are little affected. Schatz *et al* also found that in patients sensitive to 5 TU and receiving corticosteroids on alternate days there was no correlation between dose and skin reactivity, confirming that alternate-day treatment does not suppress tuberculin skin-tests.[7]

We may conclude, then, that fear of reactivating tuberculosis need not affect our decisions about using corticosteroids in treating asthma and that there is no indication for prophylactic chemotherapy because of positive tuberculin reactions alone. In the light of these later studies, recommendations in line with those of Mayfield[1] in 1962 may still be made. Before long-term corticosteroid treatment for asthma (or other disease) is started, a search for evidence of tuberculosis, including a chest radiograph, should be made, If such evidence is found, and the disease is judged to be active, it can be treated concurrently with the use of corticosteroids for the presenting disease. If evidence suggesting inactive tuberculosis is found, the decision whether to treat or to undertake careful surveillance by periodic chest radiography should be made on the usual criteria.

[1] Mayfield, R B, *Tubercle*, 1962, **43**, 55.
[2] Corp, R F, *et al*, *American Review of Respiratory Diseases*, 1965, **91**, 297.
[3] American Thoracic Society, *American Review of Respiratory Diseases*, 1967, **96**, 558.
[4] Smith, J P, and Sharer, L, *American Review of Respiratory Diseases*, 1970, **102**, 821.
[5] Smyllie, H C, and Connolly, C K, *Thorax*, 1968, **23**, 571.
[6] Lieberman, P, Patterson, R, and Kunske, R, *Journal of Allergy and Clinical Immunology*, 1972, **49**, 329.
[7] Schatz, M, *et al*, *Annals of Internal Medicine*, 1976, **84**, 261.
[8] Citron, K M, and Scadding, J G, *Quarterly Journal of Medicine*, 1957, **26**, 277.

Management of diabetic pregnancy

Shortly after the turn of the century the maternal mortality[1] in diabetic women was as high as 27%. Despite the great improvement achieved with insulin, perinatal mortality remained close to 50% and was still 40% in 26 British teaching hospitals in the 1940s.[2] Since then the outlook for the fetus of the diabetic mother has changed enormously: in the more specialised units in teaching hospitals,[3] and some non-teaching ones,[4] perinatal mortality is now close to 10%. Nevertheless, this is still about five times that in non-diabetic mothers. These are rough statistics, and other circumstances have changed. Fifty years ago a pregnant diabetic had overt diabetes—though at that time none could have been insulin-dependent for long and be still alive. Nowadays many pregnant diabetics have mild disease treated by diet alone, though others have had insulin-dependent diabetes for 10 to 15 years.

Ideally, in managing diabetic pregnancy one or more interested obstetricians and physicians should work together in a single combined clinic. The obstetrician's main role is to decide the stage of gestation accurately, aided by sonar scanning, so that the time of delivery can be planned successfully and major fetal abnormality be detected. The physician must achieve painstakingly accurate control of the mother's blood sugar throughout the pregnancy, especially in the last trimester. Under such good conditions which factors govern the success of the pregnancy? Several prognostic indices have been described. That devised by White,[5] revised in 1965,[6] depends on factors present before pregnancy—such as the dependence on insulin, age of onset, and duration, as well as on vascular, renal, and ocular complications of the disease. An alternative assessment devised by Pedersen and Pedersen[8] depends on factors becoming evident during pregnancy. Their "prognostically bad signs during pregnancy" are clinical pyelonephritis, precoma or severe acidosis, toxaemia, and being a "neglector"—not reporting for treatment more than 60 days before term. In the White classification the fetal survival varied from 100% in class A to 0% in F (with diabetic nephropathy), and in the Pedersen classification the survival rate was 68·5% when one or more bad signs were present and 92% when they were absent. The most accurate assessment of the fetal prognosis comes from applying both the White and the Pedersen classification to the individual mother.

The single and most valuable treatment that can be offered to pregnant women is really good control of the blood sugar. According to the hyperglycaemia-hyperinsulinism theory it is the passage of excess glucose across the placenta (virtually impervious to insulin[9]) that stimulates islet cell hyperplasia in the fetal pancreas. This in turn may be why babies are born large and fat and why they tend to become hypoglycaemic in the first two days or so of life. Ketosis in the mother is apt to cause intrauterine death, but mild maternal hypoglycaemia appears not to increase the fetal risk.[10]

Obese "chemical" diabetics diagnosed for the first time in pregnancy can usually be managed well by diet alone, but as in women with more severe diabetes, delivery should be completed before term. Many would treat a non-obese chemical diabetic with insulin during pregnancy, knowing that the diabetes tends to worsen. On the other hand, treatment with chlorpropamide (in a dose not exceeding 100 mg a day) can reverse chemical diabetes during pregnancy without harm to the fetus.[11] If the mother was having only one injection of insulin a day before pregnancy began it is better to change her to two doses (including soluble insulin twice a day or occasionally thrice daily), for the biological half-life of insulin falls in pregnancy.[12] The mother needs to be told that good control of her blood sugar is of overriding importance during pregnancy, and she will almost certainly need further dietary and general diabetic training. This is a challenge that many such women willingly accept while they are pregnant for the sake of their baby, however lax they are at other times. The fact that the renal threshold of glucose falls in pregnancy itself tends to improve the control of the blood sugar based on tests for sugar in the urine. Sometimes the threshold falls so far that it may be advisable to reduce the proportion of urine to water in performing Clinitests if too frequent hypoglycaemia is to be avoided.

The insulin-dependent diabetic needs to be seen frequently in the combined medical/antenatal clinic to allow the dose of insulin to be adjusted. In many centres mothers are brought into hospital at about the 32nd week of pregnancy for good control to be established, but suitably motivated well-trained diabetics can sometimes manage equally well (and probably a good deal more contentedly) at home with a review every week in the combined clinic. Ketonuria in a specimen passed other than in the fasting state should, however, be a signal for immediate admission to improve diabetic control. In longstanding diabetics with vascular disease there is an increased danger of hypertension in the last trimester and of intrauterine death of the fetus. Such patients in particular need careful assessment of the best time to induce delivery (or perform caesarean section if there are independent obstetric reasons for doing this). Plasma[13] or urinary oestriol concentrations may be used

to assess fetal viability; amniocentesis to determine the lecithin-sphingomyelin ratio[14] in the amniotic fluid or the simpler and quicker shake test[14] may be used. When the results of such tests are positive the likelihood of the fetus developing hyaline membrane disease of the lung after delivery is small; this has been found to be the most common cause of death of babies born prematurely to diabetic mothers.[15]

In less complicated cases of well-controlled diabetes delivery may be delayed until 38 or 39 weeks but seldom for longer. At the time of a planned delivery the mother's diet should be replaced by a slow intravenous infusion of 10%, or sometimes 5%, dextrose. Soluble insulin should then be given eight-hourly according to the blood sugar concentrations measured either in the laboratory or in the side room by Dextrostix and a reflectance meter. Alternatively, about two units an hour of insulin may be given by slow continuous infusion, provided that the rate of glucose infusion can be varied independently of the insulin infusion.

Soon after delivery the insulin requirement falls to about the level attained before the pregnancy. Hence the standard of control should be relaxed a little when the mother takes her baby home if she is not to run the risk of hypoglycaemia. Even if the baby is of normal weight, it is best treated in a special nursery for premature infants for the first few days. There it may be observed for respiratory distress and be fed soon enough to avoid the onset of hypoglycaemia. Typically babies of diabetic mothers are heavy-for-dates, but the variation in fetal development is abnormally wide and it is the light-for-dates baby which is at greatest risk neonatally.[16] It is becoming increasingly difficult to reduce the perinatal mortality in diabetics any further, for the risk is increased even before diabetes in the mother has been diagnosed. Nevertheless, greater efforts to detect chemical diabetes in pregnant women, which is about six times as common as overt diabetes,[17] may continue to improve the prognosis.

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[3] Oakley, W G, *Postgraduate Medical Journal*, 1969, **45**, 802.
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[5] White, P, *American Journal of Medicine*, 1949, **7**, 609.
[6] White, P, *Medical Clinics of North America*, 1965, **49**, 1015.
[7] Pedersen, J, and Pedersen, L M, *Acta Endocrinologica* (*København*), 1965, **50**, 70.
[8] Pedersen, J, *The Pregnant Diabetic and her Newborn*, p 128. Copenhagen, Munksgaard, 1967.
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[10] Pedersen, L M, Tygstrup, I, and Pedersen, J, *Lancet*, 1964, **1**, 1124.
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[13] Brudenell, M, in *Carbohydrate Metabolism in Pregnancy and the Newborn*, ed H W Sutherland and J M Stowers, p 225. Edinburgh, Churchill Livingstone, 1975.
[14] Gillmer, M D G, and Beard, R W, in *Carbohydrate Metabolism in Pregnancy and the Newborn*, eds H W Sutherland and J M Stowers, p 183. Edinburgh, Churchill Livingstone, 1975.
[15] Pedersen, J, *The Pregnant Diabetic and her Newborn*, p 122. Copenhagen, Munksgaard, 1967.
[16] Farquhar, J W, *Archives of Disease in Childhood*, 1962, **37**, 321.
[17] Stowers, J M, in *Complications of Diabetics*, ed H Keen and J Jarrett, p 212. London, Edward Arnold, 1976.

Cuts and morale

The parcel of cuts in Government expenditure announced last week[1] was less harsh than might have been expected, and the NHS escaped fairly lightly. Indeed most of the proposals announced by Mr David Ennals were plans to raise more revenue rather than save money—but at least he seems aware of the near bankruptcy of many health authorities struggling to keep their current expenditure within budget.

The most depressing decision is a further reduction in capital expenditure by £20 million. Presumably the DHSS has some vision of a future oil-financed bonanza when it will be able to catch up all the ground lost by repeated postponements of building projects: meanwhile those who work in the outdated 19th century hospitals put up with the results of that decision.[2] Another saving of £20 million is expected to come from higher charges for dental and ophthalmic services. The decision to raise these charges by a large margin rather than including drug prescription charges in a smaller, overall increase is based on some bizarre logic of avoiding hardship for the chronic sick—yet the existing exemptions cover children, the retired, expectant mothers, and those with chronic disease. Equally strange is the proposal to raise £20 million next year (an estimated £40 million or so in a full year) from vehicle owners to pay for the cost of treating road accidents. As proposed, this appears to be simply a new tax on the motorist and is unlikely to lead to any cut in Government spending. In any case the scheme could take a long time to work out.

The package was completed with two less specific commitments. These were a cut in research and "other centrally financed programmes," and a reduction in the drug bill, which Mr Ennals hopes to achieve partly by forcing pharmaceutical companies to cut their promotional expenditure and partly by discussing with the medical profession ways of curbing excess prescribing. Both projects, however, would be more likely to succeed in an atmosphere of good will and co-operation, and, unpalatable though the fact may be, such co-operation is unlikely so long as the Government insists on forcing through the Health Services Bill and the Labour Party proposes nationalisation of the pharmaceutical industry.

Morale is at the heart of the matter, but we also need to examine priorities and to look at the NHS more realistically. The report on *Care of the Elderly* (see p 312) from the BMA Board of Science shows the sort of thinking required. For instance, more use needs to be made of cheaper community hospitals. Voluntary agencies could play a large part in the care of elderly and chronic sick patients, and better use could be made of existing staff and facilities. At present, however, the mood of too many NHS staff—medical, nursing, and others—is one of angry confrontation and resentment. A change in mood is needed—and the impetus must come from the top.

[1] *The Times*, 23 July, 1976.
[2] *British Medical Journal*, 1976, **1**, 787.