They can also comment on area plans for the Service, including such things as hospital closures. Hospital waiting lists and visiting times are another legitimate interest and the councils can inspect clinics, health centres, and hospitals as well as look at domiciliary services. This gives their 20 or so members, appointed largely by local authorities and voluntary organizations, quite a powerful voice in the N.H.S. If the profession can make allies of these new bodies then the consequences for the N.H.S. might be surprisingly constructive. But should the councils overplay their inspectorial role then the outcome would be less happy.

[1] *National Health Service: The Administrative Structure of the Medical and Related Services in England and Wales.* London, H.M.S.O., 1968.
[2] *National Health Service Reorganization: Consultative Document.* London, D.H.S.S., 1971.
[3] *British Medical Journal Supplement*, 1971, **3**, 101.

Analgesic Nephropathy or Phenacetin Poisoning

The argument still continues over the relative importance of various analgesic drugs in the production of progressive renal damage, more than 20 years after the first descriptions of this association by Spuhler and Zollinger.[1] The clinical, radiological, and pathological features of analgesic nephropathy are now relatively clearly defined.[2 3] Recurrent attacks occur of fever, dysuria, and the passage of large numbers of leucocytes in the urine, often with fragments of renal papilla; the episodes closely resemble acute urinary infection, and they lead eventually to impaired renal function. Papillary necrosis and severe interstitial fibrosis in both renal cortex and medulla are the major pathological changes.[4]

The frequency of recognition of excessive and prolonged analgesic intake depends entirely on a high index of suspicion; persistence in direct questioning of both patients and their relatives may be necessary, as all the drugs used are common household remedies, freely available, and their use is often denied. The incidence estimated at 450 cases each year by Koutsaimanis and de Wardener,[3] or 10% of all cases of renal failure,[5] may be considerably less than the real figure, since many patients with a similar clinical course and with no satisfactory explanation for their renal failure present to nephrologists, and the histological changes of non-specific interstitial fibrosis are not uncommon on renal biopsy in chronic renal disease.

So there is little argument as to the existence of the condition though epidemiological studies of the effects of analgesic consumption have led to conflicting information. In South Wales[6] and the U.S.A.[7] a total of 517 women who took more than 1 g per day of analgesic drugs were reported to have no greater incidence of reduced renal function than a total of 9,192 controls who did not admit to taking these substances. Tests on 623 Swiss factory workers who took phenacetin showed that they had twice the incidence of proteinuria and five times that of reduced concentrating ability than was found in others taking alternative analgesics or no drugs.[8] Phenacetin has been regarded as the major factor in the many analgesic mixtures reported to have caused the condition, but the relatively huge amounts of several kilograms required and the prolonged period over which tablets or powders have to be consumed has led some research teams to incriminate aspirin[9] or impurities in the phenacetin[10] rather than phenacetin itself. Aspirin does produce papillary necrosis in rats, especially after dehydration, in smaller dosage and more readily than does phenacetin;[11] it also appears to reduce glomerular filtration rate[12] and to increase tubular cell excretion in man.[13]

The consumption of aspirin in the community is enormous. Two studies reported in this issue (pp. 593 and 597) by a team of New Zealand physicians and by Dr. A. F. Macklon and his colleagues describe tests of renal function on patients treated with large doses of aspirin for long periods. The results do not appear to differ from accepted normal values, and support earlier work by Sørenson:[14] patients given up to 5 kg of aspirin show no evidence of any convincing association between progressive renal impairment and aspirin dosage. The two recent studies both conclude that the evidence against aspirin is extremely weak and that no convincing reason exists to restrict the sales on the basis of nephrotoxicity. The Newcastle finding of unchanging renal function two years after further consumption of aspirin conflicts with the reports[15] from Australia of relapses in patients with analgesic nephropathy who consumed aspirin mixtures without phenacetin.

Direct evidence in this area of disagreement is almost impossible to obtain. The encouraging reduction in the incidence of analgesic nephropathy reported after restriction of phenacetin in Scandinavia[16] and Scotland,[17] and the very recent decision by the Department of Health and Social Security to restrict the sale of phenacetin mixtures to pharmacists from June 1974 and to put it on prescription alone from January 1975 may settle the dispute, since closer control and reduction of phenacetin intake should eventually result in the disappearance of the condition if it is really due to chronic phenacetin poisoning. There will still be a need, however, to warn patients with unexplained renal damage of the potential hazards of analgesic drugs in large prolonged dosage and also to encourage greater awareness of the association of these drugs with urinary symptoms by their medical advisers.

[1] Spuhler, O., and Zollinger, H. U., *Zeitzschrift fur Klinische Medizin*, 1953, **151**, 1.
[2] *British Medical Journal*, 1970, **4**, 125.
[3] Koutsaimanis, K. G., and de Wardener, H. D., *British Medical Journal*, 1970, **4**, 131.
[4] Burry, A. F., *Nephron*, 1968, **5**, 185.
[5] Cove Smith, J. R., and Knapp, M. S., *Lancet*, 1973, **2**, 70.
[6] Waters, W. E., Elwood, P. C., and Assher, A. W., *Lancet*, 1973, **1**, 341.
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[14] Sørenson, A. W. S., *Nephron*, 1966, **3**, 366.
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Assessment of Kidney Transplantation

The eleventh report from the Renal Transplant Registry[1] is based on 12,389 renal transplants performed from 1951 to the end of 1972. Of these 11,264 were first transplants, 1,019 secoond perations, and 106 third and subsequent transplants. There were 10,357 patients whose follow-up was regarded as adequate, and of these 4,934 (47.6%) were alive with func-