


- 14 **Strachan DP**. Moulds, mites and childhood asthma. *Clin Exp Allergy* 1993;**23**:779–801.
- 15 **Strachan DP**, Cook DG. Health effects of passive smoking. 6. Parental smoking and childhood asthma: longitudinal and case-control studies. *Thorax* 1998;**53**:204–12.
- 16 **Eggleston PA**. Environmental causes of asthma in inner city children. The National Cooperative Inner City Asthma Study. *Clin Rev Allergy Immunol* 2000;**18**:311–24.
- 17 **Grus CL**, Lopez-Hernandez C, Delamater A, et al. Parental self-efficacy and morbidity in pediatric asthma. *J Asthma* 2001;**38**:99–106.
- 18 **Weil CM**, Wade SL, Bauman LJ, et al. The relationship between psychosocial factors and asthma morbidity in inner-city children with asthma. *Pediatrics* 1999;**104**:1274–80.
- 19 **London Health Observatory**. Compendium, 2002. http://www.lho.org.uk/DATAANDMETHODS/Local_Data/Compendium2002.asp x (accessed 11 October 2005).
- 20 **Usherwood TP**, Scrimgeour A, Barber JH. Questionnaire to measure perceived symptoms and disability in asthma. *Arch Dis Child* 1990;**65**:779–81.
- 21 **Newson R**. Parameters behind “non-parametric” statistics: Kendall’s Tau, Somers’ D and median differences. *Stata J* 2002;**2**:45–64.
- 22 **Johnston SL**, Pattemore PK, Sanderson G, et al. Community study of role of viral infections in exacerbations of asthma in 9–11 year old children. *BMJ* 1995;**310**:1225–9.
- 23 **Murray CS**, Poletti G, Kebabdz T, et al. Study of modifiable risk factors for asthma exacerbations: virus infection and allergen exposure increase the risk of asthma hospital admissions in children. *Thorax* 2006;**61**:376–82.
- 24 **Forastiere F**, Agabiti N, Corbo GM, et al. Socioeconomic status, number of siblings, and respiratory infections in early life as determinants of atopy in children. *Epidemiology* 1997;**8**:566–70.
- 25 **Gruchalla RS**, Pongracic J, Plaut M, et al. Inner City Asthma Study: relationships among sensitivity, allergen exposure, and asthma morbidity. *J Allergy Clin Immunol* 2005;**115**:478–85.
- 26 **Rosenstreich DL**, Eggleston P, Kattan M, et al. The role of cockroach allergy and exposure to cockroach allergen in causing morbidity among inner-city children with asthma. *N Engl J Med* 1997;**336**:1356–63.
- 27 **Schatz M**, Mosen D, Apter AJ, et al. Relationships among quality of life, severity, and control measures in asthma: an evaluation using factor analysis. *J Allergy Clin Immunol* 2005;**115**:1049–55.
- 28 **Tuck M**. *How do we choose?* London: Methuen, 1976.
- 29 **Gilthorpe MS**, Lay-Yee R, Wilson RC, et al. Variations in hospitalization rates for asthma among black and minority ethnic communities. *Respir Med* 1998;**92**:642–8.
- 30 **Ray NF**, Thamer M, Fadilioglu B, et al. Race, income, urbanicity, and asthma hospitalization in California: a small area analysis. *Chest* 1998;**113**:1277–84.
- 31 **Lieu TA**, Quesenberry CP Jr, Capra AM, et al. Outpatient management practices associated with reduced risk of pediatric asthma hospitalization and emergency department visits. *Pediatrics* 1997;**100**:334–41.

LUNG ALERT

Ambulatory continuous positive airway pressure without polysomnography in patients with a high probability of obstructive sleep apnoea

▲ Mulgrew AT, Fox N, Ayas NT, et al. Diagnosis and initial management of obstructive sleep apnea without polysomnography. A randomized validation study. *Ann Int Med* 2007;**146**:157–6.

 obstructive sleep apnoea (OSA) is a common condition, diagnosed by polysomnography. However, polysomnography is costly and its availability often limited, leading to a delay in diagnosis and initiation of treatment in OSA.

This randomised, controlled study compared the use of polysomnography for the diagnosis of OSA with the use of ambulatory continuous positive airway pressure (CPAP) titration as treatment initiation in a group of patients with a high probability of moderate to severe OSA. Sixty-eight patients with a high probability of OSA were randomised to either polysomnography or ambulatory CPAP. High probability was determined by the Epworth Sleepiness Score (ESS), Sleep Apnoea Clinical Score and overnight home oximetry. The primary endpoint measure was Apnoea Hypopnoea Index (AHI) after 3 months on CPAP treatment. The secondary endpoint measures were ESS, Sleep Apnoea Quality of Life Index (SAQLI) questionnaire and compliance with CPAP.

There was no significant difference in AHI between the two groups at 3 months. With the exception of one patient in the ambulatory CPAP group (subsequently diagnosed with Cheyne-Stokes respiration on polysomnography), all patients had improvements in AHI, ESS and SAQLI. Compliance with CPAP in the ambulatory CPAP group was better, perhaps due to lower starting CPAP, earlier onset of treatment and more frequent clinic attendances.

Ambulatory CPAP may be a practical alternative in this high-risk patient group and reduce the need for polysomnography. Information regarding the cost-benefit of this approach would be helpful.

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