Risk Factors for Continued Cigarette Use After Subarachnoid Hemorrhage
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Background and Purpose—Cigarette smoking is a risk factor for the formation and rupture of intracranial aneurysms. Few studies have examined predictors of resumption of cigarette smoking after a first episode of subarachnoid hemorrhage (SAH).

Methods—Of 620 SAH patients treated between July 1996 and November 2002, we prospectively evaluated continued cigarette use in 152 smokers alive at 3 months. Univariate and multivariate logistic regression analyses were used to identify potential demographic, social, and clinical predictors of continued cigarette use, defined as smoking $\geq 1$ cigarette per week in the month before follow-up.

Results—Thirty-seven percent (56 of 152) resumed smoking after their SAH. Patients who continued smoking were younger, were more often black, had begun smoking at an earlier age, and had a higher frequency of prior alcohol or cocaine use and self-reported depression or anxiety than those who quit (all $P<0.05$). Smoking at $\leq 16$ years of age (odds ratio [OR], 5.88; 95% confidence interval [CI], 2.33 to 14.29), self-reported depression (OR, 5.29; 95% CI, 2.10 to 13.35), and prior alcohol use (OR, 4.51; 95% CI, 1.45 to 14.05) independently predicted continued cigarette use. Smokers had a functional outcome similar to that of nonsmokers at 3 months but were more likely to resume alcohol consumption (OR, 3.88; 95% CI, 1.91 to 7.88).

Conclusions—More than one third of prior smokers continue to use nicotine after SAH. Young age at smoking onset and a history of depression or alcohol use are risk factors for continued cigarette use. Targeted smoking cessation programs are needed to reduce the high rate of smoking resumption after SAH. (Stroke. 2003;34:1859-1863.)

Key Words: alcohol drinking $\bullet$ cerebral aneurysm $\bullet$ cigarette smoking $\bullet$ subarachnoid hemorrhage

Subarachnoid hemorrhage (SAH) affects $\approx 21,000$ adults in North America each year.1 Even with significant improvements in the medical and surgical management of SAH, case fatality rates remain between 30% and 50%.2–4 and those who survive frequently report impaired quality of life (QOL) characterized by disruptive cognitive and emotional symptoms.3 A recurrent episode of SAH occurs in $\approx 2%$ to $3%$ of patients each year after surgical or endovascular aneurysm repair, and this risk increases with time.6–7

Cigarette smoking is an important modifiable risk factor for SAH.5–12 Tobacco use is also a risk factor for the formation of multiple13 and larger14 aneurysms and therefore may be a risk factor for recurrent SAH after aneurysm repair.8 Although it is known that up to 60% to 70% of SAH patients smoke cigarettes,12,13 few studies have examined how many of these patients resume smoking after their hemorrhage or why they return. One study found that the proportion of smokers decreased from 58% before SAH to 30% 4 to 7 years later.15 To the best of our knowledge, no studies have specifically analyzed risk factors for smoking resumption after SAH.

Identification of risk factors for continued cigarette use after SAH may allow more effective substance use interventions while patients are in the hospital and may ultimately decrease the risk of recurrent SAH and other cardiovascular events. For instance, multidisciplinary hospital-based intervention programs increase the frequency of smoking cessation after myocardial infarction,16,17 which results in reduced long-term mortality.18 In this study, we sought to determine the frequency of continued cigarette use 3 months after SAH and to identify risk factors for smoking resumption.

Patients and Methods

Patient Population and Baseline Assessment
Six hundred twenty-six SAH patients admitted consecutively to our Neurological Intensive Care Unit (NICU) between July 1996 and November 2002 were prospectively enrolled in the Columbia University SAH Outcomes Project. The study was approved by the hospital Institutional Review Board, and in all cases, written informed consent was obtained from the patient or a surrogate. The diagnosis of SAH was established by CT or by xanthochromia of the cerebrospinal fluid if the CT was negative. Exclusion criteria

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included SAH from trauma or rupture of an arteriovenous malformation, admission >14 days after onset, and age <18 years. Patients with spontaneous nonaneurysmal SAH were included in the study.

Demographic data (age, sex, race/ethnicity (nys), history and amount of substance use (cigarettes, alcohol, and cocaine), and social history (marital status, education level, employment history, living alone or with others, number of visitors to hospital during surgery, average number of phone calls to friends per week, importance of church/religion in daily life) were obtained from an interview with the patient and family shortly after admission. Psychological history was assessed by 2 methods. One method was a self-reported history of anxiety or depression as assessed by a checklist: (1) a “depressed mood most of the day, nearly every day, noticeable to either you or a significant other, for at least 6 months” and (2) “experienced excessive anxiety or worry, occurring more days than not, for at least 6 months, regarding more than one event or activity.” The second method was a self-reported history of medically treated depression or anxiety. Clinical status was evaluated with the admission Glasgow Coma Scale (GCS) score and worst Hunt-Hess score recorded during hospitalization.

For tobacco use, information was obtained from the patient or a surrogate regarding the age that the individual started smoking, age the person quit (if applicable), total number of years spent smoking, type of nicotine-containing products used, and amount of cigarettes consumed (<1 per month, 1 to 3 per month, 1 per week, 2 to 6 per week, 1 per day, 2 to 10 per day, 11 to 20 per day, ≥21 per day). Subjects were classified as baseline or resumed smokers if they had smoked at least weekly during the month before their hemorrhage or at the 3-month follow-up evaluation. All smokers were treated daily with a transdermal 21-mg nicotine patch during their hospitalization. Individuals were considered baseline or recurrent alcohol drinkers or cocaine users if they had consumed these substances at least once during the month before their evaluation; they were considered heavy drinkers if they consumed ≥2 alcoholic beverages per day during the previous month (1 drink = 30 mL of 80-proof liquor, 360 mL of beer, or 240 mL of wine). Operational definitions to quantify alcohol and cocaine use were similar to those used for cigarette use.

Three-Month Follow-Up Assessment

Three months after the onset of SAH, each subject and a relative or spouse were asked to complete a 45-minute telephone or in-person interview. All assessment instruments were administered in the participant’s native language (English or Spanish). Tobacco, alcohol, and cocaine use was again assessed on the basis of a checklist: (1) a “depressed mood most of the day, nearly every day, noticeable to either you or a significant other, for at least 6 months” and (2) “experienced excessive anxiety or worry, occurring more days than not, for at least 6 months, regarding more than one event or activity.”

Admission and discharge variables that demonstrated significant univariate associations (P < 0.05) were entered into a multiple logistic regression model using stepwise forward selection to identify baseline variables independently associated with resumed cigarette use. Clinical outcome scales (mRS, Lawton IADL scale, STAS, CES-D, TICS, SIP) were dichotomized by use of established cut points for poor outcome, with the exception of the SIP, for which poor QOL was defined as a score ≥2 SD above the published mean normative value.

Results

Of 619 enrolled patients, 525 (85%) volunteered a baseline smoking history, and of these, 245 (47%) reported smoking in the month before their SAH. Compared with those who reported on baseline cigarette use, those who did not were more often comatose (GCS score ≤8) on admission (48% [42 of 86] versus 18% [94 of 525], P < 0.0005); no other significant differences were found. Thirty-seven (15%) baseline smokers reported smoking 0 to 3 cigarettes daily; 55 (22%) smoked 4 to 10 cigarettes daily; 93 (38%) smoked 11 to 20 cigarettes daily; and 66 (24%) smoked ≥21 cigarettes daily. Of the 245 baseline smokers, 31 (13%) were dead, 6 (2%) were lost to follow-up, and 208 (85%) were alive and evaluated at 3 months. One hundred fifty-two of these patients (73%) responded to the follow-up assessment of substance use habits and were included in the present analysis. These patients did not differ significantly from the 56 who did not report substance use at follow-up with regard to age, sex, race/ethnicity, education, worst Hunt-Hess grade, and 14-day mRS scores.

The 152 individuals evaluated for resumption of smoking ranged in age from 23 to 80 years. Fifty-six of these patients (37%) had resumed smoking 3 months after their SAH, and 96 (63%) had quit. Patients who had resumed smoking (Table 1) more often were <50 years of age; had a higher rate of prior cocaine or alcohol use; had a higher frequency of self-reported depression, anxiety, or treatment for depression; and were more often of black, non-Hispanic ethnicity than those who had quit. Patients who continued to use nicotine were also significantly younger when they started smoking than those who quit. The median age at which the entire sample began their use of cigarettes was 17 years: 51% of those who began smoking at ≤16 years of age were smokers; 28% of those who began smoking at ≥17 years of age (P < 0.0005).

A multivariate logistic regression analysis of baseline predictors yielded smoking at ≤16 years of age (OR, 5.88; 95% CI, 2.33 to 14.29; P = 0.0002), history of self-reported depression (OR, 5.29; 95% CI, 2.10 to 13.35; P = 0.0004), and alcohol use before SAH (OR, 4.51; 95% CI, 1.45 to 14.05; P = 0.009) as independent predictors of continued smoking at 3 months.

At 3 months, patients who resumed smoking engaged in alcohol consumption more frequently than those who did not (Table 2). However, there was no difference in global functional outcome, cognitive status, or emotional health between smokers and nonsmokers at 3 months, although smokers were somewhat more likely to have returned to work.
Of 242 baseline alcohol consumers who responded to the substance use history, 114 (47%) had resumed alcohol consumption in the month before their 3-month evaluation. Of the 57 patients who consumed ≥2 alcoholic beverages per day before their hemorrhage, 6 had continued to consume alcohol at this level at 3 months, 35 had either stopped or reduced their drinking, and 6 were dead; this information was not available in 10. Of the 38 patients who reported using cocaine in the month before their hemorrhage, 5 continued to use it, 13 had quit, and 7 were dead; this information was not available in 13.

**Discussion**

In this study, the frequency of returning to smoking after SAH was 37%, and smoking resumption was independently predicted by an earlier age of initial cigarette use, prior alcohol use, and a history of self-reported depression. This frequency is lower than the one-half to two-thirds frequency of recurrent smoking that occurs after acute myocardial infarction, which may reflect the more dramatic nature of SAH and its treatment, the impact of neurological impairment, or the fact that all our patients were treated with nicotine patches while in the hospital and in many cases after discharge.

Smoking is widely recognized as an important risk factor for SAH. A meta-analysis of 10 epidemiological studies estimated a relative risk for first-ever SAH of 2.9 for a current smoker. Although no study has estimated the risk of recurrent SAH if individuals continue to smoke, several lines of evidence suggest that continued smoking may increase this risk. Smoking accelerates carotid artery atherosclerosis and may promote atherosclerosis, shear stress, and intimal degeneration in the intracranial cerebral vasculature. Smoking-induced states of acute hypertension, as well as increased vessel fragility resulting from degradation of elastin in blood vessel walls, may also contribute to the pathogenesis of cerebral aneurysms. Smoking may promote aneurysm growth by triggering reductions in α1-antitrypsin activity, an elastase inhibitor that prevents breakdown of elastin and collagen in the cerebral blood vessel walls. Finally, smoking has also been associated with symptomatic vasospasm after SAH and with the presence of multiple aneurysms. Although heavy alcohol consumption (≥150 g or 12 beverages per week) and cocaine ingestion may also increase the risk of recurrent SAH, we did not analyze these lifestyle risk factors in detail because of the small number of 3-month survivors who continued their use.

A novel finding of our study is the identification of younger age at smoking onset as a risk factor for continued smoking after SAH. This finding indicates that the addictive properties of nicotine may be especially potent in individuals who begin smoking at a younger age, an observation consistent with the well-publicized attempts of tobacco manufacturers to market cigarettes to children and teenagers in recent years. Routine inclusion of the age of smoking onset in the social history may provide a simple and effective means of identifying SAH patients at high risk for continued smoking. Careful screening for a history of prior depression or alcohol use may also identify patients at increased risk for smoking resumption. Informing patients (and families) that they are at high risk to continue smoking after hospital discharge may be an important first step in raising awareness of the problem and may provide extra motivation for some patients to quit.

The ethnic diversity of our study sample allowed us to identify race/ethnic populations that might be at high risk for continued smoking. Black patients were more likely to return to smoking than white or Hispanic patients, although the actual number of these patients in our study was small (Table 1). Patients who resumed smoking after SAH were also more likely to be ≤50 years of age and more often reported a history of cocaine use, anxious mood, or treatment for depression than those who quit. Although these variables did not add further predictive value in our multivariate analysis, they suggest that polysubstance abuse or a history of psychi-
Atrophic disorders may increase the risk for continued smoking after SAH.

Functional outcome, QOL, cognitive status, and emotional health were similar in those who resumed smoking compared with those who quit (Table 2). Our findings do not support the notion that patients who resume smoking after SAH do so as a means to “self-treat” poststroke depression or to enhance alertness or cognitive function.38–40

Our study has several limitations, the most important of which are the relatively small sample size and the 3-month follow-up interval. With a larger study population, we may have had improved power to detect additional risk factors for recurrent smoking. It is possible that the proportion of SAH patients who resume smoking increases progressively during the first year after hospitalization. Our assessment of cigarette, alcohol, and cocaine use by interview likely underestimated the amount of these substances that our subjects actually consumed, and the accuracy of surrogate information versus self-reported rates of substance use and age of smoking onset has not been established. Other more accurate methods of assessment may be necessary to further examine the frequency of these behaviors among survivors. Finally, we did not obtain a premorbid smoking history in 14% of our study sample; this subset of patients was overrepresented by poor-grade patients for whom a surrogate was unable to provide an accurate history. This may explain in part the lower frequency of prior smoking in our SAH population (47%) compared with prior reports (60% to 70%).12,13

In summary, 37% of patients who smoked before their SAH had resumed smoking 3 months after their hemorrhage. As a first step to help patients quit smoking, we routinely treat all smokers with a transdermal nicotine patch in the hospital and after discharge, which may provide the added benefit of preventing delirium secondary to nicotine withdrawal during the acute phase of the illness.41,42 Simply informing early-onset smokers (≥16 years of age) that they are at increased risk for continued cigarette use may help raise awareness of this issue and encourage family support. There is a need to develop improved psychosocial and medical treatment interventions targeted at smoking cessation after SAH, which should be routinely offered as part of the rehabilitation and ongoing care of these patients.

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References

| TABLE 2. Smoking Resumption and Clinical Outcomes at 3 Months |
|-----------------|-------|-----------------|-----------------|-----------------|
| Those With Outcome, % | OR    | 95% CI           | P               |
| Alcohol use       | 34    | 3.88            | 1.91–7.88       | 0.000           |
| Employed          | 21    | 2.77            | 1.25–6.16       | 0.012           |
| No disability or handicap (mRS, 0 or 1) | 37    | 1.91            | 0.97–3.76       | NS              |
| Independent IADLs (Lawton Scale, 8)* | 41    | 0.69            | 0.36–1.35       | NS              |
| Good QOL (SIP total score <15.5)† | 48    | 0.87            | 0.45–1.70       | NS              |
| No depression (CES-D <16)‡ | 50    | 0.79            | 0.39–1.61       | NS              |
| No anxiety (STAS state subscale <43)§ | 70    | 0.57            | 0.26–1.25       | NS              |
| No cognitive impairment (TICS >30)¶ | 72    | 1.19            | 0.54–2.60       | NS              |

*ORs refer to the risk of smoking resumption in patients with the given clinical outcome vs subjects without the outcome.
†Scored 8 = best, 30 = worst.
‡Scored 0 = best, 100 = worst.
§Scored 0 = best, 60 = worst.
¶Scored 20 = best, 80 = worst.
††Scored 51 = best, 0 = worst.


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