

(vii) Prostacycline and

(viii) 1/A Balloon counter pulsations.

Large number of studies are now available proving and disproving the value; limitations and risks of these interventions.

The difficulty in assessing the results is due to:

(1) Time of starting the intervention.

It is known that during the first Six hours of the onset of myocardial infarction (Coronary Occlusion) there is a stepwise decrease in the beneficial effects of these interventions and after this (about 9 hours) they are usually ineffective.

(2) Methods of evaluating the infarct size (Quantification)

It is generally agreed that there is no really adequate method for assessing myocardial infarct size in man. The two most commonly used parameters are Electrocardiographic and Enzymatic viz. TQ-ST Segment maps; $\Delta Q \Delta R$ indices, CPK and CPK M.B. indices.

There is lack of standardization. Secondly the natural history of S-T reduction in the 1st few hours is variable and many other factors may modify the ST segment e.g. pericarditis.

Thus new methods of assessing the infarct size in man are being sought. More recently positron emission trans axial tomography and two dimension echocardiography with computer aided semiautomated contouring system assessment of the infarct size has been applied to the study of myocardial-infarct size in man. The latter method has shown that extension of myocardial infarction may not be so common as believed earlier however expansion of myocardial infarction that is thinning and stretching with resultant widening of the previous infarction zone altering the geometry and there by function of Left Ventricle is quite commonly seen by about the 6th day. The exact cause of this is not known nor is the effect of these interventions known on "expansion" rather than extension. Thus As usual new questions has arisen, further studies of these two methods is awaited. Meanwhile among claims and counterclaims the following points appear reasonably clear:

1. Nitroglycerin improves Left Ventricular function in Acute myocardial infarction associated with Left Ventricular failure if given very early in the course of the disease without increase in M.V.O₂ whether it limits infarction or not. Its effect on Acute Myocardial Infarction without Left ventricular failure is not so clear.

2. All vasodilators are not the same and some of them may have deleterious effects.

3. Digoxin improves MV O₂ in Acute myocardial infarction with Left ventricular failure while its effect is otherwise if there is no Left ventricular failure.

4. The use of these modelities is still experimental and their use in clinical setting should be extremely careful and controlled.

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