



Snoring, sleep Apnoea and Pre-eclampsia

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Debbie Chippington Derrick reviews the possible links between pre-eclampsia and sleep problems.

Whilst reading a book on sleep¹ recently I came across the references for two papers that intrigued me. Both were related to pre-eclampsia and sleep problems. Both were published relatively recently and yet I had never heard of them, which I found surprising. Part of the reason for this would appear to be that they had both been published outside the childbirth related journals. Having read these two papers I then did a search to see if there were more on the same subject and there were, although I have not included papers published before 1990 in this review.

The first paper² had been written by a group from the University hospital in Ume. who had been studying the relationships between snoring, high blood pressure and fetal growth retardation in pregnancy. It highlights the known connection between snoring, sleep apnoea and hypertension and points out that hypertension is part of the collection of symptoms defining pre-eclampsia. The authors also acknowledge that airway narrowing is a known cause of snoring and there are known mechanisms for this occurring during pregnancy.

Sleep apnoea is where the sleeper stops breathing due to an obstructed airway, the inability to breathe then rouses the sleeper, who then breathes again usually taking in a deep and often noisy breath. The sleeper is likely to remain unaware of this arousal, although it is common for a sleeping partner to be disturbed by it. This pattern usually repeats meaning that the sleeper is constantly disturbed by these awakenings and unable to get into the essential deeper sleep, leaving the sleeper constantly sleep deprived. There is a lesser event that is called hypopnoea where breathing continues but the flow of air is considerably reduced.

Midwives administered questionnaires about women's sleeping habits and any sign of pre-eclampsia. This was carried out on the day of the birth for 518 women in a Swedish hospital. However, women who had caesareans, a baby that died or twins were not included in the study. As a rule the woman was accompanied by her partner when answering the questions (probably very helpful when trying to identify frequency of snoring).

Snoring became more common in the last three months of pregnancy, when 23% of women snored habitually and 25% occasionally, whereas before they were pregnant only 4% snored regularly and 22% occasionally. Sleep apnoeas had been observed in 11% of the habitual and 2% of the occasional snorers. Increases in snoring were noted throughout pregnancy - with 7%, 6% and 24% noting marked increases

in the first, second and third trimesters respectively.

The results showed that 14% of the habitual snorers had pregnancy-induced hypertension compared to 6% of the nonsnorers, whilst 10% and 4% met the criteria for pre-eclampsia. All the pre-eclamptic women snored, and had started to snore during the pregnancy and before any of the pre-eclamptic symptoms were seen. Witnessed sleep apnoeas were reported in 12% of the pre-eclamptic women and 10% of those with hypertension. The prevalence of daytime sleepiness in pregnancy was about 65% and similar in those who did and did not snore. Oedema occurred in 52% of the habitual snorers, compared with 30% of the occasional snorers; with facial oedema occurring in 27% compared to only 10%. It was also found that 7% (8/113) compared to 2.6% (10/379) of the babies born to habitual snorers were considered small for gestational age, and the difference was significant even when correcting for weight, age and smoking habits. (Smoking was found to be an independent risk factor for babies being small for gestational age). Apgar scores <7 were more common in the habitual snorers with 12% and 3.6% at 1 and 5 minutes and 3.5% and 0.3% for the occasional snorers. Witnessed sleep apnoeas did not relate to infant outcome (however, the study excluded caesarean born babies and babies that died).

The authors suggest that respiratory sleep studies, including the treatment of sleep apnoea in women with pre-eclampsia, are desirable and may answer whether there is causal effect. Another paper³ reported research carried out in collaboration with groups of women in Sydney, Australia and Turku, Turkey. This addressed the issue of whether treatment for sleep apnoea had any effect on pre-eclampsia. In reviewing the previous evidence the paper reports that rises in blood pressure with pre-eclampsia are greater during the night than during the day.

The study was carried out on 11 women with severe preeclampsia; all were on the hospital ward due to the severity of their condition. Two had hypertension before the pregnancy and one was asthmatic, the other women had been well before the pregnancy. All the women were being given drugs to treat the hypertension.

Measurements of blood pressure (continuously measured); air flow through the nose and oxygen saturation were taken on two consecutive nights. Polyomnography was carried out on 7 of the women. (A polyomnograph tests sleep cycles and stages using continuous recordings of brain waves, electrical activity of muscles, eye movement, breathing rate, blood pressure, blood oxygen saturation, heart rhythm and direct observation of the person during sleep.)

None of the women studied had sleep apnoea (a complete obstruction to the airflow for at least 10 seconds); but all had hypopnoea (where there is at least 50% reduction in airflow for at least 10seconds). All women were given Continuous Positive Airway Pressure (CPAP) treatment.

The women still slept for similar amounts of time as they did before the treatment. Blood pressure rises which had been seen in the women during the night were prevented with the treatment, and blood uric acid levels (which are quoted as being an "important predictor of severity and outcome of pre-eclampsia") were reduced in all the women. The paper questions whether the oedema in the upper

airway causing the hypopnoea is brought on by the preeclampsia, which then causes oedema in other organs in the body; or whether the hypopnoea occurs commonly in normal pregnancies.

Two case studies from Canada⁴, and America⁵ report on women who were found to have sleep apnoea and were given CPAP treatment, but only during the last trimester of pregnancy and duration of treatment is not clear. In both cases treatment was seen to improve the oxygenation of the women's blood. Both babies were born growth retarded and the papers suggest that earlier treatment may prevent such problems.

Three further studies from Scotland^{6, 7} and Australia⁸ also found connections between the incidence of snoring, sleep apnoea, upper airway dimensions, hypertension and pre-eclampsia.

Although some of the papers acknowledged the correlation between snoring, sleep apnoea and pre-eclampsia with being over weight or obese, the causal relationships between these were not addressed.

AIMS Comment

We are aware that there is a reluctance to diagnose and treat sleep apnoea and hypopnoea in the general population, and that a significant number of road traffic accidents may be caused by undiagnosed or untreated cases. It is concerning that there seems to be little interest within the obstetric profession in a simple low risk and drug free treatment that may be helpful to women who are snoring, suffering from sleep apnoea or have pre-eclampsia; as pre-eclampsia is a serious condition which can kill both mothers and babies.

References

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