severe disability were working. They attributed this favourable situation to the patients' robust spirit and to the effectiveness of social and physical treatment.

A very different situation had been described in 1964 by D. P. Agle,[2] who in a study of 16 haemophiliacs found that eight had recurrent depression or anxiety of incapacitating severity. Nine seemed to invite physical risks and this was interpreted as rebellion against maternal over-protection.

Neurotic reactions are so common and their causation so complex that it is important to determine to what extent their association with other conditions might be due to chance. The association between risk taking and haemophilia becomes less certain when we remind ourselves that many normal young men enjoy taking risks. For these reasons it is desirable to compare the population under scrutiny with a control population which as nearly as possible approaches the ideal of differing only in lacking the suspect variable.

Although neither of the above studies used control groups their very different findings suggest that if there is a link between haemophilia and the development of a neurotic personality then it is only very indirect. This accords with the views of E. Guttmann and W. Mayer-Gross,[3] who emphasized that the emotional response to lifelong deformity, a common complication of severe haemophilia, is very variable—ranging from unhealthy reactions such as self-pity and resentment to a cheerful determination to overcome adversity.

The requirements for healthy physical and emotional development in the haemophilic child sometimes conflict. Thus admission to hospital deprives him of his mother's company, and she must be encouraged to visit frequently and if feasible to help in his day-to-day care.[4] Rosemary Biggs and R. G. Macfarlane[5] have discussed in detail the problems of minimizing the risk of deformity by protecting the child from physical injury, but at the same time encouraging him to be active and self-reliant, and of providing suitable schooling and of educating the parents to cope effectively with the demands which the illness makes.[5] Arrangements should be made for the parents to bring the child for prompt treatment when joint bleeding occurs, for A. M. Ali and her colleagues[6] showed that the incidence of permanent crippling could be reduced to about a fifth by immediate plasma therapy.

The first report[7] of the Haemophilia Society of a survey of its members points out that "finding and keeping suitable employment is the greatest problem facing the adult haemophiliac." The society sent questionaries to 640 members aged 17 years and over. Completed replies were received from 487 men, and the data obtained were supplemented by 143 home visits made by an officer of the society. Though the information obtained may not be representative of all haemophiliacs, nevertheless the report contains much food for thought. Nearly one-fifth of the group were unemployed, a rate about four times that in the general population, and nearly half of these had been out of work for five years or more. The experience of individual patients suggested that disablement resettlement officers and prospective employers have a considerable fear of haemophilia, an impression borne out by experience in haemophilia centres. Doctors can help considerably by explaining that haemophiliacs are not in danger of serious bleeding from very trivial injury as is commonly supposed, and also that modern plasma treatment greatly reduces the length of absences from work following spontaneous bleeds into joints and muscles.

Unemployment was three times as common among those who were permanently crippled as among those who were not. This again emphasizes the importance of early and vigorous treatment for bleeding episodes. The report also brings out the relation between employability and loss of schooling. Of 165 men who had lost up to a quarter of their schooling in childhood only 7% were unemployed; but of 89 men who had lost more than half their schooling 40% were unemployed. Education is particularly important for haemophiliacs because of the necessarily restricted range of jobs they can do. The survey showed that nevertheless one-quarter of those questioned were in manual occupations—which is a much smaller proportion than in the population as a whole—but those in non-manual employment were more successful at keeping their jobs. In addition it was found that over two-thirds of the non-manually employed attended haemophilia centres, whereas only just over half of the manual workers and unemployed attended. It is principally at haemophilia centres that urgent plasma therapy can be obtained.

The cost of the unemployment of haemophiliacs is illustrated by the calculation that the 65 men who had been unemployed for over a year would have been paid some £13,500 per annum in National Insurance benefits; and in addition to this there would have been the loss to the community of their working capacity.

Sewage from Boats

One of the more agreeable results of the rise in standards of living which has occurred since the war is the increase in the number of pleasure boats. Many of those who own or hire them live aboard for prolonged periods, and so have to dispose of refuse, sullage water, and sewage.

Small craft sailing in the open sea generally dispose of their waste directly into it, in the same way as larger vessels, and this is unobjectionable. A similar tolerance on many tidal rivers and estuaries is inevitable when they are already heavily polluted. Until there is proper treatment of the sewage discharging into these waterways regulation of the pleasure craft that use them would be futile.

Many people believe that higher standards are expected—and exacted—from boats using inland waterways and those tidal waters that are not already polluted by other means. In practice, what is permitted varies enormously from place to place, and on many waterways there is no effective control at all of the discharge of sewage from boats. The British Waterways Board, for example, equip their own canal boats with lavatories which discharge directly into the water. Local authorities have no powers to make regulations for pleasure craft, which are specifically excluded from those provisions of the public health acts that deal with canal boats.[1]

[1] Bronks, I. G., and Blackburn, E. K., *Brit. J. prev. soc. Med.*, 1968, 22, 68.
[2] Agle, D. P., *Arch. intern. Med.*, 1964, 114, 76.
[3] Guttmann, E., and Mayer-Gross, W., *Lancet*, 1940, 2, 185.
[4] Bowlby, J., *Child Care and the Growth of Love*, 1965, 2nd ed. (pp. 175 f). Harmondsworth.
[5] Biggs, R., and Macfarlane, R. G. (editors), *Treatment of Haemophilia and Other Coagulation Disorders*, 1966, chapters IV and XVII. Oxford.
[6] Ali, A. M., Gandy, R. H., Britten, M. I., and Dormandy, K. M., *Brit. med J.*, 1967, 3, 828.
[7] *Survey of Adult Haemophiliacs: Report on Employment and Unemployment*, 1968. Haemophilia Society, London.

[1] *Public Health*, 1968, 82, 187.
[2] *Thames Conservancy (Navigation and General) Bye Laws*, 1934. London.
[3] *Thames Conservancy Launch Digest*, 1968. London.

In contrast very strict control is exercised by the Thames Conservancy on vessels using the river above Teddington lock.[2] A boat fitted with a lavatory which discharges overboard must have it sealed by the Conservators' Officer to prevent its discharging to the river. It is an offence for those using the river to throw rubbish into the water or leave it on the banks or otherwise to pollute the water. As a corollary to these regulations, the Conservancy provides for the disposal of sewage and rubbish at various points along the river. Most of these facilities are at the Conservancy's own locks.[3]

There is no doubt that a uniform code for the whole country is needed, and that the standards it imposes should be as high as those of the Thames Conservancy; but legislation will be necessary. Meanwhile the various authorities having control of the waterways could make a start by improving the standards of their own craft and by providing disposal facilities. They could then exact the same improvement and the same provision of disposal facilities from firms hiring out craft on their waters. The way would then be clear for the introduction of national regulations. Where it is proposed to build marinas for yachts, local authorities should insist on the adoption of suitable regulations as a condition of their permission to construct. Portsmouth has draft regulations for its own proposed yacht marina, and these provide a useful model which could be adapted to local circumstances.[1]

Probenecid and Renal Failure

When a drug has to be given continuously for many months or years it should clearly have a low degree of toxicity, particularly as other drugs may have to be given with it. During the 16 years or so that probenecid (Benemid) has been in use in Great Britain it has proved to be a relatively safe and effective uricosuric agent. At the start of treatment, particularly if it is given in full dosage from the beginning, acute attacks of gout may be precipitated. Though generally well tolerated, gastrointestinal irritation may occasionally occur. Goodman and Gilman[1] report an incidence of at least 2% rising with larger dosage, and an incidence of skin reactions at 2–4%. Sensitivity reactions occur rarely but may be severe. On the whole, toxic effects have not been a great problem and have remained relatively uncommon.

The possibility that long-term therapy might damage the kidney was seriously considered from the start. Renal colic[2] was reported, and in three cases nephrotic syndrome occurred but cleared up when the drug was withdrawn.[3] One patient developed the nephrotic syndrome after three months on probenecid, recovered on withdrawal of the drug, and relapsed on starting it again.[4] More recently J. T. Scott and P. K. O'Brien[5] have reported two patients in whom oedema and proteinuria occurred during treatment with probenecid. Both were elderly men, the first uraemic, the second hypertensive. In the first patient, a severe, long-standing case of tophaceous gout with chronically raised blood urea, the syndrome appeared after a year on 1 g. daily of probenecid. Massive oedema developed, with albuminuria, hypercholesterolaemia, and reduction in serum albumin. The patient recovered rapidly on stopping the drug, and has remained well since 1964 on daily allopurinol. In this case the blood urea did not rise during treatment but remained raised at about the pretreatment level of 80 mg. per 100 ml. The second had had only two acute attacks of gout. After 16 months on 1 g. of probenecid daily he developed progressive anorexia, drowsiness, oedema of the limbs and trunk, heavy proteinuria, and a steadily rising blood level of urea. Treatment was continued, and he died a month later. Necropsy disclosed large pale kidneys showing widespread dilatation of cortical tubules with flattening of the lining of the epithelium. Crystals were visible in several distal convoluted and collecting tubules, and in the interstitial tissues of the medulla. The authors wisely state that there is no more than a possibility of causal association with the drug, for proteinuria and renal failure develop occasionally in patients with gout untreated by uricosuric agents. Cases such as these are apparently rare, and, when the symptoms are due to the drug, seem usually to end in recovery if treatment is stopped.

[1] Goodman, L. S., and Gilman, A., *The Pharmacological Basis of Therapeutics*, 1965, 3rd ed., p. 874. New York.
[2] Boger, W. P., and Strickland, S. C., *Lancet*, 1954, **1**, 420.
[3] Ferris, T. F., Morgan, W. S., and Levitin, H., *New Engl. J. Med.*, 1961, **265**, 381.
[4] Sokol, A., Bashner, M. H., and Okun, R., *J. Amer. med. Ass.*, 1967, **199**, 43.
[5] Scott, J. T., and O'Brien, P. K., *Ann. rheum. Dis.*, 1968, **27**, 249.

Löffler's Syndrome

In 1932 W. Löffler[1 2] first drew attention to a syndrome in which infiltration of the lung fields in the chest *x*-ray film and eosinophilia were the main features. The radiological abnormalities were variable, being unilateral or bilateral, small and discrete or large and fluffy, restricted or extensive, while the proportion of eosinophil leucocytes ranged from 6 to 66%. Characteristically both these features were transient, lasting usually only for about two weeks. From the beginning infection with *Ascaris lumbricoides* was suspected as a cause and salads during summertime the mode of infection. Since then these findings—often associated with respiratory symptoms, such as cough and wheeze—have been observed following exposure to allergens or certain drugs.

A. P. Gelpi and A. Mustafa[3] have recently reported on 108 patients with this syndrome from Dhahran in Saudi Arabia. These patients, who were mainly adults, had had a transient illness in the early part of the year consisting of pyrexia, cough, wheeze, and rash. On investigation they were found to have pulmonary infiltration and considerable eosinophilia, and in some *A. lumbricoides* larvae were found in the sputum; in others ova were later found in the stools, which had been free of them during the acute illness. Gelpi and Mustafa also observed a similar illness in patients suffering from schistosomiasis in whom specific treatment had been started a few days previously.

H. French[4] in 1909 was probably the first to describe the condition which in 1943 was named "tropical eosinophilia" by R. J. Weingarten.[5] There are four major components of this syndrome: firstly, respiratory symptoms consisting of cough, exertional dyspnoea, and wheeze; secondly, an

[1] Löffler, W., *Beitr. klin. Tuberk.*, 1932, **79**, 368.
[2] Löffler, W., *Int. Arch. Allergy*, 1956, **8**, 54.
[3] Gelpi, A. P., and Mustafa, A., *Amer. J. Med.*, 1968, **44**, 377.
[4] French, H., *Guy's Hosp. Gazette*, 1909, **23**, 533.
[5] Weingarten, R. J., *Lancet*, 1943, **1**, 103.
[6] Donohugh, D. L., *New Engl. J. Med.*, 1963, **269**, 1357.
[7] Buckley, J. J. C., *E. Afr. med. J.*, 1958, **35**, 493.
[8] Beaver, P. C., Snyder, C. H., Carrera, G. M., Dent, J. H., and Lafferty, J. W., *Pediatrics*, 1952, **9**, 7.