PART VI

SUMMARY and SUGGESTIONS FOR FURTHER WORK

Endothelin *is newly discovered powerful vasoconstrictor peptide that may be secreted from the vessel wall endothelium in response to various stimuli such as hypoxia, or cold.*

To examine whether endothelin modulates coronary tone in acute myocardial infarction (AMI), plasma immunoreactive endothelin-1 "ir-ET-1" were measured in twenty patients with acute myocardial infarction diagnosed on standard electrocardiographic changes, and serum enzymes criteria, twenty patients with stable angina pectoris (SAP) two days after exercise stress test, and, twenty control subjects.

Other investigations were done such as serum uric acid, creatine kinase, fasting blood sugar, cholesterol, and triglycerides. Wall motion abnormality index (WMAI) was done for patients with acute myocardial infarction. Blood samples in patients with AMI were taken on the first day of admission, on the second day and on the fourteenth day.

In patients with SAP, blood samples were taken two days after exercise stress test. In patients with AMI, the plasma level of ir-ET-1 was elevated in the first and seventh days of AMI, and, dropped by the fourteenth day to normal control levels.

There was no difference between patients with SAP, and, control subjects. The plasma ir-ET level showed a positive correlation with AMI, and, was highest in patients in whom Killip Subset was Class IV.

It has been found that:

1 - ***Plasma ir-ET-1 increased in the acute phase of AMI.***

2 - ***The elevation in plasma ir-ET-1 can be considered among other factors partially responsible in establishing AMI.***

3 - ***There is a direct correlation between the degree of rise in ir-ET-1, and, the extent of AMI.***

4 - ***The level ir-ET-1 in AMI can be used as a serological marker that can assess the severity, and, the extent of AMI.***

In summary, our findings suggest that: endothelin could play an important role in the modulation of coronary vascular tone. Further studies are required to define the exact role, and precise mechanism of action in healthy and diseased coronary arteries.