DIFFERENTIAL INTRAMYOCARDIAL PLATELET BEHAVIOUR IN DIFFERENT FORMS OF ISCHEMIA.

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Although platelet activation has been suggested to occur during transient acute myocardial ischemia, the evidence supporting this hypothesis is still conflicting. We measured the great cardiac vein-arterial difference (GCV-A) of platelet factor 4 (PF4) on serial simultaneous samples obtained in patients (pts) with stenosis of the left anterior descending in whom myocardial ischemia was induced by atrial pacing (7 pts with exertional angina) or ergonovine maleate (5 pts - 3 with variant angina and 2 with angina at rest and ST segment depression). Six patients with normal coronary arteries and a negative pacing test, and 3 pts unresponsive to ergonovine served as controls. During pacing induced ischemia the GCV-A difference decreased from 43 ± 3 to -47 ± 3 ng/ml (x±SD, P < 0.001, student t test); no significant differences were found in the control group. In contrast, during ergonovine induced ischemia the difference increased significantly from -9 ± 26 to 103 ± 66 ng/ml (P < 0.001); no appreciable changes were observed in the 3 pts unresponsive to ergonovine. Thus, our data indicate that activation of platelets passing through the coronary circulation is opposed by substances released during ischemia induced by pacing, whereas it is enhanced during ischemia induced by ergonovine. These findings suggest that vasoconstriction can favour platelet activation which may contribute to obstruction of the lumen and initiate thrombus formation.

EFFECTS OF ANTIANGINAL DRUGS ON HUMAN PLATELET FUNCTION.

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Calcium antagonists and long acting nitrates have proven particularly effective in both reversing and preventing episodes of acute myocardial ischemia due to a primary reduction in coronary flow. Their beneficial effects have been mainly attributed to coronary vasodilatation, but evidence is now accumulating which indicates that platelet aggregation could play a major role in the pathophysiology of this form of angina. Since "classical" antiplatelet agents such as aspirin, dipyridamole and prostacyclin have been so far disappointing in preventing acute myocardial ischemia consequent to reduction in myocardial blood flow, we investigated the effects of verapamil and isosorbide dinitrate on human platelet function. Both drugs markedly impair platelet aggregation in vivo and in vitro. The effect of verapamil is likely to be mainly consequent to slow channel blockade, while ISDN impairs platelet aggregation partially by preventing TXA2 release. The relevance of this effect for patients' management needs further assessment.