Table *Mean (SEM) platelet phenolsulphotransferase P and M activities in children with abdominal migraine and in controls*

	Specific activity (nmol/mg protein/10 min)	
	Phenolsulpho-transferase P	*Phenolsulpho-transferase M*
Patients (n=21)	0·037 (0·006)	0·382 (0·025)
Controls (n=13)	0·031 (0·007)	0·298 (0·043)

phenolsulphotransferase P or phenolsulphotransferase M activity between patients and controls (table). No significant correlation was found between platelet phenolsulphotransferase activity and age (r=0·09).

Discussion

The condition to which the clinical label of 'abdominal migraine' has been applied does have certain characteristic features in common with 'cranial' migraine in terms of family history, symptomatology, and triggering factors. The fundamental difference between the two conditions is in the location of the perceived pain and, as discussed earlier, this fact renders the label etymologically unsound. The justification for its continued use would be some firm physicochemical confirmation that a relationship does exist between 'abdominal migraine' in childhood and common or classical migraine in later life. The lack, to date, however, of any diagnostic laboratory test for migraine makes this confirmation difficult.

The finding that adult patients with migraine caused by diet have lower mean platelet phenolsulphotransferase activity than those with migraine not caused by diet or controls suggested the possibility that a similar biochemical deficiency might exist in children suffering from 'abdominal migraine' with probably dietary trigger. The results of this study, however, fail to show any such common biochemical deficiency. Even so, interpretation of these negative results must be made with caution because of the relatively small numbers of patients investigated. With this reservation, we would state that, at least, no further evidence has been educed pointing to a common aetiology of so called abdominal migraine and the more firmly established 'cranial' variety.

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Epidemiology of rheumatic heart disease

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SUMMARY We compared the incidence of rheumatic heart disease in elementary schoolchildren from low and high socioeconomic groups; children from one of the schools were rescreened 10 years later. The results showed that the incidence of rheumatic heart disease was significantly higher in low socioeconomic group but it is gradually declining.

Rheumatic carditis is one of the most important preventable heart diseases in children. Although in developed countries there is a dramatic decline in the incidence of rheumatic fever and rheumatic heart disease, they still remain a leading cause of heart disease in developing countries such as Turkey.[1 2] This difference is reported to be related to factors such as crowding, nutrition, hygiene, urban-rural residence, and access to medical care.[2–4]

This study was undertaken to assess the effect of socioeconomic state on the prevalence of rheumatic heart disease and whether there was any decline in the morbidity rate over the last decade with the improvement of medical facilities.

Method

The study consisted of two surveys. In the first part, in 1976, a total of 3039 children (1421 girls and 1618 boys) were screened. One group consisted of 1590 children from a shanty town who attended three primary schools; the other group consisted of 1449 children of a high socioeconomic group from a primary school in Ankara, Turkey. All the children were white.

Cardiac examination of each student was carried out by a paediatric cardiologist. For a diagnosis of rheumatic heart disease to be established a murmur was required to be of at least grade II/IV loudness, to have typical characteristics, and radiation. Children judged to have possible heart disease were further evaluated with electrocardiography, and chest radiography and echocardiography in some. Congenital cardiac diseases and innocent murmurs were excluded on these grounds.

The parents of the children filled in a questionnaire inquiring into the annual income, how many rooms their housing had, how many members their family consisted of, and whether or not the parents or a member of their families had had rheumatic fever or rheumatic heart disease.

In the second part of the study, in 1986, 538 children of the low income group from one of the primary schools previously studied were evaluated. The same procedures were undertaken.

The significance of the difference between parameters in different groups was analysed by Student's t test; $p<0.01$ was considered significant.

Results

In the first part of the study, in 1976, a total of 20 cases (nine girls, 11 boys) of rheumatic heart disease were identified, so that the total prevalence rate was 0·658%. Table 1 shows that the rate differed significantly between the groups. All of the children with rheumatic heart disease had mitral insufficiency, one with accompanying aortic valve insufficiency. Only three of them had mild exercise intolerance, one with dyspnea on effort. Four of these 20 children had a definite history of rheumatic fever.

On examining the questionnaires returned it was found that 75 out of 1317 (5·7%) of the children in the low socioeconomic group had a history of rheumatic fever; in the high socioeconomic group this was true in only 18 out of 815 (2·2%) children. Unfortunately some of these findings were not confirmed by medical records. Thus they may not be totally reliable.

In the low socioeconomic group, where the income levels were significantly lower than in the other group, the children were living in houses with about four people/room. In the high socioeconomic group all the children lived in houses with about one person for the comparable area.

Ten years later, when the pupils of one of the primary schools in the low socioeconomic group were screened, three (two girls and one boy) of the 538 children were diagnosed as having rheumatic heart disease; this gives a rate of 0·56%. One of the children also had rheumatic activation according to the Jones criteria. The previous rate in the same school was 0·94%. The difference between the two periods was significant (table 2).

Table 2 *The prevalences of rheumatic heart disease in a school in the low socioeconomic group 10 years apart*

	No of children	*No with rheumatic heart disease*	*Rate (%)*
1975	637	6	0·94
1985	538	3	0·56

The difference between the two groups was found to be significant ($p<0.01$).

Table 1 *Prevalence of rheumatic heart disease in the two groups*

	No of children	*No with rheumatic heart disease*			*Prevalence (%)*
		Girls	*Boys*	*Total*	
Low socioeconomic group	1590	7	10	17	1
High socioeconomic group	1449	2	1	3	0·2
Total	3039	9	11	20	0·6

The difference between the two groups was found to be significant ($p<0.01$).

Discussion

The prevalence of rheumatic heart disease among schoolchildren has previously been assessed in various countries.[3 5] In 1970 Morton *et al* found a relatively high prevalence of rheumatic heart disease among the schoolchildren in the San Luis Valley, which is a region of low socioeconomic level.[5]

Our higher rate for rheumatic heart disease in children from a low socioeconomic group is consistent with these reports.[4 5] The overall incidence, which was 0·6% in our series, is also considerably higher than in developed countries.

The second part of our study shows a decline in the morbidity rate of rheumatic heart disease in the last decade. As the children from only one school from the low socioeconomic group were screened we compared the result with the rate found in this school alone, in 1976. Although the number of children may seem inadequate a parallel decline noted in the number of patients with rheumatic heart disease seen in our cardiology clinics seems to support our thesis. The reasons for the decreasing incidence in rheumatic fever and rheumatic heart disease has not been completely understood. Markowitz agrees that widely disseminated programmes for the use of penicillin play an important part but insists that other unknown factors could be important.[3] Gordis has concluded that changes in the occurrence of rheumatic fever cannot be fully accounted for just by medical care and antibiotic use, but that we lack adequate information regarding other possible cofactors and that although group A streptococcus is the critical aetiologic agent of rheumatic fever, the disease is probably multifactorial in origin.[6] In Turkey we believe that improving medical care and greater access to health care have also appreciably influenced the documented decline. Further studies from developing countries might serve to enlighten the subject.

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Endemic bladder stones in Nepal

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SUMMARY Bladder stones account for a large proportion of surgical admissions in many developing countries. We report in detail the clinical features and risk factors of one such case, a 5 year old Nepali boy, and propose the theory that a low calcium intake, by causing hypocalciuria, predisposes to bladder stone formation.

Bladder stones are endemic in an area stretching as a belt from Turkey and Iran across India to Thailand and beyond to Indonesia and Papua New Guinea. However, they are rare in Africa.[1] They are also rare in the developed world, although this has not always been the case: they were common in England before the turn of the century when itinerant lithotomists flourished especially in 'stone districts', where the prevalence was unusually high, such as Norfolk.

In these areas of endemic bladder stones, boys are affected more than girls with a peak incidence at 5 years of age and the stones, which are rarely present in the kidneys, usually consist of uric acid, ammonium acid urate, or oxalate.[1] This is in contrast with the stones found in people from developed countries which are rare, occur in adults rather than children with a roughly equal sex distribution, are more common in the upper urinary tract, and usually consist of phosphates.

The following is, as far as we are aware, the first detailed case report and analysis of a bladder stone from Nepal. In addition to the commonly held dietary cause, we suggest that a low calcium intake may contribute to stone formation.