Enuresis in children

Sir,

I was interested to read your correspondent’s personal view of her enuresis and her observations on its management.1 One can’t but help sympathise with her. In my experience most parents of enuretic children are bothered not so much by the nuisance that it causes them but by whether it could be a symptom or sign of disease in soma or psyche; if they are reassured on that score they can usually cope until the child demands to be ‘cured’. As we all know, wetting can be a manifestation of many diseases: of the urinary tract, of the upper respiratory tract (obstruction at night), of the endocrine system (diabetes mellitus, diabetes insipidus, and Addison’s Disease), of the gut (coeliac disease with intestinal water retention), of the cardiovascular system (improving renal perfusion at rest), of the hypothalamus (failure of development of diurnal rhythms), of the central nervous system (nocturnal or early morning fits), or of the psyche; these should be carefully excluded before the symptom is ‘treated’ on its own merits. For this reason, and because it represents a stigma, attendance at so called enuretic clinics may be harmful, as is the provision of a star chart—implying that the wetting is under voluntary control—or the imposition of an alarm (although this can be very helpful when managed by the child himself). Drugs like imipramine do seem to help but they bring ‘magic’ into the management, are dangerous in overdose, and do not have a convincing rationale in relation to bladder innervation, if indeed they do act by increasing capacity. In this context it is interesting that nearly all nocturnal enuretics are wet before their parents go to bed.

Perhaps as members of a profession whose practice is supposed to be science based we should confine ourselves to sorting out and acting on what we do know of the pathogenesis and leave the rest to growing up, practical sympathy, education for all concerned—particularly teachers—and common sense measures to mitigate nuisance like the provision of a washing machine, using a covered mattress and easy to wash sheets (supplemented by newspapers used as blotting paper), and a potty under the bed.

Reference


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Hearing loss due to mumps

Sir,

We share the opinion of Hall and Richards that mumps is a major cause of severe sensorineural hearing loss.1 During an epidemic of mumps in Israel in 1984, 85 children with mumps were admitted to the paediatric department of the Beilinson Medical Center, which serves an area of roughly 68 000 children (age 0–14 years). Seventy nine of the patients had symptoms or signs of meningencephalitis. Three children (3.5%) developed unilateral profound sensorineural deafness in association with mumps.

The first case was a 3 year old boy who had bilateral parotitis and meningal irritation. Cerebrospinal fluid examination showed 1020 cells/mm³ (98% mononuclear cells), and mumps virus was subsequently isolated from this fluid. During his stay in hospital his parents noticed that he did not respond to calls, and audiology showed right severe sensorineural hearing loss.

The second patient was a 10 year old girl with bilateral parotitis, severe headache, and vomiting. On the sixth day in hospital she complained of inability to hear a telephone conversation through her right ear, and severe sensorineural hearing loss was documented by audiometry. The third patient was a 8 year old girl who was admitted for observation with fever of 39°C. In the ward she complained of inability to hear with the right ear, and this was confirmed by audiometry. As we were aware of the association of hearing loss and mumps this possibility was tested, and her complement fixation antibody for mumps rose from 1/20 to 1/240 within three weeks, which confirmed recent infection. Routine hearing screening tests done on these three patients before the disease gave normal results. All the other children with mumps who were in hospital were tested and no significant hearing abnormalities were found.

A postal inquiry, albeit with an incomplete response, showed three additional cases of deafness after mumps in 1984, one of them with severe bilateral hearing loss.

References


Correspondence

During the same year, 6584 cases of mumps were reported to the health authorities. Although one considers many unreported and subclinical infections, the occurrence of six cases of deafness due to mumps seems significantly higher than the assumed rate of 1/20,000 infections.1 According to Sullivan et al.,2 about 2.5% of patients with mumps may require treatment in hospital. Considering this possible rate of admission the incidence of severe hearing loss associated with mumps may have been as high as 1/3400 cases of clinical mumps.

Two groups of patients with deafness related to infection with mumps may have been missed by the study of Hall and Richards: firstly, cases of mumps that were not apparent, which are considered to be in the range of 30–40% of all infections with mumps.3 In these cases, it is impossible to obtain a history of parotitis, but, as shown by our third patient, severe hearing loss may occur. Secondly, there are rare cases of bilateral hearing loss caused by infection with mumps.4 Therefore, the importance of mumps as a cause of acquired deafness may be greater than usually estimated.

References


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Non-convulsive status epilepticus

Sir,

We have read Manning and Rosenbloom’s interesting paper concerning non-convulsive status epilepticus.1 They report 13 patients, five of whom presented with fluctuating neurological symptoms (ataxia, dysphasia, and unresponsiveness) and developmental deterioration coinciding with continuous paroxysmal activity on the electroencephalogram. The authors stress the ‘epileptic’ nature of such symptoms. They have not, however, reported whether the evidence of status epilepticus was also detected on an electroencephalogram taken during sleep.

We have observed five boys and five girls with a mean age of 6 years who have long standing ‘electrical status epilepticus during slow sleep’. In six cases focal or unilateral epileptiform activity occurred during more than 85% of non-rapid eye movement sleep, but electroencephalograms taken while they were awake disclosed only a frontal or frontal/central focus. The remaining patients presented with typical electrical status epilepticus during slow sleep characterised by a generalised epileptic pattern on electroencephalography during non-rapid eye movement sleep.2 All patients had fluctuating neurological symptoms as well as disturbance of gait and motor coordination, speech impairment, and developmental deterioration. All these children were suffering from epileptic seizures and were mentally retarded (six presented with congenital cerebral palsy). Remission of electrical status epilepticus during slow sleep was observed in two patients who fully recovered.

We believe that such fluctuating symptoms are not directly related to paroxysmal activity seen on electroencephalograms, but probably reflect a more complex brain disorder. In our cases a continuous epileptic activity was recorded on electroencephalograms only during sleep, while neurological symptoms were present when the children were awake. Moreover, our observations suggest that electroencephalography should be performed during spontaneous sleep in children with fluctuating neurological symptoms and mental deterioration, even if the recording made when awake did not show non-convulsive status epilepticus.

References


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Topical iodine, breastfeeding, and neonatal hypothyroidism

Sir,

The case of transient congenital hypothyroidism after topical iodine in pregnancy reported by Danziger et al1 confirms and extends our previous observation2 that the cutaneous application of iodine-iodine (PVP-I) in mothers at the time of delivery results in iodine overload and in slight impairment of the thyroid function of their breastfed infants due to a Wolff-Chaikoff effect.3 We recently observed severe transient congenital hypothyroidism in a breastfed infant born to a mother who had performed vaginal douching with PVP-I for gynaecological reasons twice a day since delivery. As shown in the figure the concentration of thyroid stimulating hormone (TSH) in serum was moderately raised at the time of systematic screening for congenital hypothyroidism, but thyroxine (T4) was normal. Control examinations performed on days 14 and 22 showed an appreciable increase in serum TSH.
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