarterly meeting of the Royal Medico-Psychological Association, while in principle welcoming the proposed grouping, were unanimous in objecting to a diagnostic term being adopted and newly adapted for legal and administrative purposes, especially one so controversial as "psychopath" or "psychopathic."

It was J. L. A. Koch[2] who in 1891 introduced the term in a book on what he called the psychopathic inferiorities. "Psychopathic" came to mean any kind of abnormal personality type which was not the manifestation of psychosis or mental defect. In one of the best-known monographs on the subject[3] psychopaths are defined as "those abnormal personalities who suffer from their abnormalities or cause society to suffer." This definition embraces the neurotic as well. While many psychiatrists regard psychopathic personality traits as manifestations of a variation within the normal range, Sir David Henderson[4] has regarded it as a true illness of obscure origin and spoken of psychopathic states which he proposed to divide into predominantly aggressive, inadequate, and creative types. So great has been the terminological confusion that some modern classifications of mental disorders avoid the term altogether and replace it by the more general one of "abnormal personality," of which they distinguish a considerable variety. Yet the term "psychopathic" is so well established and so handy grammatically that repeated proposals for its abolition have been disregarded. The *British Journal of Delinquency*[5] published a special number devoted to the problem of the psychopathic offender, without, however, offering a precise definition of psychopathy.

There has been a tendency in the British and American literature to narrow the meaning of the term, with the result that it has become almost identical with "moral insanity." The most recent American monograph[6] on the subject describes the psychopath as an "asocial,

[1] Cmd. 169. 1957. H.M.S.O., London.
[2] Koch, J. L. A., *Die psychopathischen Minderwertigkeiten*, 1891, Ravensburg.
[3] Schneider, K., *Die psychopathischen Persönlichkeiten*, 1950, Vienna.
[4] Henderson, Sir David, *Psychopathic States*, 1939. London.
[5] *Brit. J. Delinq.*, 1951, **2**, 77.
[6] McCord, W. and J., *Psychopathy and Delinquency*, 1956, New York and London.

aggressive, highly impulsive person, who feels little or no guilt and is unable to form lasting bonds of affection with other human beings." This definition has the approval of G. W. Allport, the author of a classical book on personality. Whatever one may think about this use of the term, it is a fact that the general public, the members of the legal profession, and most medical men regard psychopaths as antisocial, usually aggressive members of the community and as potential criminals. Many psychiatrists and psychologists use the term as a diagnosis. But to give, by law, this label to a large group of citizens in need of treatment, most of whom have never been and never will be in conflict with the criminal law, would cause much misunderstanding and distress. Besides, through the arbitrary stipulation of an official meaning of this controversial term, British psychiatrists would be at a considerable disadvantage in communicating with their colleagues abroad, who would not thus be restricted in the use of this term. It should be possible to devise a better one for that particular group of patients. Diagnosis by legislation would be as undesirable as legislation by diagnosis.

BOTULISM

Botulism has always aroused an interest out of all proportion to its frequency as a cause of food-poisoning. Every medical student probably knows that botulism is the model example of the action of an exotoxin which can cause death even if the organisms which produced it are no longer alive, and that the dose necessary to cause death is minute. A recent outbreak of botulism among Labrador Eskimos[1] illustrates the characteristic features of this deadly intoxication. A party consisting of six men, two women, and some children moved to an autumn sealing station, a hut which had not been used for some months. The women were asked to prepare an Eskimo dish called *utjak*—made by "placing seal flippers, complete with fur, in a container, and leaving this with a lid on it behind the stove for a variable period of several hours to several days." The temperature of the food during this period probably varied between 20 and 40° C. (75–120° F.). On this particular occasion there was considerable reluctance on the part of the women to prepare the meal at all. When it was finally prepared and eaten about noon on December 10, 1956, it did not seem to be very appetizing even to Eskimo taste. One man "thought it tasted bad, and vomited almost at once. He was the only man to survive." The first victim was a girl aged 6, who died within two to three hours, then at intervals from four hours up to three days five men died. The two women who prepared the meal under protest ate none of it and were unaffected. One other survivor was a girl of 8, who recovered in a few days. The main clinical features of the illness were difficulty in swallowing, regurgitation of fluids through the mouth, abdominal pain and distension and in some cases peculiarity of speech, various forms of paralysis, headache, and cramps. *Clostridium botulinum* type E was isolated from a sample of seal flipper from the food consumed, and type E toxin was found in the flipper in a concentration of about 1,000 minimum lethal doses per gramme of flipper.

This grim story illustrates well the clinical features of the disease. Its epidemiological importance is discussed by K. F. Meyer[2] in a review of botulism as a world health problem. He states that "throughout the world during the past 50 years approximately 5,635 persons contracted botulism and 1,714 of these persons died." Meyer discusses successively the incidence of human and animal botulism throughout the world, the distribution of *Cl. botulinum* in nature, and the foods and types of *Cl. botulinum* associated with human botulism. It is interesting to learn that, even with such a serious condition as botulism, in about 10% of recorded episodes in the U.S.A. the causative food was not determined and that bacteriological proof was furnished in only about a third of clinically recognized episodes. The risk of botulism exists wherever there are large numbers of type A, B, or E spores of *Cl. botulinum* in the soil, and Meyer suggests that surveys are especially needed of the extent to which these occur in the soils of Asia and Africa. It is estimated that in the Union of South Africa alone 100,000 cattle die of botulism every year. Animals reared on phosphorus-deficient veldt apparently crave the bones of carrion, which may be highly toxic from contamination with *Cl. botulinum*. So far as the human disease is concerned, the danger arises always from carelessness in the preparation and preservation of vegetable and animal foods. Many outbreaks have followed home canning of vegetables, and it is essential that all forms of food processing should be carefully controlled. Only in this way is it possible to eliminate completely the risk of botulism.

SICKLE CELLS AND SALMONELLAE

A curious association between sickle-cell anaemia and salmonella infection has now been reported in a number of cases. F. J. Hodges and J. F. Holt[1] mentioned having seen five cases of *Salmonella paratyphi B* osteomyelitis in patients with sickle-cell anaemia. L. W. Diggs and colleagues,[2] who in 1937 had been the first to state that patients with sickle-cell anaemia were particularly susceptible to bone infections, included in their series one patient with suppurative arthritis caused by that organism. Earlier in 1925 G. L. Carrington and W. C. Davison[3] described salmonella osteitis in a Negro child, but a test for sickling was not performed. J. Vandepitte and his colleagues[4] published in 1953 a series of five cases of salmonella osteitis in patients with sickle-cell anaemia observed in the Belgian Congo. They were not aware at the time of the reference to this association made by Hodges and Holt, but they collected five more isolated reports from the American literature, and one by B. Ehrenpreis and H. N. Schwinger,[5] who, in an article on the radiological changes in sickle-cell disease, had mentioned two observations of infection of the bones by salmonella. There was also one description of an infection of the tibia with *Salmonella enteritidis* in an Arab child suffering from a long-standing haemolytic anaemia, but no examination for sickling had been

[1] Brocklehurst, J. C., *Brit. med. J.*, 1957, **2**, 924.
[2] Meyer, K. F., *Bull. Wld Hlth Org.*, 1956, **15**, 281.

made.[6] Not all the observations are restricted to infections of the bones. J. and C. Lambotte-Legrand suggested in 1951[7] that carriers of the sickle-cell trait as a whole (not only patients with sickle-cell anaemia) seemed to have a higher incidence of salmonella infections than non-sicklers, and A. I. Chernoff and A. M. Josephson[8] described an aplastic crisis in a 6-months-old child with sickle-cell anaemia which had been precipitated by an infection with *Salmonella cholerae suis* without osteomyelitis. However, in ten further cases of sickle-cell anaemia complicated by salmonella infections only one showed no involvement of the bones. These include two single cases[9 10] and two series of four patients each.[11 12] Infections of the bone by salmonella are rare. Of a total of 2,000 salmonella cultures sent for identification to the New York Salmonella Center only three had been isolated from bone lesions.[13] Of 233 salmonella cultures isolated in the Belgian Congo five only came from bone infections,[14] and all these were associated with sickle-cell anaemia.[4] It seems therefore established that there is a highly significant association between salmonella osteitis and sickling. No clear distinction is drawn in all instances between sickle-cell anaemia and sickle-cell disease in general. Indeed, if the observations of the Lambotte-Legrands[7] and A. B. Raper[15] can be extended, it might turn out that carriers of the sickle-cell trait are also more liable to suffer from salmonella infections than non-sicklers, a situation which would have important consequences for our conceptions of the population dynamics of sickling.

Hodges and Holt[1] suggested that capillary thromboses in the gastro-intestinal tract might lower the resistance of patients with sickle-cell anaemia to invasion of the body by intestinal organisms, and E. W. Hook and his colleagues[12] point out that the general debility and disease of the spleen associated with sickle-cell anaemia might well interfere with the general defence mechanisms after the body had been invaded by the salmonella. Once the organism had entered the blood stream it would find favourable conditions in areas of ischaemia and necrosis which result from the blockage of small vessels by sickled cells. The recognition of salmonella infection of the bones can be difficult in sickle-cell anaemia. Fever, pain, and swelling of the limbs and joints are found in both diseases. On *x*-ray examination the bone infarction of sickle-cell anaemia and of osteomyelitis look very much alike. Even if the diagnosis of osteomyelitis is made it will be difficult to decide whether the infection is not streptococcal or staphylococcal. Hook and colleagues[12] emphasize the need for aspiration of the infected bone if the diagnosis is in doubt.

The most effective treatment of salmonella osteitis is chloramphenicol and surgical drainage. However the results are not dramatic. Holt and his colleagues found in one patient drug-sensitive organisms to persist in the tissues for months, and in another a recurrence of the osteomyelitis two years after the apparent healing of a previous infection.

[1] Hodges, F. J., and Holt, J. F., Editorial Comment, *Year Book of Radiology*, Chicago, 1951.
[2] Diggs, L. W., Pulliam, H. N., and King, J. C., *Sth. med. J.*, 1937, **30**, 249.
[3] Carrington, G. L., and Davison, W. C., *Bull. Johns Hopk. Hosp.*, 1925, **36**, 428.
[4] Vandepitte, J., Colaert, J., Lambotte-Legrand, J., Lambotte-Legrand, C., and Perin, F., *Ann. Soc. belge de Méd. trop.*, 1953, **33**, 511.
[5] Ehrenpreis, B., and Schwinger, H. N., *Amer. J. Roentgenol.*, 1952, **68**, 28.
[6] Giaccai, L., and Idriss, H., *J. Pediat.*, 1952, **41**, 73.
[7] Lambotte-Legrand, J., and Lambotte-Legrand, C., "L'anémie à hématies falciformes chez l'enfant indigène du Bas-Congo," *Mem. Inst. roy. Coll. Belge*, 1951, Brussels.
[8] Chernoff, A. I., and Josephson, A. M., *Amer. J. Dis. Child.*, 1951, **82**, 310.
[9] Goldenberg, I. S., *Surgery*, 1955, **38**, 758.
[10] Ellenbogen, N. C., Raim, J., and Grossman, L., *Amer. J. Dis. Child.*, 1955, **90**, 275.
[11] Hughes, J. G., and Carroll, D. S., *Pediatrics*, 1957, **19**, 184.
[12] Hook, E. W., Campbell, C. G., Weens, H. S., and Cooper, G. R., *New Engl. J. Med.*, 1957, **257**, 403.
[13] Seligmann, E., Saphra, I., and Wassermann, M., *J. Immunol.*, 1946, **54**, 69.
[14] Van Oye, E., *Ann. Soc. belge Méd. trop.*, 1952, **32**, 179.
[15] Raper, A. B., *Brit. med. J.*, 1956, **1**, 965.

DEATHS FROM CIRRHOSIS OF THE LIVER

According to statistics prepared by the Metropolitan Life Insurance Company of New York, cirrhosis of the liver is now one of the ten leading causes of death in the United States.[1] In fact, in late middle age there are only three diseases with a higher mortality—namely, heart disease, cancer, and cerebral haemorrhage. There has apparently been a steady rise in the number of deaths from cirrhosis since the nineteen-thirties, and the national rate for the United States now averages 10 per 100,000 population. Part of the increase is undoubtedly related to the falling death rate from infectious diseases, and improved methods of diagnosis may be a further contributory factor, but it seems justifiable to assume that there has been a real increase in the number of cases of cirrhosis. If this is so, then it is a disturbing fact, since the causes of portal (Laennec's) cirrhosis, the only common variety, remain as obscure to-day as they have always been.

Alcohol and infective hepatitis are at present the only two unequivocal aetiological factors, but there is no evidence that either is responsible for the current trend. The facts are against such a view. There has been no sharp rise in mortality rate in the post-war years, such as might have been expected as an aftermath of the great wartime epidemics of hepatitis. Moreover, the mortality rate in England and Wales, where infective hepatitis is a much commoner cause of cirrhosis than alcohol, has remained constant at 2.6 per 100,000 population since 1952.[2] Over a quarter of the deaths from cirrhosis in the United States in 1956 were attributed to alcohol, a figure which should be compared with the results of the survey by O. D. Ratnoff and A. J. Patek,[3] in which alcoholism accounted for more than 50% of the cases. The discrepancy between these two findings is almost certainly due to incomplete reporting on death certificates rather than to any true decrease in the incidence of alcoholism as a causal factor.

Two features emerge from all investigations into the incidence of portal cirrhosis in temperate climates. The first is the marked difference in the importance of alcohol and infective hepatitis as aetiological agents in different countries,[4] and the second is the fact that no antecedent cause is demonstrable in something like 40% of all patients with portal cirrhosis. There is surely a need for further epidemiological studies of this serious and by no means uncommon disease.

[1] *Statistical Bulletin of the Metropolitan Life Insurance Company*, 1957, **38**, 8
[2] *Registrar General's Statistical Review of England and Wales for 1956*, 1957 H.M.S.O., London.
[3] Ratnoff, O. D., and Patek, A. J., Jun., *Medicine*, 1942, **21**, 207.
[4] *See* Sherlock, S., *Diseases of the Liver and Biliary System*, 1955, Oxford.