Noise

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Exposure to Loud Noise and Risk of Acoustic Neuroma

The study finds weak evidence of a moderate link between exposure to loud noise and risk of acoustic neuroma.

This was a case control study of 146 cases of unilateral acoustic neuroma and 564 age, gender and residence matched controls (age range 20 to 69).

Acoustic neuroma (AN) is a slow growing benign tumour of the sheath of the eighth cranial nerve (hearing and balance), in close proximity to the brain. The tumour results in hearing loss, tinnitus and imbalance. If untreated, there may be complete loss of auditory function. It can be surgically removed but this often leads to permanent hearing loss through damage to the associated nerve [unless the tumour is very small] but it rarely recurs following surgery. Post op may take 2 weeks before discharge from hospital and two to three month recovery; adaptation to hearing loss and imbalance can be quite complex.

AN is first detected in one out of every 100,000 people in the general population each year, usually affecting those between 30 and 60 years of age. Around 5% of AN cases are of the neurofibromatosis type 2, inherited and usually bilateral.

Exposure to high noise levels at work will often be accompanied by health surveillance including audiology. The highly exposed will therefore have an increased rate of early detection of AN. It is usually unilateral, unlike noise induced deafness or tinnitus.

All cases diagnosed with AN, resident in a defined region of Sweden, during the study period were eligible. The region included approximately 3.1 million people. Controls were randomly selected from this region 67% of identified controls took part (18% could not be readily contacted, 15% refused to take part).

Exposure to loud noise (> 80dB(A)) was determined by interview, using references to common noise exposures to provide a sense of scale. Start date and years of regular exposure to the given noise level were ascertained by recall. In the analysis of type of loud noise exposure, the following categories were created: 1) exposure to machines, power tools, and/or construction; 2) exposure to motors, including airplanes; 3) exposure to loud music, including employment in the music industry; and 4) exposure to screaming children, sports events, and/or restaurants or bars. The remainder was classified as ‘‘other’’ types of loud noise exposure. Use of hearing protection was also recorded.

The median age of cases was 52, the incidence rate was less than 1 per 100,000. 59% of AN tumours were on the right.

Around 10% of both cases and controls made regular use of hearing protection. There was no evidence of an association between loud noise exposure and AN in this hearing protection cohort.

Among all those with regular exposure to loud noise (combined occupational or/and non occupational) the odds ratio was 1.55 (95% CI = 1.04 to 2.3). Separate analysis of occupational and non-occupational exposure groups showed no statistically significant association. Detailed analyses showed only one significant association out of 9 calculations but with a very wide confidence interval (OR = 3.3 (95% CI = 1.3 to 8.4)) for females with more than 15 years exposure to loud noise.

The data were also analyzed for those study participants with less than 13 years, 13–26 years, and 27 or more years since the first regular loud noise exposure. The odds ratio for 13 or more years [i.e. 13-26 combined with 27+] since exposure began was 2.1 (95% CI = 1.4 to 3.2). The result was not affected by use or non use of mobile phones or by medical history of exposure to ionising radiation (a known risk factor).

Comment
In our view, most of the evidence presented for an association between loud noise exposure and risk of AN is very weak, the great majority of it failed to reach statistical significance. Recall bias must be considered a major deficiency of this study.

The exception is the analysis of the effects of latency. In this analysis, latencies of between 13 and 26 years and > 26 years were both significantly associated with AN; odds ratios of 1.7 and 2.1 respectively. This pattern is as would be expected if the association was causal. However, given that the incidence of

AN is age-dependent a small inconsistency in methodology or bias in case ascertainment could lead to this finding. It would also be very difficult to explain why AN is usually unilateral.

AN is a serious, but relatively rare disease.
Exposure to loud noise at work is commonplace; HSE estimate that more than 1 million UK workers are exposed to loud noise at work. Hearing difficulty is most prevalent in transport and machinery operatives, construction workers, material moving and storage workers and repetitive assembly and inspection workers; and in women, among cleaners and caterers. In construction workers, the prevalence of moderate or worse hearing difficulty is 11.5% (vs. 5% for all occupations), and that of severe hearing difficulty is 5% (vs. 1.9% for all occupations).

A remote possibility is that those with occupational noise induced deafness (est. 170,000 working people in the UK) would consider themselves at significant risk of AN (following the precedent set for plaques and mesothelioma). Industrial injuries disablement benefit claims for occupational noise induced hearing loss number approximately 300 per year.