It is well known that the formation of a platelet aggregate is the first step in an arterial thrombotic process.

The changes of platelet metabolic activity, particularly thromboxane A2 release and intraplatelet c-AMP changes, are determining manifestations for platelet aggregation and platelet sensitivity to aggregating agents.

Besides a possible primitive platelet disturbance an increased platelet activity can be secondary to an activation of coagulation caused by the presence of small concentrations of thrombin or activated factor X; furthermore, changes of the plasmatic environment can induce thrombophilia through an increased platelet activity and particularly the conversion of arachidonic acid into thromboxane is significantly influenced by plasmatic cholesterol levels (1, 2, 3). An increase of shear stress (hypertension) (4, 5) can be responsible for platelet hyperaggregability; finally an increased platelet aggregation can also be due to the loss, also if partial, of endothelium athrombogenic properties.

Several prethrombotic states have, as main cause factor, a platelet activation (Fig. 1). Platelet micro-thrombi are responsible for some cases of T.I.A. carotid or vertebral; fatal and non-fatal arrhythmias (sudden death) can find in their pathogenesis the microvessels of the cardiac conduction occluded by platelet microemboli; one can postulate that some cases of non transmural myocardial infarction, not associated with coronary thrombosis, can be caused by platelet microthrombi; the release of thromboxane by platelets can influence the genesis of cerebral or coronary spasm that has a notable importance in some kinds of coronary pathology as Prinzmetal's angina or some cases of myocardial infarction arisen without a pre-existing coronary stenosis. Important is also the role played by
PRETHROMBOTIC STATES AND PLATELETS

T.I.A.
- SEVERE BUT NON FATAL ARRHYMIA
- SEVERE AND FATAL ARRHYMIA
  (SUDDEN CARDIAC DEATH)

PLATELET MICROTHROMBI
- NON TRANSMURAL MYOCARDIAL INFARCTION
  NOT ASSOCIATED WITH CORONARY THROMBOSIS?
- MICROCIRCULATION WORSENING IN PERIPHERAL ARTEROPATHIES?

PLATELET AGGREGATION WITH
THROMBOXANE A2 RELEASE AND
PROSTACYCLIN DEFICIT
- CORONARY SPASM?

ACTIVATION OF BLOOD COAGULATION ASSOCIATED WITH
HYPOFIBRINOLYSIS AND PLATELET ACTIVATION
- ATERO-THROMBOTIC BRAIN INFARCTION
  MYOCARDIAL INFARCTION WITH THROMBOSIS
  ACUTE PERIPHERAL ARTERY OCCLUSION

Fig. 1

CORONARY Atherosclerosis

INCREASED PLATELET ADHESION ON SUBENDOTHELIUM
THROMBOXANE A2 RELEASE
- LDL
- SMOKE
- 15 HPAA
INHIBITED GENERATION OF PROSTACYCLIN
- CORONARY SPASM

Fig. 2