Persistent increase in caffeine concentrations in people who stop smoking

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Cigarette smokers on average drink more coffee than non-smokers and can metabolise caffeine much more rapidly. One possible explanation for their increased consumption is that smokers need to drink more coffee to achieve the desired concentrations of caffeine in the body and hence the desired effects. When smokers stop smoking the rate at which caffeine is metabolised declines. If consumption of caffeine remains the same concentrations of caffeine in the body should rise. Persistent increases in caffeine concentrations may cause concern in view of recent data showing an increased risk of coronary heart disease in those who used to drink coffee but who have recently given it up.

The trends suggest that caffeine concentrations increased in those who gave up smoking and continued smoking.

Subjects, methods, and results

Ninety five healthy subjects were recruited for a stop smoking programme. The subjects' average age was 38 (range 19-60); 38 were men, 87 were white, and almost all were middle or working class. Subjects were assessed before entry to the programme and 12 and 26 weeks afterwards. Assessment included analysis of plasma samples for cotinine (a primary metabolite of nicotine used to indicate nicotine intake) and caffeine concentrations, body weight, and a three day record of weighed food intake.

Sixty eight subjects completed the study. Data were available on 64 subjects at 12 weeks and 61 subjects at 26 weeks. The subjects who subsequently continued smoking were significantly older (mean 42.8 ± 36.2 years) but cigarette consumption, numbers of years of smoking, and plasma caffeine and cotinine concentrations were not significantly different between those who gave up smoking and those who continued smoking. At 12 and 26 weeks plasma caffeine concentrations in subjects who stopped smoking were more than double the baseline value (table). Caffeine concentrations in smokers changed little throughout the study. Subjects who stopped smoking had significantly decreased their caffeine consumption at 26 weeks.

Comment

Our study confirms the hypotheses that plasma caffeine concentrations increase after people give up smoking and remain increased for at least six months. This increase was substantial, averaging more than 250%. In subjects who continued to smoke caffeine concentrations were unchanged, as expected.

The average caffeine concentration in the body is determined by the consumption and rate of metabolism of caffeine. Consumption was either unchanged (at 12 weeks) or decreased (at 26 weeks) in subjects who stopped smoking. Thus the increased caffeine concentrations were the result of slowed metabolism due to abstinence from tobacco. Caffeine consumption was significantly decreased at 26 weeks, which is consistent with a degree of self regulation of caffeine concentrations. Regulation of caffeine concentrations could explain why cigarette smokers drink more coffee. As they metabolise caffeine more quickly, the half life of caffeine is shorter and the desired effects of the caffeine disappear or symptoms of withdrawal appear more quickly. Smokers would need to drink coffee more frequently to maintain caffeine concentrations similar to those in non-smokers. The rate of metabolism of caffeine decreases within three or four days after a subject stops smoking. Assuming that consumption of caffeine is unchanged, high caffeine concentrations could contribute to the tobacco withdrawal syndrome.

An increase of 250% in caffeine concentrations is...
Identifying hypoxaemia in children admitted for adenotonsillectomy

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Most children with obstructive sleep apnoea have adenotonsillar hypertrophy, but what proportion of the children with large tonsils and adenoids have obstructive sleep apnoea is unknown. We studied this and whether affected children might be identified without recourse to studies during sleep.

Subjects, methods, and results
Forty-four children (aged 2 to 7) were studied on the night before adenotonsillectomy. Twenty controls with no active symptoms of upper or lower respiratory tract disease were studied on the night before another elective procedure. Both groups underwent a preoperative clinical assessment by an otorhinolaryngologist, who noted symptoms, signs, and the indication for adenotonsillectomy.

Oxygen saturation was measured continuously overnight with a pulse oximeter (Novametrix). Chest impedance was also measured and electrocardiography performed. The baseline oxygen saturation during sleep was obtained by sampling the saturation three times an hour outside dips. Dips in the saturation of 10% or more below the baseline value were studied. A stepped fall in saturation with steady pulse waveform and continued breathing movements suggested obstruction of the airways. These episodes were counted as incidents per hour and their length noted as time of hypoxia per hour. Oxygen saturation when the child was awake was noted at the beginning or end of the night.

The figure shows the full results of measuring oxygen saturation. For all measures of hypoxaemia a substantial minority of the patients admitted for adenotonsillectomy fell completely outside the normal range. Fifteen children who suffered night time hypoxaemia were identified, with baseline oxygen saturation below 90% (nine patients) or at least one hypoxic episode an hour (13), or both (seven). Twelve children had episodes of hypoxia (totaling >1 minute per hour). The 15 children who suffered night time hypoxaemia were then compared with the remaining 29 from the group admitted for adenotonsillectomy.

There were no notable differences between the groups regarding history of smoking, sex, age, family history of snoring or sleep disturbance, body weight, or any features found on examination of ears, palate, mandible, tonsils, or neck glands. The clinical features that did distinguish the hypoxic group were younger age (median 3.5 vs 5.6 years; p<0.01), non-white ethnic origin (80% vs 12% (12/7/29); p<0.001), and physical signs of nasal obstruction—namely, open mouth and audible respiration at rest (93% vs 14% (11/29); p<0.001). In addition, oxygen saturation when the patient was awake was lower (median 95% vs 97%; p<0.001).

Hypoxic patients could not be identified by clinical features alone, but by combining the presence of absence of breathing through the mouth with the oxygen saturation when awake we identified affected patients with 93% sensitivity (95% confidence interval 70 to 100%) and 86% specificity (73 to 99%).

Comment
We showed that significant night time hypoxaemia is common, occurring in 34% of children admitted for adenotonsillectomy. We have clearly defined the importance of breathing through the mouth as a clinical sign of obstruction and the usefulness of measuring oxygen saturation when awake. Children who clearly breathe through the mouth will suffer night time hypoxaemia unless their oxygen saturation when awake is at least 97%. We hope that this will help paediatricians and otorhinolaryngologists to assess children correctly and avoid unnecessary operations.