Does caffeine affect breathing and contribute to sleeping problems?

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Introduction

Caffeine is the world's most widely consumed psychoactive agent and is used for both medical and non-medical purposes due to it's stimulatory and somnolytic effects (Daly 1993). Purified caffeine is used medically in asthma preparations, cold medications and analgesics as well as for fatigue (e.g. No Doz). The warm beverages coffee and tea are natural sources of caffeine while cold carbonated beverages, such as cola and "energy drinks", contain purified caffeine most often derived from coffee beans and tea leaves. On average a 150ml cup, i.e. one serving, of drip or percolated coffee contains 120mg caffeine, instant coffee 70mg and tea 50mg, cold carbonated beverages contain 30-60mg caffeine per 350ml serving (Benowitz 1990).

The medicinal use of caffeine for asthmatic conditions is based on studies showing that caffeine increases breathing rate. It has been proposed that the increase in breathing rate is due to caffeine sensitising the respiratory centre in the medulla to carbon dioxide (Benowitz 1990). A number of local mechanisms in the respiratory tract have also been suggested as implicated in the increased breathing rate (Daly 1993). Caffeine's effectiveness as an asthma treatment has recently been reviewed by the Cochrane Collaboration (Bara and Barley 2001). This review reported that caffeine, in doses of 5-10mg/kg body weight, is a bronchodilator which can modestly improve asthmatics lung and airway function for 2-4 hours after consumption. However a recent study reported that 250mg caffeine capsules had no effect on the breathing rate of healthy habitual caffeine users in the seated position (Barry et al. 2005) and this is contrary to a previous study, also using 250mg caffeine capsules, which reported a 20% increase in breathing rate for supine participants who had not consumed caffeine in any form for 3 weeks (Robertson et al. 1978).

This study aimed to test whether caffeine used non-medicinally at doses experienced by coffee and tea drinkers could influence breathing rate and so contribute to insomnia or other sleeping problems. Only regular consumers of coffee and tea were tested and only at caffeine levels obtained by drinking one or two cups of tea or coffee.

Method

Testing was conducted at the Polyclinic, University of Westminster, London, using a randomly assigned, crossover, double blinded experimental model with the approval of the university's Ethics Board. Participants were voluntarily recruited from students and staff at the School of Integrated Health. They were required to be daily consumers of caffeine as either tea of coffee and healthy (i.e. not taking medication). Smokers and pregnant women were excluded. Participants were tested at 4 different sessions with capsules containing either 0mg (placebo), 67mg 133mg or 200mg caffeine. Caffeine capsules were alphabetically coded in sets by a member of the Polyclinic, and participants choose their own set. Normally there was a period of 4 weeks between test sessions and sessions were held at a similar time to minimise diurnal effects. Room temperature was maintained at 22-25°C. Participants were required to abstain from food and caffeine containing substances for 2 hours before testing and not to engage in any heavy or unusual exercise for 24 hours before testing. Compliance to instructions was assessed at the beginning of each session. Participants were required to give informed written consent at their first session and undergo the General Health Questionnaire at the beginning of each session. 14 participants started in the experiment of which one dropped out due to scheduling problems.

Beat-to-beat finger arterial pulse contour measurements were made with Finometer© (FMS, Amsterdam) and a Biopac (USA) Respiratory Effort Transducer© (TSD201) was used to record thoracic movements. The respiratory transducer was connected to a Finometer external port for recording purposes. 90 second recordings were made with eyes closed to minimise sensory input (Barry et al.). No instructions were given regarding breathing. After the baseline recording a caffeine capsule was administrated with water. Further recordings were made at 30 minutes and 60 minutes after caffeine ingestion. Breathing frequency was calculated from the number of full oscillations in the recording. Due to equipment problems the respiratory transducer recordings were not available for approximately half of the test sessions. When the direct recordings of thoracic movement were not available breathing frequency was derived from stroke volume oscillations, see Figure 1, using a technique developed by the authors (McMullen et al. 2007)

Breathing frequency measures for the caffeine capsules were adjusted by dividing by the placebo measures to allow for the baseline drift between test sessions. Prior to analysis two participants were rejected. One was a non-medicated asthmatic and the other had irregular heart function at the placebo session. A further participant was excluded after examination of the results with box plots (SPSS v15) revealed 3 extreme values for this participant – see Figure2 . Measures from 10 participants (mean age 36, 8 women) were analysed.

For the analysis baseline frequencies were contrasted (planned) with both 30 and 60 minute frequencies at each caffeine level using repeated measures ANOVA (SPSS v15). *F* tests reported have (1,9) degrees of freedom. If *F* tests were significant then within-subject *t* tests would be used with Type I error (**a**) restricted using Holm's multistage Bonferroni adjustment (Howell 2007).

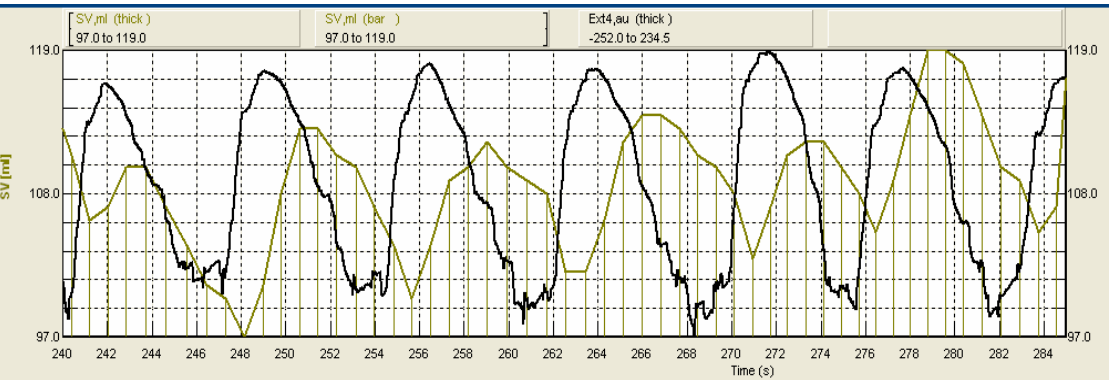

Figure1. Stroke volume (brown line) oscillates at the same frequency as breathing frequency (black line).

	Test time	*F*	*p*
Caffeine 67mg	30 minutes	1.156	0.310
	60 minutes	1.547	0.245
Caffeine 133mg	30 minutes	0.588	0.463
	60 minutes	2.222	0.170
Caffeine 200mg	30 minutes	0.607	0.456
	60 minutes	3.315	0.102

Table 2. *F* and *p* values for the within-subject contrasts

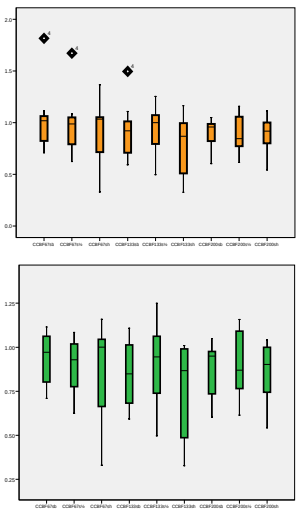

Figure 2. Upper: boxplot with extreme values. Lower: boxplot after removal of the participant with extreme values

Condition	Mean	Std. Dev.
67mg baseline	.93	.15
67mg 30 minutes	.90	.16
67mg 60 minutes	.86	.27
133mg baseline	.85	.19
133mg 30 minutes	.89	.24
133mg 60 minutes	.74	.27
200mg baseline	.88	.16
200mg 30 minutes	.91	.18
200mg 60 minutes	.86	.17

Table 1. Placebo adjusted breathing frequencies

Results

Descriptive statistics for placebo adjusted frequency measures are presented in Table 1. *F* and *p* values for the ANOVA testing of the within-subject contrasts are presented in Table 2. These tests show no significant changes in breathing frequency due to caffeine ingestion in any of the conditions

Conclusion

The experiment shows no effect of caffeine (67, 133 and 200mg) ingestion on the breathing frequency of habitual caffeine users in the supine position. These doses represent the amount of caffeine obtained by normal consumers of coffee and tea. This result is consistent with earlier results on habitual caffeine users (Barry 2005) but not with results obtained with non-habitual caffeine users (Robertsson 1978). The outcome of caffeine experiments is often dependent on whether the participants are habitual users or non users of caffeine. Where real-life experiments on caffeine are used there are few negative effects and caffeine is now viewed as a substance that regulates sleep rather than disturbs sleep (Snel et al. 2004). From these results it is not possible to speculate on the effect of caffeine on asthma suffers for two reasons. Firstly only people with normal breathing were tested and secondly the amounts of caffeine administered were lower than has been used in asthma research.

In conclusion the present study indicates that it is unlikely that the regular use of caffeine or caffeine containing beverages before sleeping will cause breathing rate changes that will affect sleep patterns.

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