ADENO - TONSILLAR HYPERTROPHY AS A CAUSE OF SLEEP APNOEA SYNDROME

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SUMMARY

An interesting case is described here in which partial airway obstruction resulted in sleep apnoea and which was relieved by adeno-tonsillectomy. Enlarged tonsils and adenoids causing upper airway obstruction represent one facet of a continuum of hypoventilation - sleep disorders and clinical examination with x-rays will help in determining patients at risk of developing these syndromes.

INTRODUCTION

Pulmonary hypertension with severe congestive cardiac failure in children secondary to adeno-tonsillar enlargement was first reported in the Journal of Paediatrics in 1968. Seven years later Simmons [1] reported the finding of chronic hypersomnolence and periodic sleep apnoea in patients with upper airway obstruction. Subsequently through the work of Guilleminault [2] and others the obstructive sleep apnoea syndrome in children was identified. Rowe, [3] reported nine patients with a sleep apnoea syndrome of whom three had adreno-tonsillar hypertrophy.

The following unusual case illustrates the varied problems which may arise from partial upper airway obstruction. Patients with this condition can present as a diagnostic enigma to paediatricians and otorhinolaryngists alike until the airway problem is recognised as a primary factor responsible. The correction of this partial airway obstruction relieves the patient of the multifarious symptoms.

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Case Report

The patient, a four year old Malay boy from a well-to-do family, was admitted to the Paediatric Ward of the General Hospital, Johor Bahru, in early 1980 for complaints of cyanotic spells during sleep and loud snoring. The mother reported that she had noticed that her son had difficulty in breathing during the nights for over six months. His sleep was disturbed and punctuated with spells of cyanosis and apnoea which the mother noted and by awakening the child the cyanosis improved. Of late the frequency of these apnoeic episodes had increased. This sequence of events had increased the irritability of the child and had affected his feeding habits. He had been investigated for his cardiac problem by the paediatrician when he was detected to have pulmonary hypertension.

On admission to hospital he was found to be irritable, continuously crying, agitated and had an irritant cough. He was transferred to the I.C.U. where he was investigated for peripheral cyanosis and episodes of progressively laboured breathing accompanied by mild stridor. These were followed by a period of apnoea terminating with arousal and accompanied by agitation and crying. With oxygen and parenteral antibiotics for the chest infection his condition improved slightly but he still continued to have sleep apnoea and snoring. E.N.T. consultation was sought to identify the cause of his mild stridor. Clinically the patient had the typical adenoid facies with a small nose and thick lips. He had grossly enlarged tonsils which

almost kissed each other when he gagged. Radiologically an enlarged adenoid was also noted which probably contributed to his airway problem and sleep apnoea.

After some persuasion the mother consented for an adeno-tonsillectomy which was carried out. The child had no problems post-operatively. In about twenty four hours the child was noted to be completely free of the sleep apnoeic episodes. The pulmonary hypertension which existed also settled and the child was discharged well on the fourteenth day. The child was seen at the E.N.T. Department for no less than six follow-ups. The mother was indeed happy at the child's complete recovery from the sleep apnoeic episodes. The child had put on weight, was more relaxed and was able to play games.

Diagnosis

The presenting symptoms due to sleep apnoea are often remote from the primary problem. Direct enquiry is essential. By far the history is the most important. Guilleminault [2] has described the most common symptoms in great detail. The daytime symptoms such as early morning headache, easy fatiguability, mood and behaviour disturbances should alert the examiner to a possible sleep apnoea background. Loud snoring may be the first clinical symptom in most patients. In others, restlessness during sleep, somnambulance, insomnia, nightmares, nocturnal enuresis may be the other symptoms.

Physical examination is mandatory in all such cases and should exclude local causes for upper airway obstruction. The investigation should then proceed to direct observation of the sleeping patient. The nature, duration and degree of respiratory irregularities can often be well appreciated at the bedside. Other diagnostic techniques which are more sophisticated used by larger centres for evaluation of such patients include waking pulmonary function tests, sleep EEG pattern, measurement of airflow by the use of termistors, blood gas analysis, oxygen saturation to be analysed by the use of an air oximeter, continuous cardiac monitoring and the use of fibre-optic nasopharyngoscopy. Cine fluoroscopy in sleeping patients may add yet another tool in the diagnosis of sleep abnormalities and thus enable us to locate and dramatize the actual collapsing airway.

DISCUSSION

Upper airway obstruction usually produces a readily recognizable clinical picture. A chronic partial obstruction (as in this case) may however lead to a sequence of clinical events of which the root cause may easily be overlooked. Partial airway obstruction is normally evidenced during sleep and is usually episodic. Enlarged tonsils and adenoids have traditionally been evaluated on the basis of inflammatory disease. It was well documented by Menashe [4] that this partial airway obstruction due to adeno-tonsillar hypertrophy brought about pulmonary hypertension and cor pulmonale and cardiac failure in children and that this situation could be reversed by the performance of adeno-tonsillectomy. Gastaut and Tassinari [5] demonstrated that intermittent airway obstruction could produce hypersomnolence and pulmonary hypertension. He reported several patients who developed hypoxia and hypercapnoea during apnoeic episodes in sleep. Tilkian [6] showed that intermittent respiratory abnormalities during sleep could result in the same cardio pulmonary changes as are evident in chronic hypoventilation syndromes. Mangot [7] revealed that a polysomographic study could reveal irregularities during sleep prior to the development of pulmonary hypertension and cardiac failure in children with enlarged tonsils and adenoids. These sleep irregularities could be reversed following tonsillectomy and adenoidectomy. It has been suggested that this association may be important in the aetiology of the sudden infant death syndrome. [8]

Sinister cardiovascular events have been reported following upper airway obstruction produced by micrognathia [9] and nasal packing for epistaxis. [10] Others who are prone to this syndrome are those with short thich necks and generalised obesity. [11] Gastaut and Tassinari [5] did demonstrate that during sleep there is a general hypotonia in the muscles of the mouth and tongue and that the supine position aggravated it.

The secondary alveoler hypoventilation consequent on the upper airway obstruction has to be distinguished from the primary alveoler hypoventilation of patients with the Pickwickian Syndrome [12] where the respiratory movements cease due to a loss of central drive. In upper airway obstruction there is an increase in respiratory movement to try to overcome the respiratory

obstruction.

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