THE IHS PRIMARY CARE PROVIDER

A journal for health professionals working with American Indians and Alaska Natives

April 2002 **Volume 27, Number 4**

Management of ST-Segment Elevation Myocardial Infarction: Thrombolytic Guidelines

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The fundamental pathophysiology of acute myocardial infarction involves the rupture of lipid-laden plaque in a coronary artery, creating a highly thrombogenic milieu. The formation of clot leads to activation of fibrin, resulting in expansion of the clot and complete occlusion of the coronary artery. The administration of fibrinolytic agents to recanalize occluded coronary arteries was a breakthrough in the treatment of acute myocardial infarction (AMI).

Authors Comment

As the incidence and prevalence of cardiovascular disease (CVD) increases among Native Americans, we in Indian health must continuously strive to further support and improve the prevention efforts related to CVD and its risk factors, with a primary focus on diabetes mellitus, as well as efforts to reduce and eliminate hypertension, dyslipidemia, smoking, obesity, and physical inactivity among those we serve. In addition, for those of us providing direct care to our Native American patients, our responsibilities also include our resolve to provide the optimal care to the patient with CVD, including the aggressive acute management of myocardial infarction.

We, at the Native American Cardiology Program, hope that we may be able to assist by offering a succinct review and basic guidelines for the treatment of acute myocardial infarctions, which could be used or modified to provide the optimal treatment plan at your facility. This is the second of a number of articles and reviews we at the Native American Cardiology Program, as your partners in Indian health, will offer for your review. We hope you will find this series helpful to you and the patients we mutually serve.

James M. Galloway, MD, FACP, FACC

Thrombolysis unequivocally reduces mortality in patients who present with symptoms of AMI and ST-segment elevation or left bundle branch block on electrocardiogram (ECG).[1] Patients with ST-segment elevation MI treated with thrombolytics had a 25% relative reduction in mortality, with a 2% absolute reduction in mortality; this translates into 26 lives saved per 1000 people treated.[2]

Higher risk patients benefit more from reperfusion therapy, including those with anterior wall ST elevation or left bundle branch block; they have greater mortality benefits with lytics than do those with uncomplicated inferior myocardial infarctions. Patients over age 75 achieve greater absolute mortality reduction than younger patients but have higher complication rates. Patients with ST depression or without ST elevation do not benefit from thrombolytic therapy.

Thrombolytic therapy salvages myocardium; the earlier blood flow to the heart muscle is restored, the smaller the infarct size and the better the prognosis.[3,4,5] Nonetheless,

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thrombolytic therapy administered up to 12 hours after symptom onset still achieves significant reductions in mortality. Patients with pain for greater than 24 hours generally do not benefit from thrombolytic therapy.

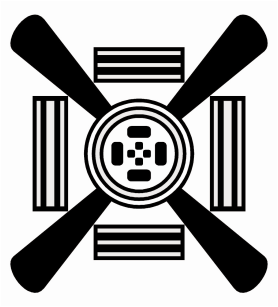
An important confounder in the evaluation of patients with possible AMI is the presence of symptoms for longer than 6 to 12 hours. Many of these patients have a stuttering course and days of unstable angina that culminates in AMI. It is important to determine whether there was a time at which their symptoms intensified or became continuous, resulting in their decision to seek medical care. This would then be the moment at which their acute MI symptoms began.

The national standard calls for a door-to-EKG time of 10 minutes and a door-to-needle time of 30 minutes for the delivery of thrombolytic therapy to patients with AMI. To achieve these goals, the emergency room staff must have a high level of vigilance for high-risk patients and for those with symptoms suggestive of an acute myocardial infarction. A classic presentation may be that of a patient with substernal chest pressure radiating to the neck, jaw, or back with associated dyspnea, nausea, or diaphoresis. Patients may describe their chest symptom as a heaviness, squeezing, or pressure rather than a pain.

It should be remembered that certain patient populations, namely diabetics, women, and the elderly, might have more atypical presentations: their primary complaint may be dyspnea, abdominal pain, fatigue, or back pain, although most will also have a component of chest pain or discomfort. Cultural and language differences may make assessment of the symptoms more difficult; for those for whom English is not their primary language, the use of a translator, if available, will produce a more useful history. Clearly, people communicate better in their primary language under stressful circumstances.

A rapid triage and EKG, focused exam, bloodwork, and the placement of two large bore intravenous (IV) access sites must be done quickly. Aspirin, 162 to 325 mg, should be given to the patient to chew. If the initial ECG is unremarkable but the patient continues to have signs or symptoms suggestive of AMI, the ECG should be repeated. If the patient is not hypotensive, sublingual nitroglycerin should be given in an effort to relieve the pain. Caution should be used in patients who have taken sildenafil (Viagra) in the preceding 24 hours, given the significant risk of hypotension caused by concurrent nitrate administration.

The presence of l mm of ST-segment elevation in two contiguous leads should result in a rapid review of exclusion criteria to determine if there are any contraindications to thrombolytics. If there are no contraindications, thrombolytic therapy should be given immediately with care to administer the proper dose for the chosen agent over the appropriate infusion time. The patient should be monitored with continuous telemetry and blood pressure checks to watch for reperfusion arrhythmias, resolution of ST elevation, or hypotension. After administration of the thrombolytic agent, the patient should be transferred to an intensive care unit or tertiary care center for further monitoring for arrhythmias or complications associated with myocardial infarction or thrombolytic therapy. If interventional cardiology services are readily available (door-to-angioplasty time of 90 minutes or less), this may be an approach preferable to thrombolytic therapy.



Thrombolytic Agents

Fibrinolytic agents convert plasminogen to plasmin that then degrades fibrin, a central structure of acute thrombus, with the goal of opening the infarct-related artery. Nonspecific fibrinolytic agents, streptokinase being the prototype,[6] activate both circulating and fibrin-bound plasminogen, creating a systemic depletion of fibrinogen, plasminogen, and factors V and VIII. Second generation fibrinolytics are recombinant tissue-type plasminogen activators, tPA being the prototype. They are more fibrin-specific, create less systemic depletion of fibrinogen and may lyse more highly cross-linked fibrin.[7] This has been borne out by the findings that patients who present later after symptom onset are more likely to achieve optimal reperfusion with the newer, more fibrin-specific agents such as tPA and TNK-tPA than with streptokinase.

Third generation thrombolytic agents are even more tissue-specific and can be delivered in bolus form; TNK-tPA is the prototype of this group. These newer, more fibrin-specific agents result in less systemic coagulopathy and a more focused action at the site of thrombotic occlusion. They also achieve higher arterial patency rates and improved blood flow, which correlates with survival.[8,9] When various regimens are compared, accelerated tPA with heparin showed a 40% improvement in survival over standard regimens. Recent data on TNK-tPA found it to be equivalent in effect to tPA.[10]

A major breakthrough in the fibrinolytic field has been the development of bolus agents with equivalent efficacy and cost to front-loaded tPA. Ease of administration with equal effect make them particularly appealing. Dosing and efficacy trials highlight the importance of appropriate dosing and administra-

tion: there was a significant decrease in the risk of intracranial bleeding when the total tPA dose was decreased from 150 mg to 100 mg over a three-hour infusion; front-loaded tPA resulted in better patency rates than standard infusion tPA without higher intracerebral bleed rates.

Evaluation of larger thrombolytic trials reveals significant dosing errors in the administration of both streptokinase and tPA ranging from 11.5% to 13.5%. These errors resulted in significantly higher mortality rates when compared with those who received the properly administered medication, making cost and outcome analysis more complicated. Bolus therapy markedly decreases the likelihood of dosing or administration errors, thereby improving the actual efficacy of the lytic agent and reducing mortality by eliminating complications from misadministration.[11,12,13]

The determination that rapidity of administration of lytic agents improves outcomes makes ease of administration a central issue. In facilities where the presentation of acute myocardial infarction is relatively infrequent, the ability of the staff to rapidly reconstitute and deliver lytic agents at a specific, weight-adjusted dose over a very specific infusion time is a major challenge. Given a 13% rate of errors in thrombolytic administration in hospitals with adequate volume, staff, and training to run clinical trials, facilities with lower frequencies of thrombolytic administration and less staffing could expect similar if not significantly higher error rates, making bolus therapy an important consideration for both staff and patients.

Adjunctive Therapies

Aspirin has clearly been shown to reduce mortality in acute myocardial infarction and other acute coronary syndromes both with and without the administration of thrombolytic agents. Any patient without an aspirin allergy, presenting with symptoms suggestive of an acute coronary syndrome should immediately receive 162 to 325 mg of aspirin to be chewed. In the event of an aspirin allergy, clopidogrel at a dose of 75 mg may be substituted.

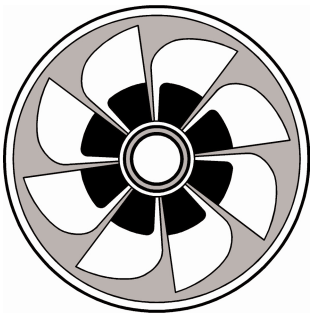
Beta blocker therapy should be administered to patients with acute myocardial infarction in the absence of contraindications such as active bronchospasm, heart block, significant bradycardia (a rate of less than 60 bpm), hypotension, or congestive heart failure. The initial dosing should be metoprolol, 2.5 to 5 mg intravenously over 2 minutes every 5-10 minutes as tolerated, stopping for bradycardia, hypotension, or other signs of intolerance to the therapy. Beta blocker therapy reduces the risk of arrhythmias in the face of ischemia, as well as decreasing the ischemic burden by lowering heart rate, blood pressure, and contractility.

Nitrates are also helpful in patients with ongoing ischemia, although their benefit is less well proven. Sublingual nitroglycerin should be administered to patients when first presenting with chest pain, if it is in the absence of hypotension or other contraindications. Nitrates work by dilating the coronary arteries, thereby improving myocardial perfusion; they also decrease blood pressure, thereby decreasing the work of the myocardium.

If the patient continues to have chest pain, intravenous nitroglycerin at a starting dose of 5 mcg/min should be initiated in an effort to relieve the pain. Care should be taken not to induce hypotension, since this decreases coronary and cerebral perfusion. In patients with inferior myocardial infarctions, who may have concurrent right ventricular infarction and hence be very volume sensitive, nitroglycerin may induce a profound and rapid decrease in blood pressure. These patients respond to fluid resuscitation but may require liters of fluid. Nitroglycerin should be stopped if hypotension is not easily and rapidly resolved. If blood pressure is borderline and a single adjunctive therapeutic agent must be chosen, beta blocker therapy is of more importance than ongoing nitrate therapy.

Pain increases sympathetic drive that works the heart harder and increases the risk of arrhythmias. Morphine sulfate may be given in order to provide pain relief to the patient having coronary ischemia or an acute myocardial infarction. The main goal should be coronary reperfusion and prompt thrombolytic administration. Intermittent doses of morphine (1-4 mg intravenous boluses every 20 minutes) may be given for comfort if the patient is not hypotensive. Since thrombolytic administration may engender hypotension, care should be given to avoid simultaneous administration of multiple agents that might lower the blood pressure. Morphine does not generate any mortality benefit and is the least important medication in the armamentarium of therapies for acute myocardial infarction.

Angiotensin converting enzyme inhibitors (ACEI) have been shown to decrease dilation of the left ventricle early after myocardial infarction. They have also been shown to improve survival after myocardial infarction, especially in patients with depressed left ventricular function. Intravenous administration of these agents should be avoided. Oral administration of



ACEI may be appropriate in patients who are not hypotensive after administration of beta blocker therapy, nitrates, and thrombolytics. Unless the patient has been on an ACEI as an outpatient, a shorter acting agent should generally be used, since hypotension may result.

Caution should be taken not to administer too many agents that may cause hypotension simultaneously. Hypertension may increase ischemia, infarct size, and complication rates. For patients with uncontrolled hypertension and very high pressures (>180/110 mm Hg), target goals should be modest so as not to cause stroke. These patients are not candidates for thrombolytics. For patients with moderate hypertension, the goal should be a normal blood pressure of 100- 130/60-80 mm Hg. Patients not on dialysis, with serum creatinine levels above 2.0 mg/dl should not receive ACEI acutely unless they have been on them chronically with a stable creatinine.

Hypokalemia potentiates arrhythmias in ischemic myocardium. Numerous studies have shown that maintaining serum potassium levels above 4.0 mg/dl decreases the risk of arrhythmias such as ventricular tachycardia. Hypomagnesemia also increases arrhythmic potential but, more importantly, prevents the correction of hypokalemia since magnesium is exchanged for potassium in the kidneys. Data on magnesium replacement in acute myocardial infarctions has not shown any clear benefit; however, replacement for patients with low magnesium levels (<2.0 mg/dl) is appropriate in order to prevent hypokalemia and arrhythmias.

Heparin therapy is an essential adjunct to tPA and TNK-tPA. The fibrin-specific character of these agents results in a targeted effect and a short half-life, with the potential for reocclusion of the infarct artery. The addition of intravenous heparin to these agents prevents that reocclusion and partly accounts for the higher patency rates and improved outcomes in patients treated with the newer generation fibrinolytics. Higher doses of intravenous heparin have resulted in higher bleeding complications, and a strict weight-adjusted protocol is recommended to decrease complications.[14]

The recommended heparin protocol is 60 units/kg as a bolus, with a maximum of 4000 units, followed by an infusion of 12 units/kg/hour, at a maximum rate of 1000 units/hour.[15] Recent smaller studies have found weight-adjusted, subcutaneous low molecular weight heparin to be equivalent to[16] or better.[17,18,19] than unfractionated intravenous heparin; this may be due in part to a very predictable, lasting anticoagulation effect. The benefit of using subcutaneous low molecular weight heparin is ease of administration, less potential for dosing errors, and lower bleeding complications. Renal failure impairs the clearance of low molecular weight heparin and clear recommendations on appropriate dosing in this situation are not yet available.

Streptokinase, on the other hand, results in systemic fibrinogen depletion and a hypocoagulable state that can last for up to 24 hours. The addition of intravenous heparin to streptokinase has been shown to increase bleeding complications and it is only recommended in high risk circumstances. Intravenous heparin may be given with streptokinase cautiously in patients with atrial fibrillation, large anterior wall myocardial infarction, an embolic event, or left ventricular thrombus, and then only when a PTT is documented to be less than 70 seconds, and no sooner than four hours after streptokinase administration. Subcutaneous, unfractionated heparin may be given at a dose of 12,500 units every 12 hours for 48 hours in other circumstances, but is not requisite.

Complications

The most dreaded complication of fibrinolytic therapy is intracerebral bleeding. Serious bleeding occurs in a dose-response fashion, which has led to the adjustment of dosing recommendations to minimize bleeding while maximizing reperfusion benefits. Most intracerebral bleeds occur within the first 24 hours of thrombolytic administration. Age greater than 65 years old, weight less than 70 kg, and uncontrolled hypertension on presentation increase the risk of bleeding. One of the goals of developing newer thrombolytic agents has been to decrease the risk of CNS complications. Unfortunately this has not come to pass, and the more fibrin-specific agents actually have higher intracerebral hemorrhage rates than strep-

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— *The Editors*

tokinase. Of those treated with tPA or TNK-tPA, 1.9% suffer intracerebral hemorrhage; streptokinase without concurrent heparin therapy has the lowest intracerebral hemorrhage rate, especially in the elderly.

Hypotension during infusion of the fibrinolytic agent is possible and occurs most commonly with streptokinase. Patients must be monitored closely, and other agents that may induce hypotension should be stopped first if this occurs. Many patients will respond to fluid administration or initiation of dopamine; the thrombolytic should be continued, if at all possible. For refractory hypotension the infusion may need to be stopped or slowed. Staff should be vigilant for signs of acute blood loss. Persistent hypotension should evoke consideration of potential complications of acute myocardial infarction, such as right ventricular infarction, heart block, ischemic mitral regurgitation, pericardial tamponade, pump failure, or myocardial rupture. Auscultation for new murmurs or rales, evaluation of telemetry rhythm, and scrutiny of right-sided leads in patients with inferior MI may help elucidate the etiology of the hypotension.

Reperfusion arrhythmias are common and usually do not cause hemodynamic compromise. The most common is accelerated idioventricular rhythm (AIVR), which is a wide-complex ventricular rhythm at a rate of 110 bpm or slower. Less frequently, ventricular tachycardia occurs and may cause hemodynamic compromise or degenerate into ventricular fibrillation. Close telemetry monitoring of the patient during administration of the fibrinolytic agent enables documentation of reperfusion arrhythmias and provides resources should the patient develop a hemodynamically significant rhythm disturbance.

Hypokalemia on initial laboratory studies should be treated expeditiously to decrease the risk of serious dysrhythmias. Beta-blocker therapy also decreases the risk of ventricular ectopy and tachycardia, and should be given whenever possible, as discussed above.

Conclusion

Acute myocardial infarction remains a major cause of mortality in the US. Fibrinolytic therapy was a tremendous advance in the therapy of myocardial infarctions and has led to significant reductions in mortality. Rapid administration and accurate dosing are essential to achieve optimal benefit at the lowest risk possible. Newer agents available in bolus form provide easy administration with optimal effect and less risk of dosing errors. Close telemetry and blood pressure monitoring are essential to assure safety. Aspirin, electrolyte replacement, beta-blocker therapy, and pain control with nitrates and morphine are all important adjuncts to reperfusion therapy. Administration of heparin, when indicated, is essential for optimizing and maintaining arterial patency. The goal of therapy is to reduce mortality and salvage myocardium through reperfusion. Time is myocardium and myocardial salvage is survival.

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THROMBOLYTIC GUIDELINES

Inclusion Criteria:

- Chest pain of duration greater than 30 minutes and less than 12 hours with EKG findings of 1 mm ST-segment elevation in two or more contiguous leads, or LBBB. If the patient has had pain for longer than 12 hours, the presence of a stuttering quality or a time at which the pain suddenly intensified and became unrelenting, if less than 12 hours from presentation, would prompt consideration for the administration of thrombolytic agents.
- Chest pain and ECG changes persisting after sublingual nitroglycerine.

Exclusion Criteria:

- Absolute Contraindications:
  1. Active internal bleeding (does not include menses).
  2. Suspected aortic dissection.
  3. History of hemorrhagic cerebrovascular accident at any time.
  4. History of nonhemorrhagic cerebrovascular accident within one year.
  5. Known intracranial neoplasm.

- Relative Contraindications:
  1. Severe hypertension on presentation (BP > 180/110).
  2. History of prior cerebrovascular accident, known intracranial aneurysm, or AVM.
  3. Current use of anticoagulants with INR > 2 - 3 or PT > 15.
  4. Known bleeding diathesis.
  5. Recent trauma (< 4 weeks).
  6. Traumatic or prolonged (>10 minutes) CPR.
  7. Major surgery within 3 weeks.
  8. Puncture of a noncompressible vessel.
  9. Recent internal bleeding (within 4 weeks).
  10. Prior exposure to streptokinase or APSAC (this contraindication is particularly important in the initial two year period after streptokinase or APSAC administration and applies to reuse of any streptokinase-containing agent, but does not apply to tPA).
  11. Previous allergic reaction to streptokinase.
  12. Pregnancy.
  13. Active peptic ulcer.
  14. History of chronic severe hypertension (BP > 160 systolic, > 100 diastolic).

Preparations:

1. All patients should have aspirin administered: 162 mg to 325 mg orally/chewed, unless contraindicated.
2. At least two, preferably three, large-bore IV lines should be placed.
3. Stool guaiac should be done to rule out heme-positive stool.
4. Blood work should be drawn before lytic administration, including type and hold.
5. Unnecessary invasive procedures should be deferred given added risk of bleeding.
6. Electrolytes should be replaced with a goal to keep potassium level above 4.0 mg/dl and magnesium level above 2.0 mg/dl. This should not delay lytic administration.
7. Beta-blocker therapy should be given to all patients unless they are in congestive heart failure, have active history of recent bronchospasm, are bradycardic to a heart rate of 60 bpm or less, have greater than first degree heart block, or are hypotensive with a systolic blood pressure below 100 mm Hg. Ideally, administer meto

prolo1 2.5 - 5 mg IV over 2 to 5 minutes and repeat, if necessary, every 10-15 minutes for up to 15 mg; if the patient falls below the above parameters at any point, the remaining doses are held.

8. Continuous EKG monitoring and BP monitoring is required to watch for arrhythmias and hypotension. Oxygen therapy should be administered if indicated for hypoxia or cyanosis.

TNK-tPA (Tenectaplase)

Patient Weight*(kg)	Patient Weight* (lbs)	TNKase (mg)	Reconstituted TNK (mL)
<60	<132	30	6
>60 to <70	>132 to <154	35	7
>70 to <80	>154 to <176	40	8
>80 to <90	>176 to <198	45	9
>90	>198	50	10
*Dosing based on actual or estimated patient weight.			

Dextrose-containing solutions cause precipitation, and all lines should be flushed with saline prior to administration if dextrose solutions have been administered.

Adjunctive therapy: Heparin, unfractionated, 60u/kg bolus, maximum 4000u, followed by 12u/kg/hr IV infusion, at a maximum rate of 1,000u/hr, following PTT levels every 6 hours to target range of 55-70 sec. Low molecular weight heparin, enoxaparin, 30 mg IV then 1 mg/kg SQ every 12 hours by a strict dosing schedule may be used instead of IV heparin. Heparin should be given before or at the time of TNK-tPA bolus.

tPA (Alteplase)

15 mg. bolus followed immediately by IV infusion of 0.75mg/kg (up to 50 mg) over 30 minutes followed immediately by 0.50 mg/kg (up to 35 mg) over the next 60 minutes (total infusion time 90 minutes).

Adjunctive therapy: Heparin, unfractionated, 60u/kg bolus, maximum 4000u followed by 12u/kg/hr IV infusion, at a maximum rate of 1,000u/hr, following PTT levels every 6 hours to target range of 55-70 sec. Low molecular weight heparin, enoxaparin, 30 mg IV then 1 mg/kg SQ every 12 hours by a strict dosing schedule may be used instead of IV heparin. Heparin should be given before or at the time of tPA infusion.

Streptokinase

1.5 million units IV over 1 hour.

Adjunctive therapy: Only in high risk patients, after discussion with a cardiologist, with a large or anterior MI, atrial fibrillation, previous embolus, or known LV thrombus, give IV unfractionated heparin, 60u/kg bolus, maximum 4000u, followed by 12u/kg/hr IV infusion, at a maximum rate of 1,000 u/hr. Start no sooner than 6 hours after streptokinase administration and only once the PTT is less than 2 times control. Check PTT levels every 6 hours with a target range of 55-70 sec.

Routine heparin administration in patients receiving streptokinase is not recommended unless the above, high-risk factors are present.

The Native American National Cardiovascular Disease Prevention Program

An alarming rise in the rate of coronary artery disease has been noted among Native Americans throughout the past several decades. Indeed, cardiovascular disease has become the leading cause of death among American Indians. This increase appears to be related to a rise in many cardiovascular disease risk factors, including the current epidemic of diabetes, as well as high blood pressure, dyslipidemia, obesity/overweight, smoking, and high cholesterol levels among American Indians.

Now, a prevention program has been created to address these disturbing trends in cardiovascular disease in Native American communities. While the focus on cardiovascular disease prevention is vital to develop the critical effectiveness needed in the specialized primary, secondary, and tertiary prevention efforts for cardiovascular disease, it is also clear that effective partnerships and collaboration with other programs, agencies, and organizations with a focus on primary prevention are essential to maximize our mutual efforts. The leadership, wisdom, and guidance of each effort by the Indian communities involved, as well as those of tribal/urban leaders, are well-recognized and important components of success. The cardiovascular disease prevention project is currently collaborating with the National IHS Diabetes Program as well as components of the National Institutes of Health, with efforts underway to work as one team to improve the health of American Indian people. Efforts are underway to develop even stronger relationships with these groups, as well as to develop multiple other collaborations.

There are currently three initial approaches of the Native American CVD Prevention Program:

- **Development of a broad national leadership.** This focus is based on sharing information, presentations, and conferences at many different levels within the Indian health community. Multi-agency efforts and potential resource development for the prevention of CVD among American Indians include American Indian leaders, government agencies, foundations, and specialty organizations.
- **Primary prevention efforts.** Activities supporting and dovetailing with the current IHS diabetes and the NIH CVD initiatives are underway. A number of multi-agency initiatives are under development to further support these efforts, including the development of Native American-specific community education programs, literature, and courses. A number of widespread CVD education programs for Public Health Nurses and Community Health Representatives will be held with topics ranging from prevention to CVD assessment and follow up. Community-based community health fairs, with a focus on CVD and diabetes, are being developed.
- **Secondary and tertiary prevention efforts.** A series of no-cost, regional, one-day intensive seminars on the "Prevention, Diagnosis and Treatment of Acute Coronary Syndromes" is being offered for medical providers working in Indian health. These one-day conferences are designed for primary care, emergency room, and internal medicine physicians, physician assistants, advance practice nurses, registered nurses, and others interested in the recent advances in the diagnosis, treatment, and prevention of acute coronary syndromes.

Current dates scheduled for the National ACS Conferences (Save the dates!):

- Flagstaff, Arizona: Flagstaff Medical Center, May 17, 2002
- Rosebud, South Dakota: June 20, 2002
- Gallup, New Mexico: August 7, 2002 (limited to PHNs/CHRs at this time)
- Cherokee, North Carolina: August 20-21, 2002
- Bismarck, North Dakota: September 16, 2002

These are confirmed dates; please watch for future postings for additional dates.

Accuracy of Using RPMS Data for Assessing Dental Exams in Individuals with Diabetes

Karen Carver, PhD, Statistician; and Stanley P. Griffith, MD, Medical Informaticist, both from the IHS Information Technology Support Center, Albuquerque, New Mexico; and Pat Ramsey, RN, Data Quality Consultant, Young, Arizona

GPRA measures, stemming from the Government Performance and Results Act of 1993, are reports that are required of the Indian Health Service (IHS) to assure that our agency is appropriately using its budgeted funding to provide a high quality of care to American Indians and Alaska Natives. This article is another in a series[1-3] reporting results from the GPRA Pilot Study, a study designed to investigate whether or not data already contained in the Patient Care Component (PCC), the primary clinical component of the Resource and Patient Management System (RPMS), IHS's healthcare information system, can be used to perform GPRA measurements with acceptable accuracy.

These data either are already or could be exported to a national database from which the measurements could be derived, thus reducing reporting burdens on Areas and local programs. Previous articles looked at using these data to measure the prevalence of childhood obesity, Papanicolaou (Pap) smear rates, and blood pressure control in individuals with diabetes. This article reports the results of the analysis of using data already in a national database to assess dental exams in individuals with diabetes.

Methods

In this study, a simple random sample of approximately 200 women between the ages of 18 and 65 who were diabetic (we used these criteria so the sample could be used simultaneously for an analysis of other measures) were selected at each of four geographically diverse facilities using data from the Headquarters (HQ) ORYX system, a national IHS database for local facilities participating in the Indian Health Performance Evaluation System. HQ ORYX data are derived from the data routinely exported from the PCC to the national level.

We then gathered pertinent information from the HQ ORYX system (demographics, date of visit, clinic code, ICD-9 diagnosis code) on all visits for each of these individuals during specified 9-12 month study periods. Detailed listings of these visits and associated information were provided to the manual chart reviewer. The individuals' charts were pulled and manually reviewed to determine if the individual had had a dental exam during any visit during the study time period.

These determinations were then compared with determinations from HQ ORYX data. For HQ data, a dental exam was said to have occurred on that visit if it was to a dental clinic or if the ICD-9 diagnosis code was V72.2 (dental examination). In addition, each visit for each of the study individuals was reviewed to determine 1) if any visits were missing from either the written chart or HQ data; 2) if there was documentation of a visit in the written chart, but not in HQ data or vice versa; or 3) whether or not a dental exam had occurred based on either the written chart or HQ data, which we then termed "best available" data.

Results

The numbers and percentages of individuals who had had a dental exam during the specified time period at each of the four facilities according to the written chart, HQ, and "best available" data are shown in Table 1. Table 2 lists the numbers and percentages of visits missing in the written chart or in HQ data at

Table 1. Numbers of individuals with diabetes who had a dental exam within the specified study period

	# Individuals	HQ Data		Chart Data		Best Available Data	
		#	%	#	%	#	%
Facility A	238	70	29.4	70	29.4	70	29.4
Facility B	200	71	35.5	71	35.5	71	35.5
Facility C	198	56	28.3	56	28.3	56	28.3
Facility D	200	52	26.0	52	26.0	52	26.0
Overall	836	249	29.8	249	29.8	249	29.8

Table 2. Agreement in visit data between the written chart and HQ data

	Total Visits	Visits with Errors		Visits Missing from HQ		Visits Missing From Chart		HQ Missed Dental Exam		HQ and Chart Matched	
	#	#	%	#	%	#	%	#	%	#	%
Facility A	3,912	12	0.3	5	0.1	2	0.1	5	0.1	3,900	99.7
Facility B	2,508	17	0.7	3	0.1	3	0.1	11	0.4	2,491	99.3
Facility C	3,822	22	0.6	17	0.4	0	0.0	5	0.1	3,800	99.4
Facility D	4,411	5	0.1	4	0.1	0	0.0	1	0.0	4,406	99.9
Overall	14,653	56	0.4	29	0.2	5	0.0	22	0.2	14,597	99.6

each facility, the numbers of visits for which the written chart documented a dental exam but HQ data did not, and the total number of visits for which the data elements described above exactly matched for both the written chart and HQ data.

Conclusions

Our data show that there is remarkable agreement between HQ data derived from the PCC and the written chart data both on whether or not a dental exam had occurred within a specified period for each individual (Table 1) and whether or not a dental exam had occurred during a specific visit (Table 2). This agreement was consistent across all facilities.

We suspect this accuracy of both individual and visit level data is because the combination of clinic codes and this diagnosis is reliably entered within the PCC system. Additionally, individual level data were even more accurate than visit level data. This was despite the fact that this measure looks for an event that is more likely to occur once during a relatively short time period in contrast to other measures that are derivations from multiple values or episodes, especially those that tend to be consistent over a period of time (e.g., median blood pressure, obesity[2,3]). For this measure, omitted data or data errors may still not skew the final calculation. For example, if there were actually two dental exams rather than one for an individual during the specified period and one was "lost," the individual will still be counted as having had a dental exam.

Limitations to the conclusions of this study include that this study only provides some of the first formal and rigorously studied, empiric data we have on this specific question. In addition, results and conclusions are based on data from only four facilities. As we begin to use PCC data for these kinds of measures, we need to continue to evaluate more and different kinds of data and measure their accuracy, in an ongoing fashion, at multiple and even more varied facilities.

Acknowledgements

The authors would like to thank Danny Macias for his assistance in providing the HQ data.

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NCME #794

Childhood Obesity II: Prevention and Community Intervention *(60 minutes)* Preventing childhood obesity is

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THE PROVIDER is published monthly by the Indian Health Service Clinical Support Center (CSC). Telephone: (602) 364-7777; Fax: (602) 364-7788; e-mail: *the.provider@phx.ihs.gov*. Previous issues of THE PROVIDER (beginning with the February 1994 issue) can be found at the CSC home page, *www.csc.ihs.gov*.

Opinions expressed in articles are those of the authors and do not necessarily reflect those of the Indian Health Service or the Editors.

Circulation: THE PROVIDER (ISSN 1063-4398) is distributed to more than 6000 health care providers working in the IHS and tribal health programs, to medical and nursing schools throughout the country, and to health professionals working with or interested in American Indian and Alaska Native health care. If you would like to receiver a copy, send your name, address, professional title, and place of employment to the address listed below.

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