

David Evans, MD  
 January 28, 2008  
 EVMS  
 Journal Club

**P-In pt with cocaine induced chest pain**  
**I- does beta-blocker therapy**  
**C- opposed to conservative therapy**  
**O- lead to worse outcomes (increased MI, Death)**

Question:

In Pt with cocaine induced chest pain does the addition of beta-blocker therapy improve risk of MI and Death?

Clinical Scenario:

A 23y male comes to your ED complaining of chest pain, nausea, and jaw pain after snorting cocaine. Vitals are BP 152/110 HR 149 Spo2% 94% RA. Pt is diaphoretic and tachycardic on exam with nonreproducible CP. EKG shows sinus tach, CXR normal, troponin is negative. You wonder if the addition of Beta-blockers would help in this scenario as it would in a patient with CAD.

Search Strategies:

PubMed was searched using the words “beta-blockers in cocaine induced chest pain” AND English [language] AND Humans [terms]. 5 articles resulted from search of those five articles that were reviewed 5 relevant articles were identified.

Author	Patient Group	Study Type	Outcomes	Key Results	Study Weakness
Ramoska E, Sacchetti D 1985 USA	1 patient with hyperadrenergic state	Case report	Change in HR and BP after 1 mg IV propanolol followed by a further 1mg	Decrease in HR followed by exacerbation of hypertension	Case report
Lange RA. et al 1990 USA	Thirty clinically stable patient volunteers referred for catheterization for evaluation of chest pain  Patients were given intranasal cocaine 2mg/Kg followed by either an IV push of saline or 2mg Propanolol over 2 minutes.	Randomized, double-blind, placebo-controlled trial	Change in coronary sinus blood flow after cocaine	Decreased post cocaine by 10% (+/-7%)	Low numbers equate to low statistical significance
			Coronary sinus blood flow after cocaine then saline	No change	
			Coronary sinus blood flow following cocaine and then treated with propanolol	Decreased by 15% (+/- 18%)	7/30 had normal coronary arteries
			Change in coronary vascular resistance after initial cocaine	Increased by 22% (+/- 11%)	Investigation only involved gross coronary anatomy  No markers of

			Coronary vascular resistance after cocaine then saline	No change	myocardial ischemia
Boehrer 1993 USA	15 patients (7 men and 8 women) aged 40-79 years, non cocaine users undergoing catheterization for evaluation of chest pain who were administered 2mg/kg cocaine intranasal and 15 minutes later received intravenous labetalol(n=6) or saline (n=9)	Prospective clinical trial	Results on angiography	6 were angiographically normal and 9 had sclerotic disease (>70% narrowing)	Poor methodology
			Change in heart rate after cocaine	No change	Very small sample size
			Change in Mean arterial pressure after cocaine	Increase of around 8% in both groups	Significant number had coronary vascular disease and all were not related directly to cocaine use
			Change in coronary arterial area after cocaine	Around 6% reduction in Group 1 who got saline after compared to around 8% reduction in those who got labetalol	No markers of ischemia studied
			Change in Heart rate after saline or labetalol	No change	Method of reporting of data is poor and the percentages have been calculated from their tables by the reviewers The tables merely report gross change in parameters with deviations in values measured
			Change in mean arterial Pressure after saline/labetalol	Almost 3% reduction in saline and 6% in labetalol group	
			Change in coronary arterial area after saline/labetalol	No significant change	
Sand IC 1991 USA	7 patients with cocaine-associated cardiovascular complications	Observational prospective	Reduction in heart rate or conversion to sinus rhythm.	Decline in HR 23% No consistent haemodynamic benefit. Serious adverse effects in 3 patients.	Small study IV diazepam was given prior to esmolol in 5 patients, thus treatment protocol not standardized.
Dattilo et al. 2008 USA	363 Pt admitted to telemetry/ICU with positive UDS for cocaine. 60 pt received beta-blockers	Retrospective	Reduction in MI and in-hospital mortality	MI reduction from 26% to 6.1%. Mortality was reduced from 4.5% to 1.7% but was not	Retrospective study can rule out selection bias Cocaine can be

				significant	<p>found in the urine in up to 2 wks</p> <p>Not all pt were admitted for chest pain</p> <p>Not clear if the reviewers were blinded to the hypothesis</p>
--	--	--	--	-------------	--

**Clinical Bottom Line:**

Past research based almost solely on small case reports have shown that beta-blockers exacerbate vasoconstriction. In this months Annals Dattilo postulates that it is not vasoconstriction which is the problem, but rather a beta-regulated build up of calcium within the myocardial cell causing myonecrosis. In this first of its kind retrospective study he showed that risk of MI was reduced with the addition of bet-blockers. These results need to be held with caution and should be viewed as hypothesis generating for future prospective trials. At this time it would be ill advised to consider beta-blocker therapy in acute onset of cocaine induced chest pain.

**Bibliography:**

Rappolt RT, et al. Propranolol in Cocaine Toxicity. Lancet. 1976; 2:640-641  
 Lang RA et al., Potentiation of cocaine induced coronary vasoconstriction by beta-adrenergic blockade. Ann IM. 1990;112:897-903  
 Sand IC et al., Experince with esmolol for the treatment of cocaine associated cardiovascular complications. Am J Emerg Med. 1991;9:161-163  
 Dattilo PB et al., Beta-blockers are associated with reduced risk of myocardial infarction after cocaine use. Ann EM. Feb 2007  
 Page et al. Should beta-blockers be used in the treatment of cocaine-associated acute coronary syndrome. Ann Pharmacother. 2007 Dec;41(12):2008-13. Epub 2007 Oct 23  
 Bohrer JD et al. Influence of labetalol on cocaine-induced coronary vasoconstriction in humans. Am Jour Med. 1993 Jun;94(6):608-10