Wheezy bronchitis was originally used to describe preschool children who wheezed only after viral respiratory tract infections and who seemed relatively resistant to symptomatic and steroid treatment. It was thought that only a minority of these children went on to develop 'asthma'. With the increasing awareness of the underdiagnosis of asthma in children, the term was reassessed.3,6 Wheezy bronchitis was attacked as an overused euphemism for childhood wheeze. Far from avoiding the diagnosis 'asthma', implying a chronic illness, the use of 'wheezy bronchitis' led to undertreatment and inappropriate use of antibiotics in many true asthmatics. It was suggested that all who wheezed should be lumped together as asthmatic unless proved otherwise. In keeping with this concept most doctors and many parents now perceive asthma as a common condition with a wide range of severity and symptoms throughout childhood.

Last year Dr Nicola Wilson re-examined the idea of splitting up wheezy disorders in childhood.7 Like Professor Margaret Turner-Warwick, who described different patterns of airflow obstruction in adult asthmatics,8 she has argued that difficulties in treating asthmaic preschool children related to a failure in identifying clinical subgroups with different patterns of illness. Lack of a better test and the existence of a split renal group of 'wheezy bronchitics' as children who wheezed only in response to viral infections with little or no atopy compared with asthmatics of the same age. If the term is to be used at all it should refer to these kind of children. This is clearly not the group of patients studied in Finland. Forty three percent of these were highly atopic and atopic features were common elsewhere. We are given no information about a family history of atopy. Although viral infections were identified in 45% of respiratory episodes, wheezing occurred in 73% of respiratory episodes in which no virus was found. These children might have had typical asthma with wheeze secondary to precipitants unrelated to viral infections. Thirty nine percent of these children were receiving regular prophylaxis. Therefore we have prevented persistent asthma symptoms that would otherwise have been present.

If childhood asthma is to be split up carefully attention must be paid to clearly define separate subgroups. This is particularly important when considering pathogenesis and treatment of airway narrowing in our young wheezers.

Spontaneous resolution of congenital nephrotic syndrome in a neonate

SIR,—We were interested to read the article by Banton et al referring to a baby with congenital nephrotic syndrome whose condition resolved.1 We too have observed a child with congenital nephrotic syndrome who made a spontaneous recovery, and whose case raises important ethical considerations.

The mother had a raised serum α1-fetoprotein concentration on routine screening in pregnancy and anencephaly, performed at 21 weeks' gestation, shown an α1-fetoprotein concentration of 325 mg/l (normal <20) with normal cholestérine. An ultrasound scan showed no gross fetal abnormalities. A diagnosis of congenital nephrotic syndrome was made and the mother was offered termination of her pregnancy but declined on religious grounds. A boy was born at term weighing 3750 g, the placenta weighing 1000 g. He had gross proteinuria (Albustix 4+ at birth but no haematuria. He also became hypoalbuminaemic, with a serum albumin of 18 g/l at 2 weeks. He had mild pitting oedema in the first week but none subsequently and at no time did he become hypertensive. The selectivity index (IgG urine : serum as a percentage of transferrin urine : serum ratio) was initially 7% (highly selective proteinuria) but rose to 17% at 7 weeks. The proteinuria fell back to 9% at 10 weeks of age and the proteinuria had resolved by 10 months. A renal biopsy specimen at 2 months of age showed normal glomeruli by light microscopy with the exception of one obsolescent glomerulus. The tubules, interstitium, and blood vessels appeared normal. Immunofluorescent stains for complement components and immunoglobulins were all negative. These findings were compatible with 'minimal change' nephrotic syndrome. He was treated with a high protein diet (6 g/kg/day), replacement immunoglobulin, and penicillin prophylaxis up to 5 months of age; neither steroids nor diuretics were used. The child is now 6 years old, has not relapsed, and is entirely well.

The congenital nephrotic syndrome is heterogeneous and various types can be recognised histologically. The Finnish type, and the forms exhibiting diffuse mesangial sclerosis, or focal segmental glomerulosclerosis all have a poor prognosis. A policy of prenatal diagnosis is therefore justified in families with a previously affected child whose clinical course or histological type is known to be adverse. However from time to time screening for neural tube defects by maternal α-fetoprotein in serum and amniotic fluid will lead unexpectedly to the diagnosis of congenital nephrotic syndrome. Whether the diagnosis of congenital nephrotic syndrome and the actual prognosis will not be known. In view of the benign course of our patient, and the one described by Banton et al, the counselling of such families can be extremely difficult.

A review of patients with congenital nephrotic syndrome at this hospital over a period of 24 years (5 Yoshida, RHR White, F Raafat, unpublished data) showed that, of 26 cases proved at biopsy, eight were of the Finnish type, three had the form exhibiting diffuse mesangial sclerosis, 12 had focal segmental glomerulosclerosis, and the histological appearances compatible with minimal change nephrotic syndrome. Of the latter, two siblings were steroid resistant and continue to have mild proteinuria at 19 years of age, with normal renal function, although one is hypertensive. The other child responded to prednisolone 60 mg/m2/day and remained in complete remission up to 6 years of age, when proteinuria recurred; a further biopsy specimen taken at this time showed type 1 mesangiocapillary glomerulonephritis. One of the 12 infants with focal segmental glomerulosclerosis was partially steroid resistant and is currently on treatment after treatment with cyclophosphamide.

The distinction between the histological subtypes can sometimes present difficulties. Although the glomeruli in Finnish nephrotic syndrome show mild mesangial hypercellularity, this can also be a feature of minimal change nephrotic syndrome. Moreover the cystic tubular dilatation which characterises Finnish nephrotic syndrome is also seen in severe and also occurs in focal segmental glomerulosclerosis, while segmental sclerosis in the latter condition may initially be so minimal that it is not apparent in the biopsy specimen obtained during the first three months of life. Experience in different diagnostic can only be gained if patients are referred to regional paediatric nephrology centres for evaluation.

If the differential diagnosis remains in doubt, it is reasonable to offer a trial of corticosteroid treatment, with antibiotic prophylaxis because of the increased risk of infection, rather than to dismiss the condition as unresponsive. The counselling of a woman who had not previously had a child with congenital nephrotic syndrome requires an awareness that there is, perhaps, as much as a 10% prospect of steroid responsiveness if she produces an affected, liveborn infant.

The authors are grateful to Dr Partridge for permission to report this patient.