mographic differences [3]. We evaluated the prevalence of *H. pylori* infection in children belonging to different ethnic populations living in the same area. We used a last generation serological ELISA test (MALAKIT, Biolab, Belgium) to compare the frequency of *H. pylori* infection in asymptomatic Belgian and immigrants' children. We studied 512 children, aged 1 month – 17 years, who were admitted for elective minor surgery. All children were born in Belgium: 472 originated from Belgium, 29 were children of parents who originated from Mediterranean countries (Tunesia and Marocco), and 11 of parents from central Africa. The frequency of *H. pylori* seropositivity was 4.0%, 17.2%, and 18.2%, for those from Belgium, the Mediterranean countries and central Africa, respectively. All individuals studied were of middle to upper class socioeconomic status. The difference in *H. pylori* infection between Belgian and immigrant children is statistically significant ($P < 0.01$; Student's *t*-test), whereas no statistically significant difference was found between the immigrant populations.

This study demonstrates that there is a significant difference in *H. pylori* seropositivity between children of different ethnic origins, despite the fact that all investigated children were living in Belgium since birth and that they were all of comparable socioeconomic conditions.

Consequently the question arises whether the statistically significant difference in seropositivity between ethnic Belgian children and Mediterranean or central African children is due to an still unknown (genetic) predisposition. Subsequent studies will have to be performed in order to elucidate this question.

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3-Methylglutaconic aciduria in a patient with Pearson syndrome

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Sir: 3-Methylglutaconic (3-MGC) aciduria (McKusick 250950) has been classified into two subtypes and a group of "unspecified" patients [3], or four subtypes and a group of "unspecified" patients [2] including 3-methylglutaconyl-coenzyme A hydratase deficiency, patients with X-linked cardiomyopathy, neutropenia and normal or abnormal mitochondria, Iraqi Jews with optic atrophy and spastic paraplegia, and patients with dysmorphic signs, congenital malformations, severe retardation and cerebellar dysgenesis [2, 3]. The group of "unspecified" patients consists of a variety of phenotypes exhibiting lactic acidaemia, severe psychomotor retardation, neurological involvement, and focal hypodensity in different areas of the brain. In this group 3-MGC aciduria may only be a marker for another metabolic disease [1, 2, 3]. 3-MGC aciduria has been detected in 3-hydroxy-3-methylglutaryl coenzyme A lyase deficiency, carbamoyl-phosphate synthetase deficiency [3], a mitochondrial respiratory chain defect [1], and glycogenosis Ib [Trefz unpublished results]. We now report a patient with 3-MGC aciduria and Pearson syndrome. The girl was the first child of healthy, unrelated parents. In the first weeks of life she developed severe aregenerative anaemia. Subsequently she exhibited exocrine pancreatic insufficiency and neutropenia accompanied by recurrent infections. At 7 months, she began to refuse food intake and vomited recurrently. Intestinal biopsy showed partial villous atrophy. Ultrasound investigation revealed enlarged kidneys with increased echogenicity. In her 2nd year, the patient was of short stature and exhibited wavy erythematous lesions on her arms suggestive of photosensitization. Recurrent electrolyte derangements appeared. At 3 years of age she was admitted to the hospital somnolent and hypoglycaemic and became comatous. Based upon antibody titre and CT scan results, herpes encephalitis was diagnosed. In the course of this crisis, lactic acidaemia was first noticed. Six months later she was seen at our hospital. At that time she was on a low-protein diet because of chronic renal insufficiency, exhibited hypotonia, and ptosis. Carnitine deficiency was diagnosed. Under carnitine supplementation the hypotonia improved. One month later she died during an acute episode of electrolyte imbalance and acidosis leading to cardiac failure. Laboratory findings during protein intake of 0.9 g/kg per day included: elevation of plasma lactate (3.9 mmol/l; normal < 1.8) pyruvate (0.22 mmol/l; normal < 0.15), 3-hydroxybutyrate (0.22 mmol/l; normal < 0.07), and acetoacetate (012 mmol/l; normal < 0.036); thrombocytopenia (45000/mm^3) and Fanconi syndrome. Levels of urinary organic acids (mmol/mol creatinine) detected were: lactate (9830; normal < 25), pyruvate (894; normal < 12), 3-hydroxybutyrate (19.2; normal < 3), acetoacetate (30.4; normal < 2), 3-MGC acid (86; normal < 9), and 3-methylglutaric acid (23; normal < 7) [normal values from 5].

The clinical symptoms described were indicative of a mitochondrial cytopathy and corresponded exactly to those described in Pearson syndrome [McKusick 26056; 4]. Analysis of the patient's leucocyte DNA demonstrated two populations of mitochondrial DNA, one normal and one carrying the 4977 base pair deletion described in Pearson syndrome [4]. We conclude that patients with 3-MGC aciduria and symptoms suggesting a mitochondrial cytopathy as described above may harbour a mutation in a gene important for mitochondrial metabolism, which may be encoded in the nuclear or mitochondrial genome.

Abbreviation: 3-MGC = 3-methylglutaconic

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Acute sensorineural deafness following herpes simplex infection

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Sir: We describe a child with acute sensorineural hearing loss in association with herpes simplex infection. A 3-year-old male born of a full-term normal delivery with normal mental and motor milestones developed a mild febrile illness with no other associated symptoms that lasted 3 days. A week later it was noticed that he had become unresponsive to sounds like the ringing of the telephone or when talked to by the parents. There were no symptoms of vertigo, tinnitus or vomiting. He had not been given any ototoxic drugs and had not had any other relevant past medical illnesses. There was no family history of deafness. Examination showed a pleasant child of normal stature and weight. He was unresponsive to verbal commands and loud noise. Auropalpebral reflex in response to loud noises was absent. He had no nystagmus, ataxia, nor any other neurological deficits. Apart from hearing loss, the ear, nose and throat were normal as were the other organ systems.

Investigations revealed normal haemogram and blood biochemistry. Serological tests for *Toxoplasma,* rubella, cytomegalovirus and syphilis were negative. However, IgG and IgM to herpes simplex (HS) were positive. Two months later the IgM was negative, but IgG was positive. Brain stem auditory evoked response at 105dB showed (Fig. 1) no response. X-ray films of the skull were normal. He was not co-operative for audiometry.

He was treated initially with prednisolone 2 mg/kg (40 mg/day) without response; by the 5th day, after obtaining the positive serological test for HS, he was started on oral acyclovir 200 mg five times a day. A week later he started responding to calls and started answering questions. Oral acyclovir was continued for 10 days. Repeat brain stem auditory evoked responses were done at monthly intervals and showed progressive improvement though the response was still not fully normal and had low amplitudes of the wave forms (Fig. 2). Follow up 6 months later showed the patient to be hearing and responding to normal conversation though not as well as before the illness. He was doing well at school and earning good grades.

Acute sensorineural hearing loss can occur as a complication of viral illnesses like mumps and herpes zoster [3–5].

Abbreviation: HS = herpes simplex

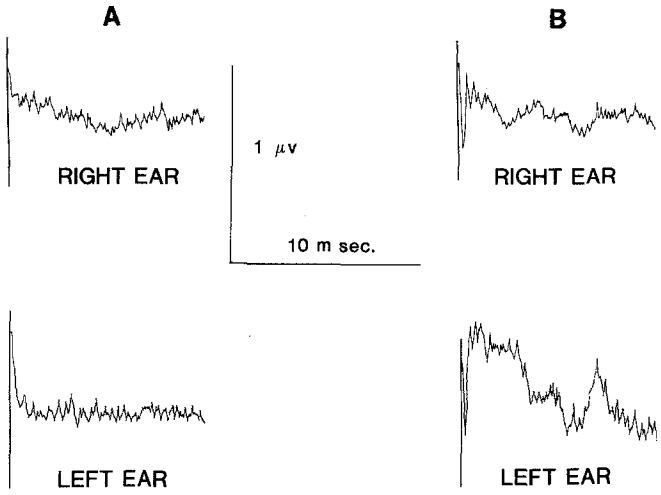

Fig. 1. A Shows bilateral absent response in the brain stem auditory evoked responses at 105 dB at initial presentation. **B** Brain stem auditory evoked responses at 105 dB, 3 months later shows partial return of the response albeit of low amplitude

Deafness in the neonate may occur as a consequence of intrauterine infection from maternal HS or neonatal encephalitis [1, 2, 6]. The present case is interesting in that deafness occurred after a recent HS infection (as judged by the positive IgM and IgG serological tests) and responded to acyclovir therapy. We would like to highlight that this treatable condition should be searched for in patients with acute hearing loss.

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Pseudohyperkalaemia in Kawasaki disease

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Sir: We read with interest the report by Shimizu et al. on pseudohyperkalaemia in children with Kawasaki disease [2]. These authors suggested that this relationship was previously unreported. We studied the relationship of serum potassium to platelet count in children and determined that for every eleva-