to the physician's reminders that, because they smoke, they risk heart disease, cancer, and chronic obstructive lung disease. Patients who thoroughly understand the health hazards and who perceive themselves at higher risk (for example, those who have a family history of cancer, heart disease, or lung disease) are among those more likely to quit.

Repeated warnings about impaired quality of life add to the impact. For example, smoking increases the likelihood of shortness of breath, prematurely wrinkled skin, upper respiratory infections and lost work days, impaired sexual performance, frostbite, and automobile accidents. The danger of house fires should not be overlooked. Finally, smoking lowers one's self esteem because the individual is controlled by the addiction.

HOW TO BE EFFECTIVE

For a smoking cessation program to be effective, the physician needs to be familiar with the physiologic process of quitting smoking, and with quit strategies that work.

Virtually every smoker who quits will experience withdrawal symptoms. The notable exceptions are some who quit following bypass surgery or a myocardial infarction. Withdrawal is brief. Symptoms generally peak 2 to 3 days after quitting and are gone 10 to 14 days later. After that, cravings for cigarettes are related to previous linkages, or associations, with situations, emotions, and people, but not to a craving for nicotine. These cravings tend to be intense but brief, rarely lasting for more than 30 seconds.

Withdrawal symptoms vary from patient to patient, and may include headaches, lightheadedness, changes in bowel habits, nausea, diarrhea, chest pain, cough, sore throat (often, a smoker is unaware of a chronic sore throat because of the anesthetizing effects of smoking), increased irritability, decreased frustration tolerance, anxiety, difficulty concentrating, emotional lability, increased appetite, and sleep disturbances. Acute depression is also possible and seems to be relatively more common among women than men.

The value of group support cannot be overestimated. Many people need it, along with physician support and encouragement, to achieve success. The average success rate of stop-smoking programs ranges from 20% to 30%. The best ones offer patients not only company, but also an opportunity to learn coping skills, relaxation and other techniques they will need to achieve smoking cessation. Hypnosis may help, but it is no panacea.

One helpful strategy is to give the patient a stopsmoking checklist in which "assignments" are separated into manageable units of time—preparing to quit, the first 2 weeks, after 2 weeks, and after 2 months. For example, when preparing to quit, the patient would be assigned to pick a quit date and mark it on the calendar, and would change to a less desirable brand of cigarettes.

Persistence and encouragement from the physician are essential to success. Smoking is a powerful addiction. If the patient "slips," it is best not to be judgmental or pessimistic, but instead to encourage the individual to learn from the experience, pick a new quit date, and get it right the next time.

GARLAND Y. DeNELSKY, PHD Head, Section of Psychology Director, Smoking Cessation Program The Cleveland Clinic Foundation

BIBLIOGRAPHY

Cummings SR, Rubin SM, Oster G. The cost effectiveness of counseling smokers to quit. JAMA 1989; **261:**75–79.

Hughes JR, Kottke TE. Doctors helping smokers: real-world tactics. Minnesoate Medicine 1986; 69:293–295.

Kottke TE, Battista RN, DeFriese GH, Brelle ML. Attributes of successful cessation interventions in medical practice: A meta-analysis of 39 controlled trials. JAMA 1988; **259:**2883–2889.

US Department of Health and Human Services. The Health Consequences of Smoking: Nicotine Addiction. 1988, DHSS Pub. No. 88–8406.

MESENTERIC ISCHEMIA: A NEGLECTED DIAGNOSIS

As the population ages, we can anticipate an increased number of patients with hemodynamically significant atherosclerotic occlusive disease. Our understanding of the features of visceral ischemia syndromes, the liberal early use of diagnostic arteriography, and prompt therapeutic intervention will improve both survival and quality of life.

Early diagnosis is not the general rule in mesenteric ischemia, partly because accurate, noninvasive diagnostic measures have not been applied to the mesenteric circulation. Mesenteric arteriography is the most accurate, and essentially the only useful, study for the diagnosis of mesenteric ischemia; but, because it is invasive, it is often obtained only after a period of observation.

CHRONIC MESENTERIC ISCHEMIA

Chronic mesenteric ischemia is nearly always caused by atherosclerosis involving the proximal visceral arteries. It is difficult to correlate the degree of atherosclerosis on arteriography with the presence of symptoms. Usually all three vessels must be involved to produce symptoms.

The predominant symptom is reproducible, crampy, postprandial abdominal pain-often so intense that "food fear" develops. The patient avoids eating in order to prevent discomfort. Some type of gastrointestinal motility disturbance is present in 50% to 85% of patients, and most have significant weight loss.

Physical signs may include cachexia, an abdominal bruit, and, in about one third of patients, clinical evidence of atherosclerosis elsewhere. The patient may possess several cardiovascular risk factors. Lateral aortography confirms the diagnosis.

Surgical intervention (transaortic endarterectomy or arterial bypass) is indicated by the severity of symptoms and appropriate arteriographic findings. In an asymptomatic patient, surgical treatment is indicated if arteriographic findings are appropriate and the patient is scheduled to undergo aortic surgery or another procedure that would jeopardize intestinal blood flow.

ACUTE MESENTERIC ISCHEMIA

Acute ischemia is caused by sudden termination (occlusive) or marked reduction (nonocclusive) of flow in the superior mesenteric artery. The natural course is progression to full thickness necrosis of the midgut with mortality in the range of 50% to 70%. If the patient survives, the extensive bowel necrosis results in loss of most of the absorptive surfaces of the intestine. Acute occlusion of the superior mesenteric artery is caused by embolization from a remote, usually cardiac, source, or by spontaneous primary thrombosis of an atherosclerotic vessel.

Patients at risk of acute disease are elderly and have valvular or atherosclerotic heart disease, longstanding congestive heart failure, cardiac arrhythmias, recent myocardial infarction, a history of other embolic phenomena, or are hypotensive or hypovolemic.

Nonocclusive mesenteric ischemia, also called vasoconstrictive or low-flow ischemia, is caused by markedly diminished cardiac output. Patients are often in intensive care units because of hemodynamic instability.

The sine qua non of the presentation is the sudden onset of severe, unrelenting, abdominal pain with few physical findings. Some 15% to 25% of patients, especially those with nonocclusive ischemia, experience no pain but have other signs—notably unexplained abdominal distention or gastrointestinal bleeding.

Treatment, including resuscitation, and diagnostic maneuvers such as aortography must start simultaneously,

without delay. Local infusion of the vasodilator papaverine is the primary treatment of nonocclusive ischemia, along with correction of the cause of the low flow state.

Surgical intervention for occlusive acute mesenteric ischemia involves embolectomy or thrombectomy with arterial endarterectomy or bypass. Without arterial reconstruction, early postoperative rethrombosis is destined to occur in the atherosclerotic vessel.

Bowel viability must be assessed during surgery with a Doppler probe evaluation or fluorescein infusion, and a "second-look" laparotomy is indicated early in the postoperative period if there is any question about viability.

With vigorous treatment, acute occlusive mesenteric ischemia has a survival rate of approximately 50%, depending on the cause and the extent of coexisting illness.

ISCHEMIC COLON DISEASE

Ischemic colon disease, which may be the most common vascular disorder of the intestines, has protean manifestations. Tissue damage ranges from simple mucosal edema to infarction and necrosis.

The disease is either iatrogenic or spontaneous. Iatrogenically provoked colon ischemia results most often from ligation of the inferior mesenteric artery during aortic replacement for aneurysmal or occlusive atherosclerotic disease. It usually progresses to frank gangrene. Iatrogenic disease is avoidable by documentation of colonic (sigmoid) viability at the initial laparotomy.

Most spontaneous episodes of colonic ischemia produce transient, reversible changes. The etiology is uncertain, but is thought to involve low colonic blood flow. The diagnosis is made by conventional modalities, including endoscopy and contrast radiography. Arteriography is recommended when the ischemia involves the right colon, because this is thought to predict hemodynamically significant superior mesenteric artery disease.

MARK E. SESTO, MD Department of General and Vascular Surgery Cleveland Clinic Florida

BIBLIOGRAPHY

Boley SJ, Brandt LJ, Veith FJ. Ischemic disorders of the intestines. Curr Probl Surg 1978; 15.

Parks DA, Jacobson ED. Physiology of the splanchnic circulation. Arch Intern Med 1985; **145:**1278–1281.

Stoney RJ, Reilly LM. Chronic visceral ischemia. An overlooked cause of abdominal pain. Postgrad Med 1983; 74:111–118.