defect, possibly through the lack of some protective enzyme. It has also been suggested that smoking, which statistically is as closely related to deaths from bladder cancer as it is to deaths from lung cancer,⁹ may effect its carcinogenic action through the intermediary of an alteration in tryptophan metabolism.⁷

What, if anything, have the tryptophan metabolites in common with the identified industrial carcinogens? Essentially, it seems that they share a common basic structure of an amine group linked to a benzene ring—they are aminophenols. But so are a great many supposedly innocuous substances, including many licit additives and dyestuffs in food as well as drugs in everyday use such as phenacetin, which is excreted largely as a para-aminophenol.¹⁰

Recently disturbing reports have been published that phenacetin abuse may not only give rise to nephropathy and necrosis of the renal papillae but also to cancer of the cells lining the renal pelvis.^{10 11} The most recent study comes from a small hospital in south Sweden serving the little town of Huskvarna. Here the main industry is a small-arms factory, where, ever since the influenza epidemic of 1918, it has been a habit for the people to take relatively large quantities of "Hjorton's powder," which contains phenacetin, phenazone, and caffeine. Of the 15 patients in their hospital who had urothelial cancer of the renal pelvis ten were Huskvarna phenacetin takers and all but two had phenacetin nephropathy and renal papillary necrosis.¹¹

Why the carcinoma arose in the urothelium of the kidney rather than in the more usual place, the urothelium of the bladder, is a question which is of considerable theoretical interest. Carcinogenesis from chemicals in the urinary tract seems to demand two things—initiation and development or cocarcinogenesis. Hyperplasia in response to irritation of many different types, from stones to paraffin wax pellets, seems to speed the development of carcinoma.¹² The fact that the phenacetin produced scarring, calcification, and papillary necrosis, and so presumably irritation in the renal pelvis, may be the reason why cancer appeared there before the bladder. Unfortunately we do not know how many of the Huskvarna victims also smoked.

Wigs and Waste

A proposed increase in patients' payments for teeth and spectacles can threaten a political crisis, but the cost of N.H.S. wigs has provoked little comment since the early days of the Health Service. This is surprising, for their initial cost and maintenance may amount to a charge of over £1m. a year on the taxpayer. Figures obtained by the B.M.A. Dermatologists Group showed that the average annual cost in the period 1965–7 for two regional hospitals serving populations of 300,000 and 100,000 was £6,778 and £2,432 respectively.

Patients are usually supplied with two wigs, which cost about £33 each. Partial transformations cost somewhat less, but they are prescribed infrequently. An accompanying leaflet, *Taking Care of Your Wig*, states that one of the two wigs should be cleaned and dressed every month. Each service costs 18s., and thus the cost of maintenance may amount to about half of the total cost of new wigs supplied. Some hospital groups are spending over £2,000 a year on the cleaning and maintenance of wigs.

The main reason why the expenditure on wigs is so heavy is the large number of elderly women with thinning hair (involutional alopecia) who are referred to dermatologists in the belief that free wigs are their right. These patients are not eligible for wigs if the regulations are strictly interpreted. Wigs may be provided under the N.H.S. only for baldness due to congenital dystrophy of the skin; alopecia totalis; alopecia areata, severe and long standing; extensive scarring following trauma, x-irradiation, or an inflammatory condition; or in cases of illness or treatment of illness where baldness, though not permanent, is likely to be prolonged. The wig must be considered necessary by a consultant.

In practice it is difficult for a dermatologist to refuse a wig to a distraught grandmother with an incipient male-type tonsure who has been told that she can obtain a wig from the hospital free of charge. But unless the prescribing of wigs is restricted to the conditions specified in the regulations they will continue to be supplied in ever increasing numbers as our aged population grows. Costs could be reduced substantially if the consultant could at his discretion order a cheaper wig—a nylon-acrilan one costs little over £3—instead of the standard, expensive one.

One of the faults of a free health service is that the easy way out of a difficult situation is often the most expensive one. A nation which spends £1m. a year on wigs has got its priorities wrong.

Neurological Sequelae of Partial Gastrectomy

Though partial gastrectomy has been blamed for a long list of troubles, the risks of their developing are often considered acceptable. Postprandial disturbances are usually an improvement on the pain that the patient experienced before his operation, and symptoms that may develop later owing to deficiency states are as a rule at least partially responsive to replacement therapy. But some sequelae occur so rarely and so long after partial gastrectomy that the significance of the operation is easily overlooked. Certain neurological disturbances come into this category.

In Birmingham a special interest has been taken in the complications and metabolic consequences of partial gastrectomy.¹ J. A. Williams and his colleagues² have recently extended this experience to an analysis of neurological disease, occasionally occurring many years after partial gastrectomy.

Reports have appeared in the past of subacute combined degeneration of the cord many years after partial gastrectomy.^{3 4} Wider recognition has been given in Scandinavia to "myelopathy" associated with low serum levels of vitamin B₁₂ after this operation.⁵ Patients with low

¹ Stammers, F. A. R., and Williams, J. A. (eds.), *Partial Gastrectomy, Complications and Metabolic Consequences*, 1963. London, Butterworths.

² Williams, J. A., Hall, G. S., Thompson, A. G., and Cooke, W. T., *British Medical Journal*, 1969, 3, 210.

³ Knox, J. D. E., and Delamore, I. W., *British Medical Journal*, 1960, 2, 1494.

⁴ Weir, D. G., and Gatenby, P. B. B., *British Medical Journal*, 1963, 2, 1175.

⁵ Olivarius, B. de F., and Roos, D., *Lancet*, 1965, 2, 1298.

⁶ Wits, L. J., *The Stomach and Anaemia*, 1966. London, Athlone Press.

⁷ Deller, D. J., *Australasian Annals of Medicine*, 1962, 2, 235.

⁸ Cooke, W. T., Johnson, A. G., and Woolf, A. L., *Brain*, 1966, 89, 663.

⁹ Cooke, W. T., and Smith, W. T., *Brain*, 1966, 89, 683.

normal or subnormal levels of serum vitamin B₁₂ after partial gastrectomy are much more common than those with overt megaloblastic anaemia.⁶ The vitamin-B₁₂ deficiency is due to a failure of secretion of intrinsic factor as a consequence of atrophic gastritis in the gastric remnant,⁷ and only in a small minority of cases is a blind-loop syndrome to blame. In the Scandinavian cases of "myelopathy" the average time elapsing between operation and the onset of neurological symptoms was more than 10 years. Such a long latent interval need occasion no surprise if vitamin-B₁₂ deficiency is responsible, for impairment of intrinsic factor secretion would take time to develop from atrophic gastritis in the gastric remnant, and the hepatic store of vitamin B₁₂ would also have to become depleted.

The recent report from Birmingham² suggests that neurological disease after gastrectomy may be more complex than was thought at first and that vitamin-B₁₂ deficiency may not invariably be responsible. Serum levels of vitamin B₁₂ were below normal, and there was evidence of malabsorption of the vitamin in most patients, but some had normal serum levels and normal absorption.

Clinically all patients complained of difficulty in walking, with unsteadiness and muscular weakness; most also had paraesthesiae or numbness and a feeling of malaise or depression. Muscle weakness and abnormal neurological signs in the legs were found in all of them, and in 10 of the 14 reported there was gross muscle wasting. Loss of vibration sense occurred in all, and loss of proprioception and rombergism was noted in the majority. In those patients studied by muscle biopsy microscopical abnormalities of the terminal motor innervation were recognized as similar to those associated with nutritional neuropathy in coeliac disease of adults.⁸ Only three of the patients responded well to vitamin B₁₂, and in them the clinical findings were typical of subacute combined degeneration of the cord. The neurological disease in the others may have been more like that which complicates adult coeliac disease,⁹ the aetiology of which is unknown.

The importance of recognizing the significance of symptoms in these rare patients is obvious. Unfortunately mental disturbances may increase the difficulties of recognition and management. Mental symptoms have been associated with vitamin B₁₂ deficiency in some patients, and "psychopathic disturbances" were also a feature of several of the patients unresponsive to vitamin B₁₂ in the Birmingham series.²

Prolonged Pregnancy

About one pregnancy in ten is prolonged beyond the 42nd week, and in such cases perinatal mortality is nearly double that at 37-41 weeks.¹⁻⁵ This is largely the result of a progressive inability of the placenta to support fetal nutrition and oxygenation in late pregnancy. Even in otherwise normal pregnancies hypoxia may reach a critical level by the end of the 42nd week. Prolonged pregnancy is also associated with an increased incidence of difficult labour. Birth trauma, pulmonary disorders, and failure to thrive create problems for the paediatrician—M. A. Zwerdling⁴ showed that the mortality rate in post-term infants was higher than average for at least the first two years of life, with the "small for dates" baby at particular risk. Surprisingly, no long-term sequelae have yet been found, as judged by studies of growth and intelligence.

Recently, N. A. Beischer and his colleagues⁵⁻⁷ have made an extensive review of the problems of prolonged pregnancy. In particular they have reassessed the value of urinary oestriol assays and examination of the liquor amnii as prognostic indices. They confirmed earlier reports which showed a good correlation between falling oestriol levels and other clinical evidence of failing placental function. Oligohydramnios and meconium staining of the liquor were associated with placental insufficiency and a high incidence of fetal distress in labour. Retrospective studies such as these have added much to our knowledge of the nature and degree of risk associated with prolonged pregnancy, but for various reasons attempts to apply the same techniques to prospective assessment in the individual case have generally been less successful.

Not the least of the many problems facing the clinician is the estimation of the duration of pregnancy; for doubt may remain even after consideration of menstrual dates and cycle, uterine and fetal size, liquor quantity and quality, and radiological and biochemical data. However, in such an assessment those features which concern chronological maturity are less important than those which more specifically reflect fetoplacental dysfunction. The commonly used methods of practical value in the assessment of fetal well-being antenatally and during labour were reviewed last year by R. W. Beard.⁸ He confirmed the value of oestriol assays, provided serial observations are possible, but questioned the value of amniocentesis as at present applied.

These techniques are relatively time-consuming, and this must limit their practical application. Few units have facilities for full assessment of all women with prolonged pregnancy, but at least those patients at exceptional risk can be singled out and given special attention.

What, then, of the majority of patients with post-dated pregnancies? After the early work of J. Walker and E. P. N. Turnbull⁹ a liberal policy of induction of labour was pursued in many units, but the response in terms of fetal loss has been disappointing.¹⁰ Some consider the hazards of induction of labour to be greater than those of allowing the pregnancy to continue.¹¹ However, this is likely to be so only if arbitrary chronological criteria are adopted and if clinical assessment and the performance of induction are delegated entirely to junior staff.

Even when selective induction of labour is practised the fetus is already at risk to some degree. In prolonged pregnancy fetal death may occur before labour commences, but the greatest hazard is associated with anoxia during labour, especially if mechanical problems supervene. If placental insufficiency is severe, induction of labour is unlikely to save

¹ Walker, J., *Journal of Obstetrics and Gynaecology of the British Empire*, 1954, **61**, 162.

² Gibson, G. B., *British Medical Journal*, 1955, **2**, 715.

³ Browne, J. C. McC., *Journal of the American Medical Association*, 1963, **186**, 1047.

⁴ Zwerdling, M. A., *Pediatrics*, 1967, **40**, 202.

⁵ Beischer, N. A., Brown, J. B., and Townsend, L., *American Journal of Obstetrics and Gynecology*, 1969, **103**, 496.

⁶ Beischer, N. A., Brown, J. B., Smith, M. A., and Townsend, L., *American Journal of Obstetrics and Gynecology*, 1969, **103**, 483.

⁷ Beischer, N. A., Evans, J. H., and Townsend, L., *American Journal of Obstetrics and Gynecology*, 1969, **103**, 476.

⁸ Beard, R. W., *Proceedings of the Royal Society of Medicine*, 1968, **61**, 1247.

⁹ Walker, J., and Turnbull, E. P. N., *Lancet*, 1953, **2**, 312.

¹⁰ Browne, F. J., *British Medical Journal*, 1957, **1**, 851.

¹¹ Gibberd, G. F., *Lancet*, 1958, **1**, 64.

¹² Huntingford, P. J., and Pendleton, H. J., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1969, **76**, 586.

¹³ Bishop, E. H., *Clinical Obstetrics and Gynecology*, 1968, **11**, 1154.

¹⁴ Saling, E., *Foetal and Neonatal Hypoxia*, 1968. London, Arnold.