Increased gastric secretory capacity in smokers without gastrointestinal lesions

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SUMMARY The relationship between smoking and gastric secretory capacity was studied in 201 25–40 year old healthy subjects with normal laboratory data and no gastrointestinal lesions. Basal acid output (BAO) and peak acid output (PAO) were determined in all, and basal and stimulated pepsin outputs were measured in 85 participants. The accuracy of the patients' statements was checked by urinary nicotine assay. Basal acid output and PAO were significantly higher in male smokers ($n=55$) than in male non-smokers ($n=49$). In women PAO in smokers ($n=38$) was higher than in non-smokers ($n=59$). Female smokers ($n=38$) had a higher pepsin output than female non-smokers ($n=23$). Eight variables were considered in relation to BAO and PAO: age, height, weight, alcohol abuse, smoking habits, duration of smoking habit, number of cigarettes per day, and the product of years of smoking multiplied by daily number of cigarettes. The daily number of cigarettes $\times$ years of smoking was most closely correlated with BAO and PAO by stepwise multiple linear regression analysis. It was concluded that smoking is related to increased gastric acid capacity.

The influence of acute nicotine abuse on gastric acid secretion has been investigated by many authors. The results of these studies are highly controversial; whereas some have reported an increase in gastric secretion under basal conditions^{1–3} or under H₂-receptor antagonists,⁴ or a decrease after stimulation by pentagastrin,⁵ others have found no significant changes.^{6–10} In a previous study, we found that duodenal ulcer patients who smoked had a higher peak acid output than did non-smokers.¹¹

The present study is designed to find out whether there is a relationship between smoking habits and gastric acid secretion in healthy subjects without peptic ulcer disease.

Methods

PATIENTS

All patients arbitrarily assigned to one of us (SM) by the reception of our outpatient department for general clinical investigation were asked to volunteer for gastric analysis, provided they met the

following criteria: (1) Age 25–40 years, (2) good general health as judged by overall physical appearance and clinical findings, (3) lack of a history or early signs of peptic ulcer disease, (4) in case of symptoms or minor complaints of abdominal discomfort, failure of abdominal ultrasound and endoscopy of the upper gastrointestinal tract to reveal evidence of peptic ulcer disease, bulbar deformity, duodenitis, gastric erosions, or gall bladder disease, (5) agreement by the patient to participate in our investigation after being thoroughly informed.

Shortly before gastric analysis all participants were questioned as to how long they had been smoking, how many cigarettes daily, and the amount of alcohol consumption. On the day of gastric intubation, 20–30 ml of urine was obtained for nicotine assay by thinlayer chromatography.¹² The patients did not know why urine was being examined, nor was the technician aware of smoking habits. The interval between the most recent cigarette and gastric intubation was not taken into consideration. Fasting blood was drawn for routine laboratory tests (erythrocyte sedimentation rate, haemoglobin, red and white blood counts, creatinine, glutamic-oxalacetate and glutamic-pyruvic transaminases, gamma glutamyl transpeptidase, cholesterol, and triglycerides).

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Gastric analysis was done on fasting subjects under basal conditions for 60 minutes, and after stimulation with pentagastrin (6 µg/kg) for an additional 60 minutes. Basal acid output (BAO) and peak acid output (PAO) were determined. Pepsin concentration in the gastric juices was measured by the method of Rick *et al.*¹³ using swine pepsin from Merck (Darmstadt, West Germany) as a standard. Pepsin output in gastric juices was established 60 minutes before and 60 minutes after pentagastrin stimulation.

The sensitivity of parietal cells was assessed by the acid secretion response to graded doses of pentagastrin (25–6000 ng/kg/h). Ten male smoker volunteers (mean age 27, SD three years) and 10 male non-smokers (mean age 29, SD two years) were studied. The former had smoked 21 (SD nine) cigarettes daily for seven (SD nine) years. Each dose of pentagastrin was infused over 30 minutes. Gastric juices were aspirated by a vacuum pump at low pressure (Gastrovac, Hirtz Co., Cologne, West Germany). Maximal acid response to pentagastrin and the pentagastrin dose at which a half-maximal response (D_{50}) was elicited in each subject were calculated according to Isenberg *et al.*¹⁴ Results are expressed as means $\pm$ 1 SD, or as median values and 20% and 80% percentiles in case of skewed distribution. Significance of differences was analysed by Student's *t* test or the Mann-Whitney U-test, depending on the distribution of data.

In an attempt to evaluate factors affecting gastric acid secretion, the following eight variables were analysed by stepwise forward multiple linear regression¹⁵: age, height, weight, alcohol (in g/day), smoking habits, duration of smoking habit, number of cigarettes smoked daily, and product of duration of smoking habit in years $\times$ number of cigarettes per day.

Results

Two hundred and forty patients fulfilled the above criteria for inclusion in the study; 69 of them underwent abdominal sonography and endoscopy with negative findings. Thirty nine subjects (23 men and 16 women) had to be dropped from the study later, in 23 instances because abnormal laboratory tests were suggestive of chronic disease (raised transaminases in 10, gamma-GT in four, creatinine and urea in two, increased erythrocyte sedimentation rate in seven cases), whereas laboratory data on 12 persons and gastric analysis in four persons was incomplete. The remaining 201 individuals included 104 men (55 smokers and 49 non-smokers) and 97 women (38 smokers and 59 non-smokers). The 21 participants (13 men and eight women) who had given up smoking at least one year before the study were classified as non-smokers.

Daily alcohol consumption was found in 15 of 103 men; 85 drank occasionally, and four were abstainers. The number of women who drank (three) was too small. Age, weight, height, daily alcohol consumption, blood pressure, daily number of cigarettes, and duration of smoking habit are given for both sexes in Table 1. Except that the mean age of female non-smokers exceeded that of female smokers by three years, no differences were found between smokers and non-smokers of either sex.

The presenting clinical complaints were referable to conditions commonly prevailing in the working population: no appreciable disease in 41, hyperlipoproteinemia in 18, hypertension in 22, hypotension in six, functional cardiac disease in 16, and functional abdominal disorders in 48. The clinical diagnoses in smokers and non-smokers of both sexes are listed in Table 2.

Table 1 Characteristics of subjects

	Men		Women	
	Smokers (n=55)	Non-smokers (n=49)	Smokers (n=38)	Non-smokers (n=59)
Age (years)	29.9 $\pm$ 4.7	31.8 $\pm$ 5.5	29.9 $\pm$ 4.5	32.9 $\pm$ 5.5*
Weight (kg)	76 $\pm$ 10	74 $\pm$ 10	62 $\pm$ 12	63 $\pm$ 11
Height (cm)	177 $\pm$ 7	176 $\pm$ 6	165 $\pm$ 6	166 $\pm$ 5
Number of cig/day	20 $\pm$ 7	—	15 $\pm$ 6	—
Duration of smoking habit (years)	11 $\pm$ 5	—	9 $\pm$ 4	—
Alcohol intake (g/day, median and range)	5 (0–100)	2 (0–75)	2 (0–125)	—
Blood pressure (mmHg) systolic	134 $\pm$ 15	138 $\pm$ 14	134 $\pm$ 17	133 $\pm$ 16
and diastolic	86 $\pm$ 10	89 $\pm$ 10	88 $\pm$ 11	86 $\pm$ 10
Nicotine in urine (n)	49	1	27	1

Mean $\pm$ 1 SD, **p*<0.01

Table 2 Main clinical diagnosis of subjects (numbers)

	Men		Women	
	Smokers (n=55)	Non-smokers (n=49)	Smokers (n=38)	Non-smokers (n=59)
Healthy	15	11	6	9
Metabolic disease	6	5	2	5
Hypertension	8	7	2	5
Hypotension	1	2	2	1
Functional cardiac disease	5	4	4	3
Functional abdominal disease	12	8	12	16
Arthralgia and myalgia	2	3	3	6
Struma nodosa	1	2	3	5
Cold infection	2	4	2	3
Sarcoidosis	2	0	1	0
Ophthalmic disease	0	1	—	—
Skin disease	1	2	—	—
Myoma uteri	—	—	0	2
Haemorrhoids	—	—	1	1
Asymptomatic pyelitis	—	—	0	2
Proteinuria	—	—	0	1

Male smokers had higher triglycerides (146 SD 102 mg/dl) than male non-smokers (110 SD 62 mg/dl, $p<0.05$). Mean values of other laboratory parameters were similar to those in a healthy population, and exhibited no differences between smokers and non-smokers of either sex.

Data on HCl secretion and pepsin output are given in Table 3. Figure 1 depicts the individual values of acid secretion. Although a large overlap exists between the smoking and non-smoking groups of both sexes (similar to the situation in patients with and without duodenal ulcers), HCl secretion under basal and stimulated conditions was significantly higher in male smokers than in male non-

smokers. The same was true of women after pentagastrin stimulation. Again like patients with duodenal ulcers, some subjects in both groups were hypersecretors. In 80 duodenal ulcer patients from our department (mean age 47 SD 14 years, 63 men, 17 women), the mean values of PAO in smokers was 40 SD 12.9 mmol of HCl/h, and 32.7 SD 15.1 mmol HCl/h in non-smokers ($p<0.05$).¹¹

Pepsin concentration under basal conditions was seen to be significantly increased in smokers of both sexes. In female smokers it was also enhanced after pentagastrin stimulation.

Of the eight variables, the smoking habit itself and the product of daily number of cigarettes $\times$

Table 3 HCl- and pepsin-secretion in men and women in relation to smoking and non-smoking (median, 20% and 80% percentiles; Mann-Whitney U-test between smokers and non-smokers for men and women).

	Men						Women					
	Smokers (n=55)			Non-smokers (n=49)			Smokers (n=38)			Non-smokers (n=59)		
	Percentile			Percentile			Percentile			Percentile		
	Median	20%	80%	Median	20%	80%	Median	20%	80%	Median	20%	80%
BAO (mmol HCl/h)	2.16 [†]	1.24	4.41	1.47	0.830	2.54	1.68	0.73	2.47	1.14	0.54	2.05
PAO (mmol HCl/h)	36.0 [†]	28.2	44.8	27.9	21.6	36.0	25.7 [†]	19.2	31.8	19.7	14.4	24.8
BAO/weight ($\frac{\text{mmol HCl}}{\text{h kg}}$)	0.028 [†]	0.018	0.055	0.020	0.012	0.036	0.028	0.011	0.042	0.017	0.009	0.035
PAO/weight ($\frac{\text{mmol HCl}}{\text{h kg}}$)	0.486 [†]	0.365	0.602	0.377	0.311	0.456	0.388 [†]	0.292	0.540	0.321	0.215	0.408
Pepsin output basal (mg/h)	62.2 [*]	44.7	95.0	40.4	24.0	62.0	47.5 [†]	31.2	55.4	32.3	6.6	49.2
	(n=21)			(n=11)			(n=23)			(n=30)		
Pepsin output maximal (mg/h)	122.4	78.3	161.8	120.6	79.5	145.0	86.9 [†]	71.3	137.2	66.1	32.1	96.8
	(n=21)			(n=11)			(n=23)			(n=30)		
Pepsin output basal/weight ($\frac{\text{mg}}{\text{h kg}}$)	0.771 [*]	0.629	1.205	0.596	0.185	0.758	0.742 [†]	0.478	1.075	0.472	0.099	0.761
	(n=21)			(n=11)			(n=23)			(n=30)		
Pepsin output maximal/weight ($\frac{\text{mg}}{\text{h kg}}$)	1.749	1.058	2.101	1.827	1.16	2.06	1.449 [*]	1.116	2.205	1.098	0.472	0.1587
	(n=21)			(n=11)			(n=23)			(n=30)		

* $p<0.05$, [†] $p<0.02$.

Fig. 1 Individual levels of BAO and PAO segregated according to sex and smoking habit.

Fig. 2 Mean gastric acid output and standard deviations (bars) in response to intravenous infusion of graded doses of pentagastrin in healthy male smokers and non-smokers.

duration of smoking habit correlated best with BAO and PAO (Table 4). Except for BAO values in women, nearly all correlations between BAO and PAO and smoking were significant ($p < 0.05$), although the absolute values of the simple correlation coefficients were not high (0.26–0.43). To assess whether the predictability of BAO and PAO levels improves when all eight variables are used, stepwise multiple linear regression analysis was carried out. We terminated the procedure after three steps, because the increment of squared multiple correlation was less than 5%. The correlations increased slightly (BAO from 0.31 to 0.43 and PAO from 0.43 to 0.54 in men, and PAO from 0.26 to 0.32 in women). There was no significant increase for BAO in the female group (Table 4). This means that the smoking habit is a better predictor of BAO and PAO augmentation in the male group and of PAO changes in the female group than, say, weight. Still, the absolute values of the multiple correlations

Table 4 Evaluation of the importance of variables contributing to BAO and PAO by stepwise multiple linear regression analysis

		Simple correlation	Multiple correlation	Squared multiple correlation	Increment	Standardised regression coefficient
Men (n=104)	BOA					
	Cigarettes/day × duration of smoking habit in years	0.31 [§]	0.31 [§]	0.096	0.096	0.32
	Weight	0.22 [†]	0.41 [§]	0.168	0.072	0.21
	Height	0.24 [†]	0.43 [§]	0.185	0.017	0.14
	PAO					
	Smoking	0.43 [§]	0.43 [§]	0.185	0.185	0.35
	Weight	0.31 [§]	0.51 [§]	0.260	0.075	0.20
Women (n=97)	Alcohol (g/day)	0.18	0.54 [§]	0.292	0.032	0.14
	BAO					
	Smoking	0.12	0.12	0.014	0.014	0.30
	Duration of smoking habit	-0.02	0.19	0.036	0.022	-0.25
	Weight	-0.08	0.20	0.040	0.004	-0.07
	PAO					
	Cigarettes/day × duration of smoking habit	0.26 [†]	0.26 [†]	0.068	0.068	0.55
	Age	-0.19	0.32 [†]	0.102	0.034	-0.17
	Height	0.06	0.32 [†]	0.102	0.000	0.07

†p<0.05, §p<0.01, §p<0.001.

are not high enough to allow predictions in individual cases.

Discussion

Maximum acid output is a function of parietal cell mass and related to body weight, lean body mass, and sex.¹⁶ Recent studies have shown that ethnic background plays a role in the amount of parietal cell mass.^{17, 18} Inheritance may be important in hyposecretion, as indicated by studies on families with hyperpepsinogenemic I duodenal ulcers.¹⁹ The significance of environmental factors is poorly understood. Although many constituents of foods and drinks are known to induce gastric acid secretion,^{20, 21} it is unclear whether such stimulants exert potential long term effects on parietal cell mass.

The acute effects of smoking on acid secretion have been extensively studied.¹⁻⁷ The results are still controversial, although more reports favour induction of acid secretion by nicotine. The effects of chronic smoking have been studied on patients with duodenal ulcers^{11, 22} and on healthy medical students.²³ Whitfield *et al*²² concluded that smoking in duodenal ulcer patients may be the major cause of their tendency toward hypersecretion of HCl. In another study on medical students, Novis *et al*²³ found smoking to be significantly correlated to BAO, but not to PAO; as their population had a

mean age of 20 years, the smoking habit could not have been of long duration.

Multiple regression analysis of the data in the present study indicates that smoking itself, and especially the product of daily number of cigarettes × duration of the habit is significantly involved in enhancement of gastric acid secretion.

Pyloric loss of acid secretion was disregarded in our study, because the quantitative effect of this error upon gastric acid secretion is minimal in patients with high rates of secretion.²⁴ Furthermore, the large number of individuals in each group tends to compensate for any error caused by incompleteness of volume recovery or the amount of bile reflux. Gastric secretory data were not corrected for body build; because weight and height were comparable in smokers and nonsmokers of both sexes, it would have no effect on the inferences drawn from our data.

The mean age of our subjects was 30 years, and the mean duration of their smoking habit 10 years. It seems reasonable to assume that advancing age and continuation of smoking habits are correlated to increased gastric acid secretion. The association between chronic smoking and gastrointestinal function is probably not limited to the augmentation of secretion of HCl and pepsin; smoking might be expected to modify the contents of gastric juice and pepsin isoenzyme patterns.²⁵ Apart from this, it has acute effects on the competence of the pyloric canal

in man,²⁶ as well as on pancreas secretion, particularly bicarbonate output for alkalinisation of the duodenal bulb in man^{27,28} and dogs.²⁹ Thus the smoking associated increase of gastric acid secretion might well be just one of many various effects of the habit on the upper gastrointestinal tract that are likely to be causally related to multifactorial peptic ulcer disease.

Increased sensitivity of duodenal ulcer patients to stimulation by pentagastrin has been reported,¹⁴ but subsequent controlled studies have failed to confirm this impression.^{30,31} Aly *et al*³¹ have suggested that the increased sensitivity of oxyntic cells to pentagastrin is related less to ulcer disease than to gastric acid capacity; subjects with high secretion of gastric acid are more sensitive to pentagastrin than those with low secretion.

We found no difference between smokers and non-smokers in parietal cell sensitivity; basal and maximally stimulated gastric acid secretion were similar in both groups.

Our results point out the relationship of chronic smoking and the gastric secretory capacity in healthy individuals. They indicate that smoking is more closely correlated to BAO and PAO than are weight and height.

From the epidemiological standpoint,³² postulation of a causal relationship between smoking and hypersecretion demands consistency, strength, specificity, and coherence of the association, as well as precedent long term smoking. Based on many reports from various countries, doubt hardly remains as to the consistency of the association. The strength of the association is indicated by our results. Nevertheless, the other requirements of epidemiologists for confirmation that smoking actually induces hypersecretion are not fulfilled. In agreement with our clinical study,³³ Sontag *et al* have recently shown that smoking appears to be a major factor in recurrence of duodenal ulcer; the course of duodenal ulcer disease in non-smokers on placebos resembles that of smokers taking acid reducing cimetidine.³⁴ Thus, the association between the strength of the smoking habit and degree of hypersecretion suggests the probable importance of smoking in the aetiology of the latter, which is related to peptic ulcer disease.

Further studies are needed to verify these conclusions, as well as to find out more about how long standing smoking influences parietal cell mass, and the reversibility of deleterious changes after long term abstinence from smoking.

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Correction

We regret an error occurred in Figure 1 of the paper by Massarraf, Enschai and Pittner entitled 'Increased gastric secretory capacity in smokers without gastrointestinal lesions' in the April 1986 issue p. 433, and the correct version is printed below: