The relationship between smoking and migraine

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Summary

The theory that smoking could be implicated in the pathogenesis of migraine has been investigated in a prospective survey by the use of a questionnaire and the measurement of carboxyhaemoglobin (COHb) levels in patients attending a migraine clinic. There was a low incidence of smokers in patients attending for routine consultations and only a small percentage of patients thought that smoking was a cause of their headaches. This percentage was approximately the same both for migraine and for tension headaches. In patients attending with acute headaches, the incidence of smoking was also low, and raised COHb levels were found only in the smokers, most of whom did not consider smoking a cause of their symptoms. It is concluded that smoking is unlikely to be a factor in the aetiology or the exacerbation of migraine.

Introduction

It has been shown that carbon monoxide intoxication is associated with headache (Forbes, 1970) and that inhalation of tobacco smoke (which contains up to 5% carbon monoxide) (Wynder and Hoffman, 1967) raises the plasma carboxyhaemoglobin level (COHb) (Castleden and Cole, 1974). When this level is raised there is relative anoxia as oxygen has been displaced from haemoglobin (Astrup, 1972). Since anoxia causes vasodilatation it is possible that smoking could be correlated with migraine in certain sensitive individuals.

There are no published reports of smoking as a precipitating factor in migraine but Astrup has shown that smoking or inhalation of carbon monoxide can precipitate the symptoms of mountain sickness, in which headache is a prominent symptom (Astrup, Kjeldsen and Siggaard-Anderson, 1971). Furthermore, headache is common in atopic subjects with tobacco sensitivity (Zussman, 1970) and smoking was associated with increased menstrual pain in a group of subjects in whom there was also

a high incidence of menstrual headaches (Procope and Timonen, 1971).

Our clinical experience suggested that some migraine patients associated smoking with either the onset or exacerbation of their symptoms and there also appeared to be a low incidence of smoking in these individuals. We therefore undertook a prospective survey to determine the incidence of smoking in migraine patients and to ascertain whether smoking and/or elevated COHb levels could be implicated in the pathogenesis of migraine or other headaches.

Methods

The study was in two parts:

(a) One hundred consecutive new patients attending a migraine clinic for routine consultations with one physician (G.N.V.) answered a short questionnaire, which was incorporated into the customary interview, on their smoking habits.

(b) Fifty patients who attended the same clinic for treatment of acute headaches were asked the same questions. In addition, the duration of symptoms before arrival at the clinic and the time of the last cigarette were recorded. Before the patient received treatment, a venous blood specimen was collected into a heparinized syringe, capped, and analysed for COHb on the same day by means of a CO-oximeter (Instrumentation Laboratories—model 182). Cross checks for accuracy were made on each batch of samples by the Commings and Lawther method.

All diagnoses of migraine conformed to the classification of the World Federation of Neurology (1970).

Results

Routine consultations

Table 1 shows that of one hundred new patients (twenty-four male, seventy-six female; mean age $\pm$ s.d. = 40 ± 12 years), twenty were currently smokers whilst twenty-four were ex-smokers. Nine patients

TABLE 1. The claimed relationship of smoking habits to headaches in 100 consecutive new patients attending a migraine clinic

Smoking habits	Sex	Cause headaches	Effect of smoking or smoke on headaches	
			Increase severity of headaches	No effect on headaches
Smokers (20)	M 12	2 } 5	4 } 9	8 } 11
	F 8		5 } 5	3 } 3
Ex-smokers (24)	M 10	2 } 4	2 } 7	8 } 17
	F 14		5 } 5	9 } 9
Never smoked (56)	M 2	—	0 } 17	2 } 39
	F 54		17 } 17	37 } 37
Totals (100)	M 24	4 } 9	6 } 33	18 } 67
	F 76		27 } 27	49 } 49

thought that smoking actually caused headaches. Only one gave up smoking because of this, and five of the nine are still currently smoking. Thirty-three patients reported that smoking or smoke exacerbated their headaches. Seventeen of these were women who had never smoked. Smoking or smoke was reported as a possible cause or exacerbating factor in approximately equal percentages of patients whether the headache was migraine or tension headache (Table 2).

Acute headaches

Of fifty patients (twenty male, thirty female; mean age $\pm$ s.d. = 35 ± 12 years), seventeen were currently smokers (eight male, nine female). Only eleven patients thought that active or passive smoking was a factor in the causation or exacerbation of their headaches and, of these, six had never actively smoked.

The mean COHb level of the smokers was 4.9% (range 0.1–7.9%) and of the non-smokers was 1.9% (range 0.9–3.8%). The highest COHb levels were

found in the patients who had smoked most recently but there was no obvious correlation between the duration of the attack and COHb levels.

Discussion

The patients in this study were divided into two groups, the 'cold cases' attending for consultation at a time when they were symptom-free and the 'acute cases' who attended for treatment during migraine attacks. If the theory that smoking caused headaches was correct it would have been expected there to be few smokers amongst the cold cases and both a higher incidence of smokers and a high mean COHb level in the acute cases. In fact, a low incidence of smoking in both groups of patients was found. Even though the majority of patients attending for consultation were female (76%), there were still far fewer smokers among them (20%) than it has been reported from random populations matched for age and sex in America (38%) or in Britain (50%) (Report of Surgeon General, 1971; Report of Royal College of Physicians, 1971). Furthermore, 24% of the subjects were ex-smokers whereas in an American survey only 13–15% had given up cigarette smoking (Report of Surgeon General, 1971). Hence, the incidence of smoking in the patients of this study was less than half that of the general population, and approximately twice as many patients had given up smoking than could be expected in the population at large.

If the cold case study suggests a correlation between smoking and migraine, the acute case study does not bear this out. Although 34% of subjects presenting with acute migraine had a raised COHb level indicating recent tobacco smoke inhalation, 66% did not, and their symptoms were no different from those of the smokers. As a group, the acute patients had a very low incidence of smoking (34%) when compared to other groups of workers in the City of London (Castleden and Cole, 1975). Random checks on the COHb level of manual and office workers showed that 68% had a raised COHb level, whilst the incidence in blood donors—a group with a high proportion of non-smokers—was 44% (Castleden and Cole, 1975).

Hence, although fewer migraine patients smoke and more give up smoking than in the general population, smoking could not be implicated in the majority of patients presenting with acute headaches at the migraine clinic. Furthermore, approximately 50% of the forty-four subjects, who said that smoking was involved in exacerbating their headache, had never actively smoked and therefore must have been referring to passive smoking. By using extreme conditions, it has been shown that it is possible to increase the COHb level of passive smokers by only 1% over a period of 78 min (Russell, Cole and

TABLE 2. The relationship of the claimed effects of smoking or smoke on headaches to the type of headache in 100 consecutive new patients attending a migraine clinic

Diagnosis	Sex		Effect of smoking or smoke on headaches	
			Causes or increases headaches	No effect on headaches
Migraine	M 13	79	2 } 26	11 } 53
	F 66		24 } 24	42 } 42
Tension headache	M 7	17	3 } 6	4 } 11
	F 10		3 } 3	7 } 7
Migrainous neuralgia	M 4	4	1 } 1	3 } 3
	F 0		0 } 0	0 } 0
Totals	M 24	100	6 } 33	18 } 67
	F 76		27 } 27	49 } 49

Brown, 1973). It is, therefore, most unlikely that a constituent of cigarette smoking was involved in the headaches of the patients who had never smoked. From this study it is concluded that the theory of an association between smoking and migraine has not been proved and that no evidence was found to suggest that active or passive smoking is a factor in the aetiology of migraine.

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