

# Guidance Offered on Cocaine-Related Chest Pain

BY MARY ANN MOON  
Contributing Writer

Patients who present with cocaine-associated chest pain or myocardial infarction should be treated similarly to patients with traditional acute coronary syndromes, with some exceptions, according to Dr. Jim McCord and his associates on the American Heart Association's acute cardiac care committee.

The committee reviewed the literature and issued a scientific statement in the March 17 online version of *Circulation*, with the aim of guiding diagnostic and therapeutic interventions for this subset of patients with acute chest pain.

Chest pain related to cocaine use is rarely caused by myocardial infarction, which develops in only 2%-6% of cases.

Therefore, risk stratification using well-established criteria, including electrocardiographic changes and a positive cardiac

troponin test, "is feasible and safe." But cocaine use can cause rhabdomyolysis and attendant abnormalities in creatine kinase and myoglobin levels, so those diagnostic tests may not be useful, said Dr. McCord of Henry Ford Hospital, Detroit, and his associates.

"In the absence of ischemic ECG changes or positive cardiac markers, intermediate- and low-risk patients can be safely managed in a chest pain observation unit for 9-12 hours, which can obviate the need for hospital admission in the majority of these patients," they added.

Unlike patients with acute coronary syndrome unrelated to cocaine use, those with cocaine-associated chest pain should receive benzodiazepines immediately to relieve the pain, improve hemodynamics, and manage any neuropsychiatric manifestations that may accompany cocaine toxicity.

Also in contrast to ACS patients, those with cocaine-associated

chest pain who do not have definite cardiac involvement may not need direct treatment of any hypertension and tachycardia they exhibit. Resolution of their anxiety with a benzodiazepine will often resolve the hypertension and tachycardia as well. If it doesn't, these can be managed with sodium nitroprusside, nitroglycerin, or intravenous phenolamine, the committee statement said.

For the few patients whose cocaine-associated chest pain is caused by ST-segment elevation myocardial infarction, percutaneous coronary intervention clearly is preferable to fibrinolysis.

There are few data regarding the use of drug-eluting stents in this patient population. Because most of these patients continue to use cocaine, they may have poor compliance with any long-term antiplatelet regimens. "Therefore, we recommend very careful consideration of the probability of long-term compliance before a drug-eluting stent

is used in cocaine-associated MI," the committee said.

$\beta$ -Blockers should not be given in STEMI precipitated by cocaine use, because it may exacerbate coronary spasm. Whether calcium channel blockers are beneficial in this patient population is uncertain at best. Therefore, they should not be given as

first-line treatment but can be considered for patients who don't respond to benzodiazepines and nitroglycerin, Dr. McCord and his associates said (doi: 10.1161/circulationaha.107.188950).

Antiplatelet and antithrombin therapies haven't been well studied for cocaine-related chest pain, but they may be beneficial, because cocaine injures the vascular epithelium, raises platelet aggregation, and impairs normal fibrinolysis. "We recommend aspirin be routinely administered and unfractionated heparin or



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low-molecular-weight heparin be given... unless there is a contraindication," they said.

Finally, recidivism is common, with 60% of patients in one study admitting to continued cocaine use 1 year after being hospitalized for chest pain.

"Preliminary data suggest that a combination of intensive group and individual drug counseling has the greatest impact on recurrent cocaine use," so these approaches should be recommended to these patients, the AHA committee said. ■

## AHA Calls for Glucose Control in ACS Patients With Hyperglycemia

BY ROBERT FINN  
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Hyperglycemia is common in acute coronary syndrome and is a strong predictor of poor outcome, but many questions remain about how to take these facts into account in clinical practice, according to a scientific statement from the American Heart Association.

Although it's still uncertain whether treating hyperglycemia in acute coronary syndrome (ACS) produces definite benefits, it's reasonable to consider intensive glucose control in patients with plasma glucose levels above 180 mg/dL, and even for some patients with milder degrees of hyperglycemia, according to the members of the writing group, led by Dr. Prakash Deedwania of the University of California, San Francisco (*Circulation* 2008 Feb. 25 [doi:10.1161/circulationaha.107.188629]).

"Most cardiologists are not aware of the importance of hyperglycemia in the acute coronary syndrome," Dr. Deedwania said in an interview. "Although some specialty centers are taking care of hyperglycemia, the majority of them are not paying any attention."

More than 2 million patients are treated in the United States annually for ACS, and as many as 50% of them might have hyperglycemia, Dr. Deedwania said. Numerous analyses and meta-analyses have found increased risks linked to hyperglycemia in ACS. The largest retrospective study, which involved 141,680 patients, found that hyperglycemia increased the risk of 30-day mortality by 13%-77%, and it increased 1-year mortality by 7%-46%, depending on the degree of hyperglycemia.

The risks appear to be greatest in hyperglycemic patients with no previous evidence of diabetes, but it's still unclear whether hyperglycemia is a marker or a mediator of adverse outcomes.

The most pressing unanswered question, according to Dr. Deedwania, is to determine which treatment for hyperglycemia has the best combination of efficacy and safety. One large recent trial showed that hypoglycemia can be more dangerous than hyperglycemia, so it's important to figure out how critical it is to control hyperglycemia and to what extent it should be controlled.

Other areas in need of further investigation include whether persistent hyperglycemia during ACS hospitalization has a greater impact on prognosis than does admission hyperglycemia alone, whether there is a critical period of vulnerability from hyperglycemia in these patients, whether the best target glucose levels differ in patients with and without pre-existing diabetes, and what the optimal timing of therapy might be.

Meanwhile, the writing group determined that there is now excellent (level A) evidence to recommend that glucose levels should be part of the initial laboratory evaluation in all patients with suspected or confirmed ACS. And there is good (level B) evidence that glucose levels should be monitored closely in patients admitted to an ICU with ACS, that it's reasonable to consider treatment in patients with high levels of hyperglycemia, that insulin by intravenous infusion is the most effective measure to control glucose in ICU patients, and that special attention should be paid to ACS patients with hyperglycemia but no history of diabetes.

In addition to informing physicians about the importance of hyperglycemia in ACS, the release of the AHA scientific statement has another goal, Dr. Deedwania said. "This is a call to action for all the different agencies such as the National Institutes of Health to consider doing trials on some of these very specific questions. This should be a priority." ■

## Angina and Dementia Are Linked in Study of Twins

BY SHERRY BOSCHERT  
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SAN FRANCISCO — Angina pectoris was associated with an approximately 50% increased risk for Alzheimer's disease or dementia within 15 years in a longitudinal study of 20,146 Swedish twins.

Ulrika K. Eriksson and her associates analyzed data from the Swedish Twin Registry and found a 56% increased risk for hospitalization or death resulting from Alzheimer's disease in people with a history of angina pectoris, compared with those without angina pectoris, they reported in a poster presentation at the annual meeting of the Gerontological Society of America. The risk for hospitalization or death from dementia was 53% higher in those with angina pectoris, compared with the controls, said Ms. Eriksson, a doctoral student in medical epidemiology and biostatistics at Karolinska Institutet, Stockholm.

The increased risk was more prominent in women. Women with angina pectoris had a significant 64% increased risk for Alzheimer's disease, compared with women without angina pectoris, but an increased risk for dementia was not significant. Increased risks for Alzheimer's disease and dementia in men with

angina pectoris also were not statistically significant.

In previous studies, some risk factors for coronary artery disease have been associated with increased risk for future dementia, she noted.

The Swedish Twin Registry is an ongoing database of twins born in 1903, 1925, and 1936. Questionnaires mailed to the twins in 1963, 1967, and 1973 identified 8% as having a history of angina. The investigators linked two population-based registries to identify hospitalizations or deaths primarily caused by dementia from 1974 to 2001. In all, 5% of the cohort was diagnosed with dementia, 68% of which was Alzheimer's disease.

The increased risk for Alzheimer's disease or dementia in people with angina was significant in the first 15 years (1974-1989) but not for the entire 28-year follow-up period. The increased risk in women with angina also occurred in the first 15 years of follow-up only. These differences over time may be a result of survival bias, the investigators said.

The results were adjusted for age, smoking, education, and BMI. By matching twin pairs in co-twin controls, the investigators adjusted for early life factors and genetic confounding. ■