IMPROVED METHODS FOR MEASURING ADENOSINE IN HUMAN BLOOD: ISCHEMIA INDUCED ELEVATION OF CORONARY SINUS ADENOSINE.

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Previous workers have detected increases in coronary sinus Adenosine (Ado) in pts with ischemic heart disease, but only in those pretreated with the Ado uptake inhibitor dipyridamole. We have developed improved methods which have enabled us to detect >2-fold increases in the concentration of coronary sinus Ado in pts who have not been pretreated with dipyridamole. Since the $t_{1/2}$ of Ado in human blood is <1.5 s, it is necessary to rapidly arrest Ado metabolism. In order to prevent breakdown or production of Ado sampled from the coronary sinus of pts, we have developed a double lumen catheter which permits mixing of stop solution and blood at its tip. We have also developed an improved stop solution to more completely arrest Ado metabolism. To improve accuracy of analysis, we developed a method utilizing two precolumns to partially purify and concentrate Ado from blood prior to analytical HPLC. With this procedure, the peak corresponding to Ado in blood is well separated from interfering compounds and is completely removed by the addition of adenosine deaminase. Addition of known amounts of Ado to plasma resulted in a linear increase in measured Ado (r=.97). Venous blood drawn from volunteers into stop solution used by others (Heparin and dipyridamole) was 3.5-fold higher than in our new stop solution (.289±.087 vs .082±.016 μM, n=5, p<.01). The new stop solution was supplemented with the adenosine deaminase inhibitor EHNA, the 5'nucleotidase inhibitor AOPCP and EDTA. Using these improved procedures, coronary sinus Ado levels were measured in patients not pretreated with dipyridamole before and at peak pacing. In pts with normal epicardial coronary arteries, adenosine did not change upon pacing (.172±.023 to .182±.014 μM, n=3, p=NS). In pts with CAD, pacing induced ischemia elevated Ado from .106±.045 to .237±.097 μM (n=6, p<.01). We conclude that improved methods of collecting, preserving and analyzing Ado have improved our ability to detect changes in Ado in coronary sinus blood. These early data suggest that coronary sinus Ado warrants further evaluation as an indicator of myocardial ischemia.

DOES PREVIOUS BYPASS SURGERY INCREASE RISKS IN ELECTIVE PERCUTANEOUS CORONARY ANGIOPLASTY?

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Reoperation for graft occlusion and/or progression of native coronary artery disease is associated with increased morbidity and mortality. To determine the efficacy of percutaneous coronary angioplasty (PTCA) in patients with previous bypass surgery (pCABG), we retrospectively examined the records of 2,008 consecutive pts with pCABG undergoing elective PTCA. The mean age of this group was 61.0±9.1 years and 83.5% were men. 72.3% had 3 vessel native coronary disease; 19.2% were ≥70 years old; 20.3% had an ejection fraction (EF) ≤40%. More than 1 lesion was attempted in 66.6% of these pts with an overall success rate of 94.5%. 37.1% of the pts had at least 1 graft dilated. 12.8% of the native vessel dilations were performed through a vein graft and 2.2% were through an internal mammary graft. The in-hospital mortality, procedure-related myocardial infarction (MI), and urgent CABG rates were 0.9%, 1.6%, and 0.9% respectively. These data were compared to 6,714 pts without pCABG who underwent elective PTCA during the same time period. There were no significant differences between the groups except pts with pCABG had poorer left ventricular function and more severe native vessel disease (EF ≤40%: 20.3% vs. 9.4%, p<.001; 3 vessel disease: 72.3% vs. 24.0%, p<.001). The rate of acute occlusion was similar in the two groups (pCABG=3.1% vs. no pCABG=3.2%). Previous CABG did not confer any additional risk for MI, death, and urgent CABG. A logistic regression model was constructed using the above data from the entire group of 8,722 pts to determine the relative risk of pCABG on procedural mortality. The multivariate odds ratio for no pCABG was 1.5 suggesting pCABG is associated with reduced mortality (p<.01). **Conclusions:** 1) PTCA is a superior alternative to reoperation with a low incidence of complications and high primary success. 2) PTCA in pts with pCABG carries no additional risk when compared with pts without pCABG and may be associated with decreased procedural mortality.

Monday, March 19, 1990
2:00PM-3:30PM, Room 37
Coronary Angioplasty

FOLLOW-UP AND BIDIRECTIONAL CROSS-OVER BETWEEN ANGIOPLASTY AND BYPASS SURGERY: 8 TO 11 YEAR FOLLOW-UP

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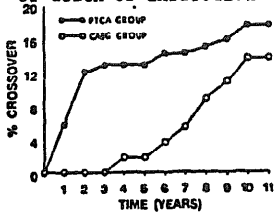
Both angioplasty (PTCA) and bypass surgery (CABG) may be followed by subsequent cross-over to the alternate therapy. To determine the frequency of cross-over, we followed 199 pts who underwent attempted single vessel PTCA between 1978 and 1981 (prior to scheduled CABG). PTCA was successful in 143 pts (PTCA group). Elective CABG was performed within 2 months of an unsuccessful but uncomplicated PTCA attempt in 56 pts (CABG group).

The reasons for unsuccessful PTCA were primarily technical (due to early PTCA equipment). There was no significant difference with respect to age, sex, risk factors, ejection fraction, prior infarction, or number of diseased vessels.

Life table analysis: follow-up was 98% complete at a mean of 9 ± 1.1 yrs. At 9 yrs, the actuarial likelihood of the PTCA group crossing over to CABG and of the CABG group crossing over to PTCA was similar (15.9 vs 10.9%, NS), however cross-over in the PTCA group occurred earlier (21±29 vs 76±29 months, p=0.004). The 9 yr risk of death or infarction was similar for the PTCA and CABG groups (10.9 vs 12.5%, NS).



Conclusion: 1) treatment cross-over was similar for PTCA and CABG groups. 2) PTCA to CABG cross-over occurred early, CABG to PTCA cross-over occurred late. 3) long term survival was excellent in both groups.

INFLUENCE OF SEVERITY OF INTIMAL DISSECTION AFTER CORONARY ANGIOPLASTY ON IN-HOSPITAL OUTCOME

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New therapies, e.g. stents, may help treat intimal dissections (ID) after coronary angioplasty (PTCA). To assess which ID will have an adverse effect on hospital outcome, all pts with ID after PTCA were evaluated. There were 991 pts with ID after PTCA; 866 pts (87%) had small ID resulting in residual stenosis <50% (Group [Gr] 1) while 125 (13%) had major ID resulting in >50% stenosis (Gr 2). Of Gr 1 pts, the artery remained patent in 803 (93%, Gr 1A), persistent coronary occlusion occurred in 9 (1%, Gr 1B), and transient occlusion redilated in 54 (6%, Gr 1C). Of Gr 2 pts, the artery remained patent in 86 (69%, Gr 2A), persistent coronary occlusion occurred in 22 (18%, Gr 2B), and transient occlusion redilated in 17 (13%, Gr 2C). In hospital outcome was:

	Gr 1			Gr 2		
Events %	1A	1B	1C	2A	2B	2C
N	803	9	54	86	22	17
MI in hospital	2.2	44.4	25.9	8.1	31.8	17.7
Repeat PTCA	3.4	0	3.7	2.3	0	11.8
CABG in hospital	5.4	88.9	18.5	32.6	72.7	58.8

Conclusion: The severity of ID after PTCA has considerable influence on in hospital events. If the ID is small and there is no in laboratory closure, outcome is excellent. With large ID, the incidence of in-hospital complications is considerably increased even if the artery is successfully redilated; in these pts, new treatment strategies are required.