the logistic model was tested according to Hosmer and Lemeshow

There were no significant differences between the two control groups with any of the risk factors considered. Body mass index was 25.55 (SD 3.59) in patients; 26.20 (3.63) in the general population controls; and 25.47 (5.15) in hospital controls. Comparing patients with each control group we found a significantly increased risk of myocardial infarction among patients who snored every night (table). Multiple logistic regression analysis showed that this association was independent of other risk factors.

Discussion

We chose to see if there was a correlation between the risk of having a myocardial infarction and snoring every night because sleep apnoea is probably more common in people who snore. A study among the general population found that out of 40 people who snored every night 25 had a sleep apnoea index over 5. People who snore every night might be at an increased risk of having a myocardial infarction because they are more commonly affected by sleep apnoea, which might put chronic stress on the cardiovascular system. Snoring in itself might, however, cause cardiovascular stress as shown by Lugaresi et al.3 Cross sectional and longitudinal studies are required to verify whether there is a causal association between snoring, sleep apnoea, and myocardial infarction. If such an association is confirmed then some cases of myocardial infarction might be prevented by treatment of snoring and sleep apnoea.4

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- 1 Waller PC, Bhopal RS. Is snoring a cause of vascular disease? An epidemiological
- review. Lancet 1989;i:143-6.
 2 Cirignotta F, D'Alessandro R, Partinen M, et al. Prevalence of every night snoring and obstructive sleep apnoeas among 30-69-year-old men in Bologna, Italy. *Acta Neurol Scand* 1989;**79**:366-72.
- 3 Lugaresi E, Coccagna G, Farneti P, et al. Snoring. Electroencephalog Clin Neurophysiol 1975;39:59-64.
- 4 Lombard RM Jr, Zwillich CW. Medical therapy of obstructive sleep apnea. Med Clin North Am 1985;69,6:1317-33.
- 5 Sullivan CE, Issa FG, Ellis E, et al. Treatment of cardiorespiratory disturbances during sleep. Interdisciplinary Topics in Gerontology 1987;22:47-67.

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Headache caused by caffeine withdrawal among moderate coffee drinkers switched from ordinary to decaffeinated coffee: a 12 week double blind trial

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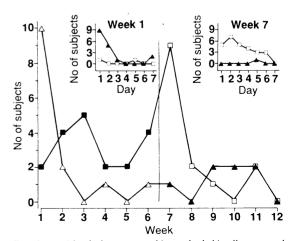
Caffeine withdrawal could be an important but often overlooked cause of headache. A study of 205 hospital inpatients found a significantly higher caffeine consumption among patients who reported postoperative headaches than those who did not (mean consumption of caffeine 528 v 339 mg/day). Information from controlled trials on caffeine withdrawal, however, is limited: published studies have used high doses or short observation periods or have incompletely controlled caffeine intake.23 We report on the withdrawal effect of caffeine in healthy subjects who habitually consumed four to six cups of coffee a day. The main results of this study, which was designed to compare the effects of ordinary and decaffeinated filter coffee on blood pressure and serum cholesterol concentration, are reported elsewhere.41

Subjects, methods, and results

We recruited subjects through stories in local newspapers. Of 150 applicants, 45 (23 women and 22 men, aged 25 to 45) met all the criteria for eligibility: aged 17-45, apparently healthy, serum cholesterol concentration <6.7 mmol/l, systolic blood pressure <140 mm Hg, abstinence from smoking for the past year, not taking drugs or oral contraceptives, not taking a prescribed diet, not pregnant, not working night shifts, and habitually consuming four to six cups of filter coffee a day as measured by a three day dietary record. After being matched for sex, blood pressure, and age the subjects were randomly allocated to receive either five cups of coffee (84 mg caffeine/cup) each day for six weeks followed by five cups of decaffeinated coffee (3 mg caffeine/cup) for the next six weeks (n=23) or the reverse treatment (n=22).4 Blank coffee cartons were labelled with the subjects' names by two people not participating in the trial; both subjects and investigators were blind to the type of coffee being consumed. Subjects were unaware whether they were being switched between ordinary and decaffeinated coffee.

Subjects were prohibited from consuming tea or other products containing caffeine, except for small amounts of chocolate. The mean caffeine intakes were 435 mg/day for ordinary coffee treatment and 30 mg/day for decaffeinated. Once a week subjects rated how easily they had fallen asleep the previous night, by placing a cross on a 100 mm bar scale running from with great difficulty (0) to very easily (100). Subjects recorded any sign of illness, and daily guesses about which type of coffee they were receiving.

Thirty eight of the 45 subjects did not realise when the coffee was switched to decaffeinated. Nineteen subjects recorded more complaints about headache during their first week of taking decaffeinated coffee compared with the mean number of complaints they recorded during the 11 other weeks (figure); five subjects recorded fewer complaints, and 21 showed no



Prevalence of headaches among subjects who habitually consumed four to six cups of coffee a day and were switched from ordinary to decaffeinated coffee at start of study (week 1; \triangle) or week 7 (\square). ▲, ■ Indicates consumption of ordinary coffee

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BMI VOLUME 300 16 IUNE 1990 change (signed rank test, p=0.0006). The headaches started on the first or second day that the subjects took decaffeinated coffee and lasted for one to six days, with a mean duration of 2.3 days. Subjects also reported falling asleep more easily when they were consuming decaffeinated coffee (mean scores $88 \ v \ 85$; paired t test mean difference 3.0, 95% confidence interval 0.2 to 5.8). Thus caffeine affected quality of sleep even in moderate coffee drinkers.

Comment

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Our results indicate that many moderate consumers of coffee develop headaches caused by caffeine withdrawal lasting two to three days after they switch to decaffeinated coffee. The result was not influenced by subjective expectations as most subjects were unable to recognise the switch. Thus caffeine withdrawal headache is not restricted only to high consumers. Clinicians should be aware of caffeine withdrawal as a possible cause of headaches, especially when ingestion of caffeine is erratic.

- Galletly DC, Fennelly M, Whitwam JG. Does caffeine withdrawal contribute to postanaesthetic morbidity? *Lancet* 1989;i:1335.
 Goldstein A, Kaizer S, Whitby O. Psychotropic effects of caffeine in man. IV.
- Goldstein A, Kaizer S, Whitby O. Psychotropic effects of caffeine in man. IV. Quantitative and qualitative differences associated with habituation to coffee. Clin Pharmacol Ther 1969;10:489-97.
- 3 Dreisbach RH, Pfeiffer C. Caffeine-withdrawal headache. J Lab Clin Med 1943;28:1212-8.
- 4 Van Dusseldorp M, Smits P, Thien Th, Katan MB. Effect of decaffeinated versus regular coffee on blood pressure. A 12-week, double-blind trial. *Hypertension* 1989;14:563-9.
- 5 Van Dusseldorp M, Katan MB, Demacker PNM. Effect of decaffeinated versus regular coffee on serum lipoproteins: a 12-week double blind trial. Am J Epidemiol (in press).

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A simple method to detect Helicobacter pylori in gastric specimens

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Most diagnostic tests for the presence of *Helicobacter pylori* (formerly *Campylobacter pylori*) in the stomach either require histological examination of samples of the gastric mucosa epithelium obtained by gastroscopy (brushing or biopsy)¹ or detect urease activity in the gastric lumen.² We report our experience of using a modified gelatin capsule containing a nylon string to collect gastric specimens.

Patients, methods, and results

The study protocol was approved by our institution's human subjects committee, and each patient gave his or her written consent to participation. Sixty adult Guatemalans (39 men and 21 women aged 21-67 (mean 46) were studied.

The length of the string contained in weighed gelatin capsules (Enterotest, Hedeco, United States) was reduced to 68 cm. Patients swallowed the sterilised string device at 7 am after overnight fasting, and it was recovered four to five hours later. We obtained smears from the mucus on the distal 10 cm of the string before placing the string in a tube with 1 ml of urea broth (Difco, United States).³ The next day biopsy specimens from the gastric mucosa were obtained by gastroscopy (four from the antrum, two from the corpus, and two from the fundus). Mucus smears and cultures (campylobacter media, E Merck, West Germany) were obtained from one antral biopsy

Results of analysing gastric specimens obtained by biopsy or with string device for Helicobacter pylori, using smear staining and urea broth

	String				Biopsy			
	Smear		Urea broth		Smear		Urea broth	
	Positive*	Negative	Positive	Negative	Positive	Negative	Positive	Negative
Patients positive for								
H pylori (n=39)	37	2	36	3	38	1	37	2
Patients negative for								
H pylori (n=19)		19	1	18		19	1	18
Sensitivity (%)	95		92		97		95	
Specificity (%)	100		95		100		95	
Predictive value of								
positive result (%)	100		97		100		97	

^{*}Patients were classed as positive for *H pylori* if the organism was detected in cultures of biopsy specimens or in histological sections, or both.

specimen, and another was placed in a tube with 0·5 ml of urea broth and crushed with the base of a sterile swab stick. The remaining specimens were sent for histopathological studies. Smears were stained with Gram and fuchsin stains. We used standard methods for culturing and identifying *H pylori*. Any colour change (to purple) in the urea broth cultures was assessed after incubation for 24-48 hours at 36°C. Histological sections were stained with Giemsa. Patients were classified as positive for *H pylori* if the organism was isolated from cultures of biopsy specimens or spiral organisms were present in histological sections, or both.

Two patients were unable to tolerate the swallowed string device. The table shows the results from the other patients. Biopsy cultures and histological sections were negative for *H pylori* in 19 cases. In one case mucus smears obtained from the string and biopsy specimens yielded negative results but the urea broth test vielded a positive result; this was due to large amounts of bacteria in the gastric mucus. In three cases mucus smears (two from the string, one from a biopsy specimen) gave false negative results owing to scant material. The three false negative results of urea broth tests on string specimens were caused by bile deeply staining the broth and masking any changes in colour. Slight to moderate staining with bile did not interfere with the reaction. No explanation was found for the two false negative results of urea broth tests on biopsy specimens. The sensitivity, specificity, and predictive value of a positive result of tests on the string specimens were similar to those obtained with antral mucosa biopsy specimens.

Comment

The viability of the organisms recovered with the string device is not known, and we are currently investigating this. Evidently, however, the string is an inexpensive, rapid, and effective method of detecting *H pylori* in gastric contents. It will be of great help in following up patients after treatment or in cases in which samples of gastric mucosa cannot be obtained by endoscopy.

- 1 Goodwin CS, Armstrong JA, Marshal BJ. Campylobacter pyloridis, gastritis and peptic ulceration. J Clin Pathol 1986;39:353-65.
- 2 Bell GD, Weil J, Harrison G, et al. 14 C-Urea breath analysis, a non-invasive test for Campylobacter pylori in the stomach. Lancet 1987;i:1367-8.
- 3 Alemania RF, ed. Manual de medios de cultivos Merck. Darmstadt: E Merck, 1987:169.
- 1987:169.
 McNulty CA, Wise R. Rapid diagnosis of Campylobacter pyloridis gastritis. Lancet 1986;i:387.
- 5 Goodwin CS, Blincow ED, Warren JR, et al. Evaluation of culture techniques for isolating Campylobacter pyloridis from endoscopic biopsies of gastric mucosa. J Clin Pathol 1985;38:1127-31.

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