mg/kg) of oral trimeprazine tartrate was given 5 days postoperatively before the sutures were removed. Four hours later she became drowsy, examination showing her to be deeply unconscious with no response to painful stimuli. Her respiratory rate was slow at 10/minute with very shallow respiration. Air entry was poor to both sides of the chest, but there was no cyanosis. Neurological examination showed dilated pupils which were equal in size, as well as a divergent squint. Her gag reflex was intact and no other focal signs or manifestations of raised intracranial pressure were evident; blood pressure was 120/70 mmHg, and Dextrostix recordings were normal. Her condition slowly improved during the next 6 hours and she made a full recovery.

Discussion

Trimeprazine has pharmacological actions intermediate to promethazine and chlorpromazine, thus having marked antihistamine effects as well as central nervous system actions similar to chlorpromazine. Antihistamine drugs in toxic doses produce complex central nervous system effects,[3] which can lead to respiratory depression. Respiratory depression has not been reported after recommended doses of trimeprazine tartrate, and it is of interest that both siblings responded in a similar manner.

Kahn and Blum.[4] suggested that trimeprazine tartrate in normal dosage (1 mg/kg per day) was the possible cause of death in 7 infants with sudden infant death syndrome. All infants tended to be sleepy and they suggested that the central nervous system action caused prolonged sleep apnoea leading to death.

In both these children there was no antecedent history of respiratory illness or drug sensitivity, and no other drugs were administered concurrently. The maximum recommended dose of trimeprazine is up to 5 mg/kg orally,[2] and each patient was given a dose considerably smaller than this. An idiosyncratic response to trimeprazine must be suggested in these 2 children leading to respiratory and central nervous system depression.

I thank the Drug Information Service at Nottingham City Hospital, and Mr Malcolm Deane, Consultant Plastic Surgeon, for permission to publish this report, and Dr A D Milner for advice.

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Correspondence to Dr N P Mann, Children's Department, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH.

Received 2 July 1980

Henoch-Schönlein nephritis: long-term prognosis of unselected patients

O KOSKIMIES, SEVGI MIR, J RAPOLA, AND J VILSKA

Children's Hospital, University of Helsinki

SUMMARY Progressive glomerulonephritis is the most serious feature of Henoch-Schönlein syndrome. In a series of 141 children with Henoch-Schönlein purpura 39 (28%) had abnormal urinary sediment for a duration of more than one month. This subgroup was followed up for 3·0 to 13·8 (mean 7·2) years. One child progressed to renal failure and 2 developed chronic glomerular disease. In this series most of the patients with Henoch-Schönlein syndrome and nephritis had a good prognosis.

The long-term prognosis of children with Henoch-Schönlein (HS) syndrome and nephritis is not yet clear.[1] One child in 4 with HS nephritis being treated in a renal unit in the UK was found to have chronic renal disease, or to be in renal failure after a follow-up of a mean of 10 years.[2] In our experience the long-term (mean 7·2 years) prognosis of a less selected patient population with HS nephritis is more favourable.

Patients, methods, and results

In 1964–76 a total of 141 patients with HS syndrome

Table *Long-term prognosis of 29 children with Henoch-Schönlein nephritis*

At the onset of the disease								*State at follow-up (3 to 13·8 years later)*			
*Renal presentation**	*No of patients*	*Histology grade*						*Normal*	*Minor urinary abnormality†*	*Active renal disease‡*	*Renal insufficiency*
		I	*II*	*III*	*IV*	*V*	*VI*				
Microscopic haematuria	1		1					1			
Proteinuria and haematuria	20	4	9	6	1			11	7	2	
Nephritic syndrome and proteinuria	1		1						1		
Nephrotic syndrome and haematuria	6			5	1			5	1		
Nephritic-nephrotic syndrome	1					1					1

*Only children who had abnormal urinary sediment present for more than 1 month are included.

†Microscopic haematuria or proteinuria <1 g/24h, or both.

‡Proteinuria >1 g/24h or hypertension, and glomerular filtration rate >60 ml/min per 1·73 m².

was seen at the Children's Hospital, University of Helsinki. Nine of them had been specially referred from other hospitals in Finland but the remainder could be considered to represent an unselected series of patients of a large paediatric hospital. Of these 141 children 102 had no constant clinical evidence of renal disease during about 6 months' outpatient follow-up apart from transient microscopic haematuria of less than 4 weeks' duration in some, nor did any child have to be referred later because of renal problems or hypertension.

Abnormal urinary sediment for a duration of more than one month was recorded in 39 patients. Eight children made a good recovery within 3 months. A renal biopsy was performed on 31 children, 14 girls and 17 boys aged between 2·5 and 14·1 (mean 8·4) years. The biopsied specimens were re-evaluated and grouped according to the classification of the International Study of Kidney Disease in Children (ISKDC)*, without knowing the clinical state of each patient.

Altogether 29 patients were followed up for 3·0 to 13·8 (mean 7·2) years. Two children were lost to follow-up; each had had normal renal histology and a normal urine specimen at the last examination. Most of these 29 children have been followed by one of us; a few have been examined by local general practitioners and the data made available to us.

The symptoms and signs of renal involvement and the renal biopsy histology at the onset of the disease were compared with the clinical state of the patients 3 to 13·8 (mean 7·2) years later (Table). Of a total of 21 patients whose renal presentation was proteinuria and haematuria or nephritic syndrome (haematuria with increased blood urea nitrogen and oliguria) 2 had signs of active renal disease at follow-up (Table). None of 6 children with nephrotic syndrome and haematuria at presentation had developed chronic renal disease. Only one patient had nephritic and later nephrotic syndrome; she rapidly developed progressive renal insufficiency and died 10 months after the onset.

If the renal histology at the onset of the disease is compared with the clinical state after 7·2 years of follow-up all patients whose renal biopsy specimens were of histological grade I, II, or IV had normal urine or minimal urinary abnormalities. Children with renal histological grade III at presentation had normal urine or minimal abnormalities at follow-up except for 2 of the patients who presented with haematuria and proteinuria. One of these had hypertension and the other haematuria and proteinuria of >1 g/24h, but her glomerular filtration rate was 95 ml/min per 1·73 m²; these findings were made after 8·9 and 6·0 years. The latter patient was the only one of the 9 children referred to us from another hospital who had long-term renal problems.

Nineteen repeat biopsies were performed on 12 patients; in one of these patients the histological findings had progressed from grade II to III, but 4·6 years later his clinical state was normal. In the rest of the renal biopsy specimens the changes were either of the same histological grade or were less pronounced.

Between 1964 and 1976 no uniform therapeutic regimen has been applied, and all the 13 children given symptomatic treatment only have fared well. Eight of the remaining patients have received corticosteroids only; two of them have signs of active renal disease. The others have received corticosteroids and immunosuppressants.

Comment

Of 141 children with HS purpura only 39 (28%) had clear renal disease and of these only 3 had serious renal involvement at follow-up. Moreover, if the 9 children referred from regional hospitals are

*Details available from the offices of the ISKDC, Department of Pediatrics, Albert Einstein College of Medicine, Bronx, New York, NY, USA. See also reference 2.

excluded, only 2 of the remaining 132 unselected children had serious renal disease at long-term follow-up (for one of whom the disease was fatal). Thus the prognosis of an unselected series of children with HS syndrome and nephritis seems to be better than more selective series have reported.[2]

We thank the Sigrid Jusélius Foundation for generous support.

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Correspondence to Dr O Koskimies, Children's Hospital, University of Helsinki, Stenbäckinkatu 11, SF-00290 Helsinki 29, Finland.

Received 1 July 1980

Correspondence

Perinatal mortality

Sir,

Pamela Davies in Current topics[1] reviewed the recommendations of the Short report[2] and commented unfavourably on them, if I understood her correctly, for three reasons.

Firstly, she said that money would be better spent on primary care and prevention of prematurity. In theory there is some truth in this view, but what evidence does she have that such expenditure would affect either the prematurity or the perinatal mortality rate. Can she quote any controlled studies to prove it? After all, as Dr Davies stated, the great improvement in the standard of living in the UK since the war has not been associated with any change in the prematurity rate. Before we spend more money indiscriminately on primary care in the fond hope that there will be some 'spin-off', let us have some clear evidence that money so spent is likely to have the desired effect.

Secondly, she implied that as money is in short supply because of the policies of the present government, we should not complain bitterly about lack of perinatal provision. What an amazing counsel of inertia from a senior paediatrician, and what an abrogation of professional responsibility. Surely one should ensure that hospital facilities for seriously ill neonates are constantly upgraded and improved.

Thirdly, she questioned whether expenditure on neonatal intensive care would have any major effect in preventing handicap. In many of these arguments I must agree with her, since I believe that the proportion of the total handicap generated in low birthweight infants is very small. However, the data showing there has been no major change in the numbers of handicapped infants arising from neonatal intensive care units, despite modern intensive care, can be interpreted in two ways. Dr Davies seemed to imply that since more intensive care has not reduced the absolute amount of handicap, it was not worth spending money on neonatal intensive care. This really is a fatuous argument. I would subscribe to the view that the fact that modern neonatal intensive care saves the lives of increasingly large numbers of babies *without* increasing the absolute numbers of handicap is a major vindication of the techniques of neonatal intensive care.

Let there be no doubt that neonatal intensive care does save lives. Comparison of the national figure for mortality in infants of 1·0–2·0 kg birthweight with that reported from committed neonatal intensive care units leads very quickly to the conclusion that about 2000 low birthweight infants die of neglect in premature baby units each year in England and Wales.

What was particularly staggering about Dr Davies's comments that we should not be getting more for neonatal intensive care was that she seemed to be unaware of what was happening on her own doorstep. Does she not know that paediatricians in the North West Thames Area are unable to find places for critically ill infants of low birthweights in the regional neonatal intensive care units in London, including her own at Hammersmith, because they are bursting at the seams? I certainly know it because often I have to take their babies into our own under-funded and under-equipped East Anglian intensive care unit at Cambridge; therefore I find it particularly galling to have my attempts to raise funds and improve facilities for the Cambridge unit described as 'money grabbing' and 'empire building'.

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N R C Roberton
Department of Paediatrics, Addenbrooke's Hospital, Hills Road, Cambridge CB2 2QQ

Dr Davies comments:

Dr Roberton asks if I can quote any controlled study to prove that expenditure on primary care would (favourably) affect the prematurity (low birthweight) or perinatal