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.

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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

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