their numbers of abnormal tympanograms with increasing cotinine concentrations. There seems to be a trend relating increasing incidence of flat tympanograms to cotinine concentration, but there is no such trend with negative middle ear pressure. If one assumes that the same pathological process causes both negative middle ear pressure and middle ear effusion through dysfunction of the eustachian tube then the association between passive smoking and middle ear effusion is quite likely to be spurious.

It seems a pity that a paper written by an epidemiologist, a psychiatrist, and a chemist about an ear, nose, and throat condition should not have had the very necessary skills of an otolaryngologist to validate its findings.

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AUTHORS' REPLY,—Impedance tympanometry may not be the definitive diagnostic test for middle ear diffusion, but most previous epidemiological surveys have relied on this technique. Tympanometric measurement of the physical volume of the ear canal guards against the common sources of error. High values (>2·0 ml) indicate a perforated eardrum or patent ventilation tube, and in the seven children with such abnormality we conservatively chose to analyse the tympanogram from the other ear. No results were recorded from ears with blockage of the probe or low physical volume (<0·5 ml) suggesting wax. It is unlikely that flat tympanograms attributable to impacted wax could have generated a spurious association with passive exposure to smoke.

Children whose tonsils or adenoids had been removed (n=104) were at substantially higher risk of middle ear effusion (22% *v* 8%). Such a history was unrelated to the presence of smokers in the household (12% *v* 13%), so it is unlikely that previous surgical treatment affected the observed relation between middle ear effusion and passive exposure to smoke.

Different relations of passive smoking to type C and type B tympanograms might be expected if tobacco smoke affects the persistence of effusions rather than their incidence. In fact, normal (type A) tympanograms were less common in the children with higher cotinine concentrations, so that among the children without effusion there was a slightly higher risk of reduced middle ear pressure with heavy exposure to smoke (table II in our paper).

Month of examination would not affect the association between tympanometric findings and the number of smokers in the household, which was ascertained by a simultaneous questionnaire survey, but it was a potential confounding variable in our analysis of middle ear effusion and salivary cotinine concentrations. The prevalence of type B tympanograms was higher among children tested in January or February (12%) than in March or April (10%) and May or June (7%). After adjustment for sex, housing tenure, and number of smokers in the household the geometric mean salivary cotinine concentration in January and February was approximately double that in May and June. Nevertheless, the relation between middle ear effusion and the logarithm of the cotinine concentration remained significant after adjustment for month of examination (odds ratio per doubling 1·12, 95% confidence interval 1·01 to 1·25, $\chi^2=4·30$, df=1). Indeed, after adjustment for log cotinine the trend in prevalence by month of examination was non-significant (χ^2 (trend)= 1·07, df=1). Greater indoor exposure to tobacco smoke during the winter may contribute to the seasonal variation in prevalence of middle ear effusion.

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SIR,—I agree with Dr D P Strachan and colleagues that middle ear effusions in children should be recognised as one of the hazards of passive smoking.[1] It has been recognised for some time that passive smoking may have deleterious effects on the respiratory tracts of children. It is only recently that the adverse effects of passive smoking on the middle ear have been reported.

Not only is passive smoking in children associated with a higher than expected incidence of middle ear effusions and abnormal results of tympanometry[2] but these children are put at an increased risk of undergoing surgery for the condition. There is an increased incidence of grommet insertion and adenoidectomy in children whose parents smoke. Such children are twice as likely to require adenoidectomy, and their chance of requiring grommet insertion is increased by half.[3]

It is important to impress upon smoking parents that they may be subjecting their children not only to a greater risk of middle ear effusions but also to an increased likelihood of surgical intervention for the condition with the possibilities of both physical and psychological complications.

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Transurethral prostatic resection: a safe operation

SIR,—I was intrigued by Mr G Williams and colleagues' use of an expandable metal mesh stent for treating prostatic obstruction in patients considered unfit for transurethral prostatic surgery.[1] I was surprised, however, by their statement that the mortality associated with transurethral resection of the prostate has "led to a search for less invasive treatments," thus prompting this innovation.

Equally surprisingly, Mr Williams and colleagues were presented (in a short time, it would seem) with nine patients who were considered unfit for prostatic resection and hence were offered a stent as alternative treatment. Although the authors did not indicate the timespan over which these cases were collected, it could not have been very great as the first urological use of these stents was reported only in 1988. Accordingly the nine patients considered unfit for transurethral resection would seem to represent an uncharacteristically high proportion of all patients referred for prostatic surgery during this relatively short period. All this is at odds with my experience and that of my colleagues. I work in a 590 bed teaching hospital. In the 12 months to March 1989, 328 transurethral resections of the prostate were performed by the urology unit. None of the patients died, and only one patient with severe ischaemic heart disease was advised that he was unfit for surgery.

Transurethral resection of the prostate, when performed by a trained urologist, is a safe operation with a low mortality. It is rare for a patient to be considered unfit for this procedure. Operative mortality has been considerably reduced in comparison to that in studies done 15 and 30 years ago. In 1962 Holtgrewe and Valk reported a 2·5% mortality in 2015 patients; the leading cause of death was myocardial infarction.[3] Mr Williams and colleagues quote a mortality of 1·6 to 6·4% "in selected high risk groups" as evidence to "highlight the need for alternative forms of treatment."[1] Their reference uses data collected between 1965 and 1971 and reports an overall mortality of 1·3% rising to 6·4% in azotaemic patients over the age of 80.[4]

Since these data were collected some 20 years have passed, during which there has been considerable improvement in patient care. In 1989 Mebust *et al* reported a study by the American Urological Association's office of education that evaluated 3885 patients who underwent a transurethral prostatic resection.[5] The data were collected from 1978 through 1987, and 78% of these operations were performed after 1984. The mortality was 0·23% despite a 77% incidence of significant pre-existing medical problems including cardiac arrhythmias (12%), prior myocardial infarction (12%), and renal insufficiency (10%). In addition, the authors concluded that "azotaemia was not related to mortality" and "current medical practice has reduced the pre-operative problems to a non-significant level in patients undergoing transurethral prostatectomy."

I commend Mr Williams and his colleagues for their novel use of the mesh stent but contend that in a well staffed and well equipped hospital modern medicine has made prostatic resection a safe procedure even for the quite elderly and sick patient, and only very rarely will other modalities be needed to relieve a prostatic obstruction.

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Isoflurane compared with midazolam in the intensive care unit

SIR,—The letter of Drs G R Park and A M Burns raises several points.[1] The first patient they described was one of a minority of patients who are a problem to sedate in intensive care by any means in view of the amount of drugs they are given.

After the preliminary report from my unit[2] isoflurane has been used in over a thousand patients in equivalent doses to those used by Dr K L Kong and others.[3] It has proved to be excellent for sedating patients after major cardiothoracic operations for generally short periods. One patient has required the large amounts given in the two cases described by Drs Park and Burns, and other forms of sedation were then used. This patient was also a young adult. He required only six bottles each day, however, whereas in the letter the second patient required 11 to 12 bottles each day (estimated from the costs quoted).