RESULTS

1) History of Previous myocardial infarction :

Out of the 10 patients include in each group.it as found that 8 patients had infarction for the first t me in the not anticoagulated group and 6 patients in th anticoagulated group which 8 patients as experienced another infarctionprior to this recent one in the not anticoagulated and 4 patients in the anticoagulated o: e.

2) Site of the infarct in those infarction for the : irst time:

8 Patients of the not anticoagulated group and 6 of he anticoagulated infarction for the first time.

* No significantly different statistically from eac other as shown from Table (1)

3) Heart failure :

Comparing both groups, not anticoagulated and auticoagulated as regards the presence or absence of heart failure, no statistically significant difference could still be found between them. Table (2)

4) Haematocrite value :

The not anticeagulated and anticoagulated groups w re also compared as regards the percentage of patients wi h high,normal and low haematocrite values. They were com ared

three times, the whole sample, males for males and fem les for females. No statistically significant difference as found between the groups.

Table (3) a : whole sample

Table (3) b Males

Table (3) c :Females

5) **Arrhythmias :**

Comparing the not anticoagulated and anticoagulate group as regards the presence of absence of serious ar hythmias, no statistically significant difference could be found.

Table (4)

6) **High Risk and Good Risk Patients :**

Comparing the not-anticaogulated and anticoagulat d groups as regards the presence of "High" or Good risk" patients in each, there is no statistically significa t difference between the two groups.

Table (5)

7) **Systolic time intervals :**

Comparing the not anticoagulated and anticoagulat d groups as regards systolic time intervals, also, there is no statistically significant difference between the tw groups.

Table (6) (a,b)

Table (7) (a,b)

8) **Comparison between** the not - anticoagulated and anticoagulated groups as regards the early complications of acute myocardial infarction and/ or Anticoagulation :

Table (8) There is no statishically significant difference between the two groups.

Table 1: Comparison between the distribution of the s tes of myocardial infarction in the not anticoag lated and anticoagulated groups.

Site of Infarct	Not-anticoagulated		Anticoagulated	
	No	%	No	%
Anterior	5	50	3	30
Inferior	1	10	2	20
Subendocardial	2	20	1	10
Anterior + Inferior	0	0	0	0
Posterior + Inferior	0	0	0	0
Total	8	100	6	100
Mean	1.6		1.2	
S.D.	± 2.573		± 1.303	
S.E.	9273		5830	
T	365			
P	>0.05			

Table 2 : Comaprison between the distribution of the patients who were developed heart failu e in the not-anticoagulated and anticoagulated roups:

	Not-anticoagulated		Anticoagulated	
	No	%	No.	%
Heart failure	5	50	4	40
No. heart failure	5	50	6	60
Total	10	100	10	100
Mean	5		5	
S.D	± 0		± 1.414	
S.E.	0		1	
T	0			
P	> 0.05			

Table 3: Comparison between the not-anticoagulated and anticoagulated groups as regards the percentage of patients with high, normal and low haematocrite values.

(a) : Whole sample

Haematocrite value	Not anticoagulated		anticoagulated	
	No.	%	No.	%
Low	2	20	1	10
Normal	6	60	7	70
High	2	20	2	20
Total	10	100	10	100
Mean	3.333		3.333	
S.D	± 2.309		± 3.214	
S.E	1.333		1.855	
T	0			
P	> 0.05			

Table 3 (b) "Males" For Males

Haematocite value	not-anticaogulated		Anticoagulated	
	No.	%	No.	%
Low	-	0	1	12.5
Normal	5	71.4	6	75.
High	2	28.6	1	12.5
Total	7	100	8	100
Mean	2.333		2.666	
S.D.	± 2.516		±2.886	
S.E.	1.425		1.666	
T	—→ 150			
P	> 0.05			

Table 3: (c) "Females for females"

	not anticoagulated		Anticoagulated	
	No.	%	No.	%
Low	-	0	-	0
Normal	2	66.7	1	50
High	1	33.3	1	50
Total	3	100	2	100
Mean	1		.666	
S.D.	±1		±.577	
S.M.	.577		.33	
T	.499			
P	> 0.05			

Table 4 : Comparison between not-anticoagulated and Anticoagul ted groups as regards the presence or absence of serious Arrhythmias.

	Not anticoagulated		Anticoagulated	
	No.	%	No.	%
Arrhythmias	4	40	3	30
No arrhythmias	6	60	7	70
Total	10	100	10	100
Mean	5		5	
S.D.	± 1.414		± 2.828	
S.E.	1		2	
T	0			
P	> 0.05			

Table 5: Comparison between the Relative Number of Hi h& Good Risk Patients in the not-anticoagulated nd anticoagulated groups.

	Not-anticoag-ulated		Anticoagulat ed	
	No.	%	No.	%
High, risk patients	5	50	7	7
Good risk patients	5	50	3	3
Total	10	100	10	1 0
Mean	5		5	
S.D.	± 0		± 2.828	
S.E.	0		2	
T	.0			
P	> 0.05			

Table 6: (a) Systolic time intervals of the not-antic agulated on admission.

Case	QA m. sec.	L.V.E.T m. Sec.	P.E.P	P.E.P/LVET
1	415	305	112	0.369
2	413	299	113	0.378
3	410	290	120	0.410
4	388	280	104	0.365
5	408	298	110	0.365
6	410	300	110	0.366
7	408	298	110	0.365
8	374	266	108	0.407
9	372	272	100	0.367
10	391	276	113	0.413
Mean	398.9	288.4	110	0.3805
S.D ±	16.312	13.761	5.395	0.02076
S.E.	5.158	4.351	1.706	0.006566

Table 6: (b) S.T.I. of the not-anticoagulated group wi h cases of Heart failure "5" cases with cli ical evidence of H.F".

Case	QA m. Sec.	L.V.E.T. m. Sec.,	P.E.P.	P,E.P/ L.V.E.T
1	415	305	122	0.369
2	375	230	145	0.630
3	385	245	140	0.571
4	445	340	105	0.572
5	365	225	140	0.366
6	410	300	110	0.366
7	380	250	130	0.520
8	360	222	138	0.621
9	390	294	96	0.326
10	391	276	114	0.413
Mean	391.6	268.7	209.4	0.4754001
S.D ±	25.6046	40.2410	264.144	0.118805
S.E	8.0969	12.7253	83.5298	0.03756

* Cases with clinical evidence of heart failure

No. : 2, 3, 5, 7, 8

Table 7: (a) S.T.I of the anticoagulated group on admi sion.

Case No.	QA m. Sec.	L.V.E.T m. Sec.	P.E.P.	PEP/LVET
1	388	284	104	0.365
2	406	298	108	0.368
3	412	299	113	0.378
4	408	298	110	0.367
5	415	305	112	0.369
6	388	284	104	0.365
7	410	300	110	0.366
8	374	260	108	0.407
9	420	290	130	0.440
10	391	276	114	0.413
Mean	401.2	289.4	117.3	0.3838
S.D ±	14.905	13.737	7.394	0.0265
S.E	4.713	4.344	2.338	0.0084 4

Table 7: (B) S.T.I. of Anticoagulated group with case of heart failure:

Case	QA_2 m. Sec.	L.V.E.T m./Sec.	PEP	P.E.P / L.V.E T
1	368	284	104	0.3 5
2	370	278	92	0.3 0
3	412	299	113	0.3 8
4	418	308	110	0.3 7
5	360	220	140	0.6 6
6	389	284	104	0.3 5
7	400	300	100	0.3 3
8	370	225	145	0.5 1
9	374	240	134	0.5 8
10	391	276	114	0.4 3
Mean	385.2	271.4	115.6	0.4 316
S.D ±	19.9432	31.7672	17.9765	0. 14395
S.E	6.30661	10.0456	5.68467	0.0 6174

Cases with clinical evidence of Heart failure

No. : 5, 8, 9, 10

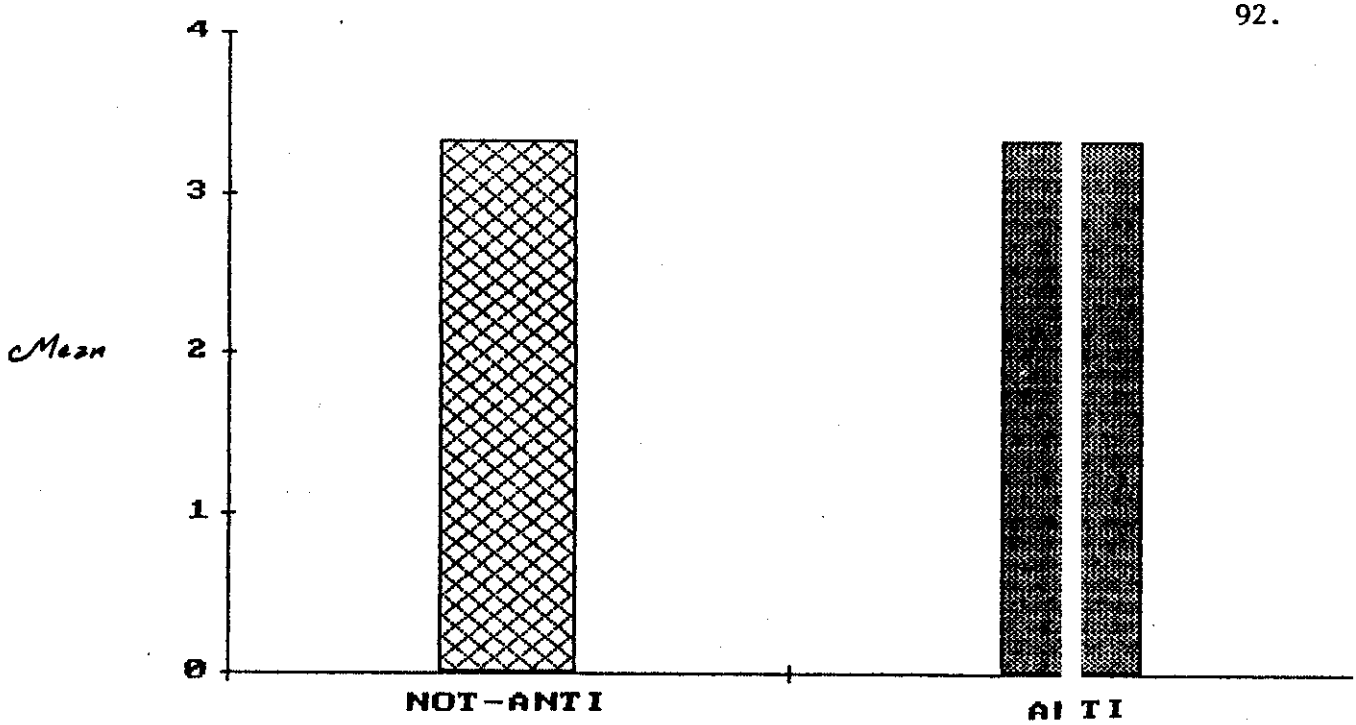

Fig (3) :Comparison between the not-anticoa ulated and anticoagulated groups as redgards th percentage of patients with high, Normal and Low aematocrite values (the whole sample)

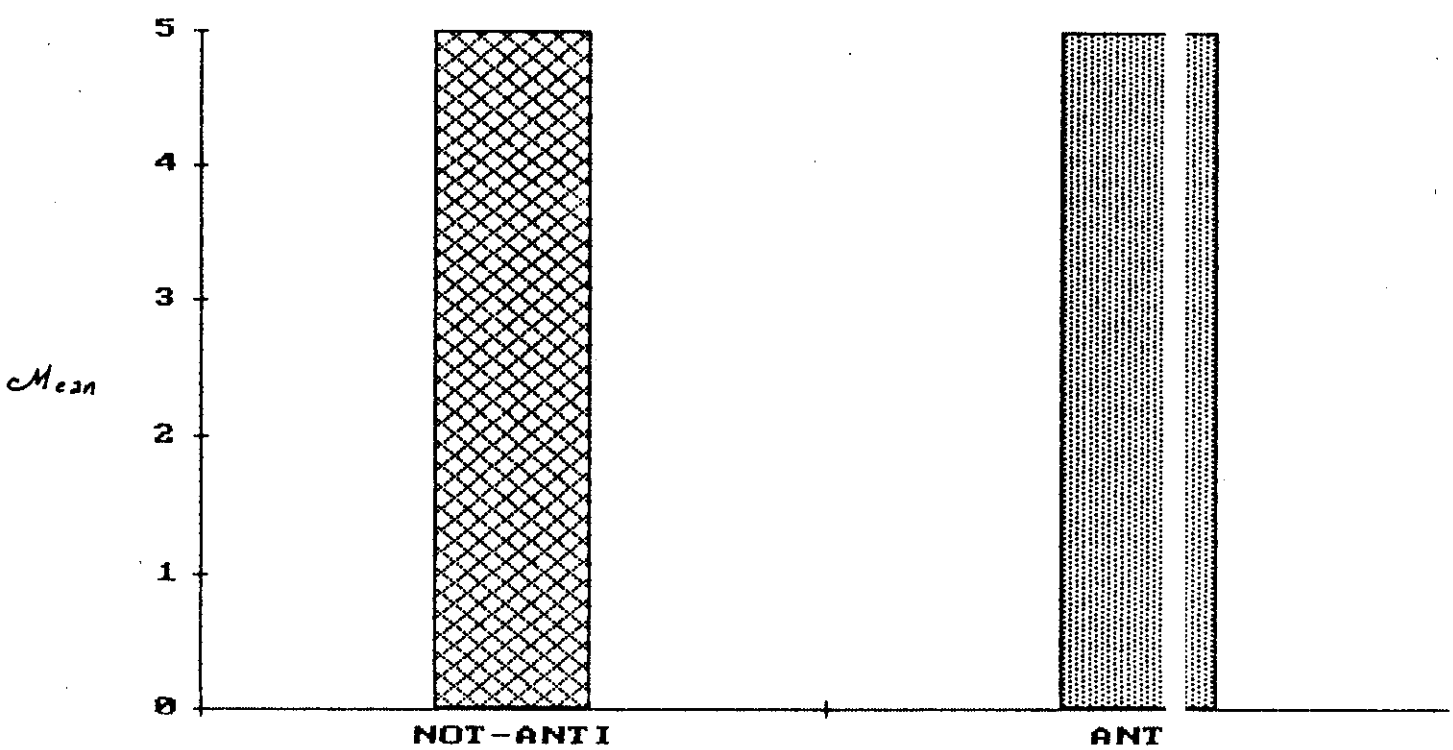

Fig (4): Comparison between thenot-anticoagulated roups as regards the presence or absence of serious arrhythmias.

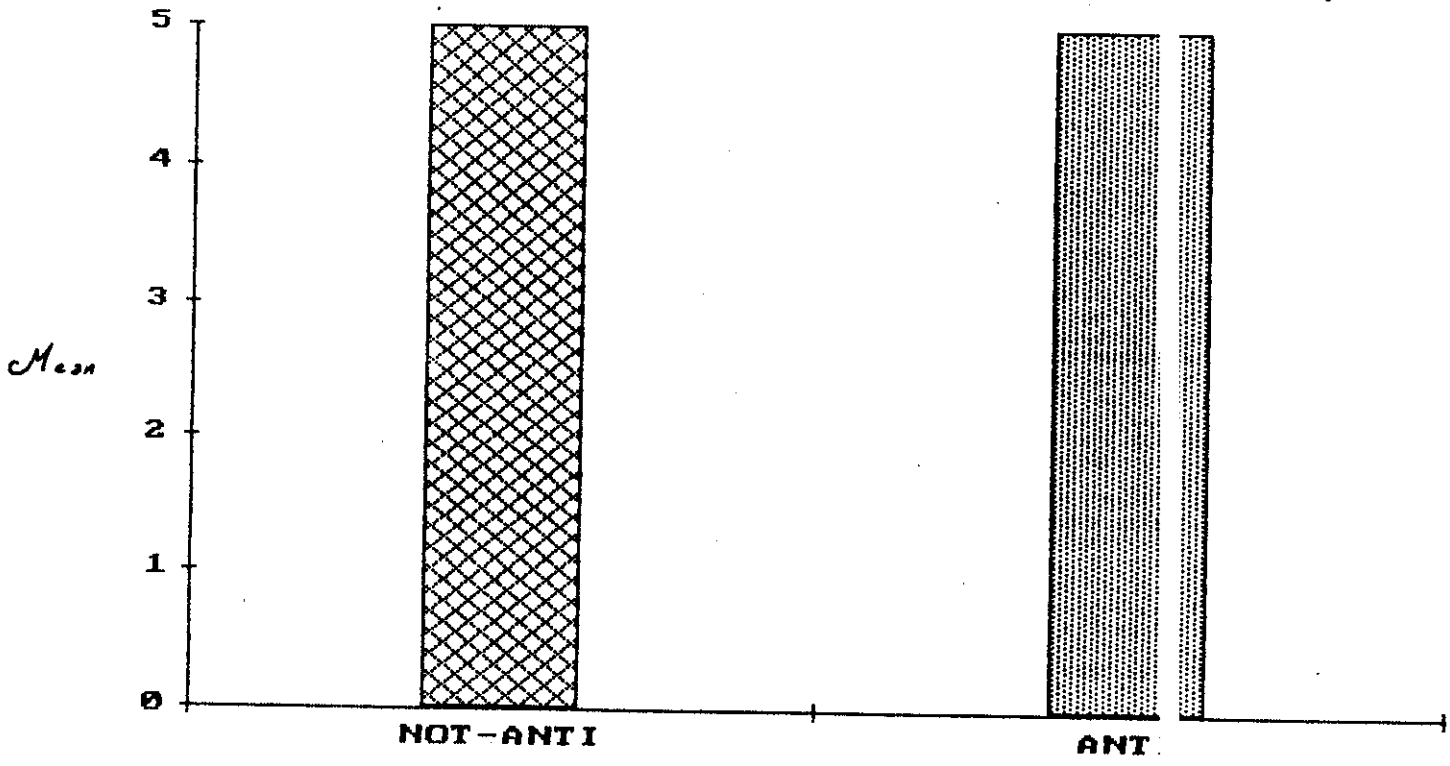

Fig (5): Comparison between the relative number of high and good risk patients in the not-anticoagulated and anticoagulated groups.

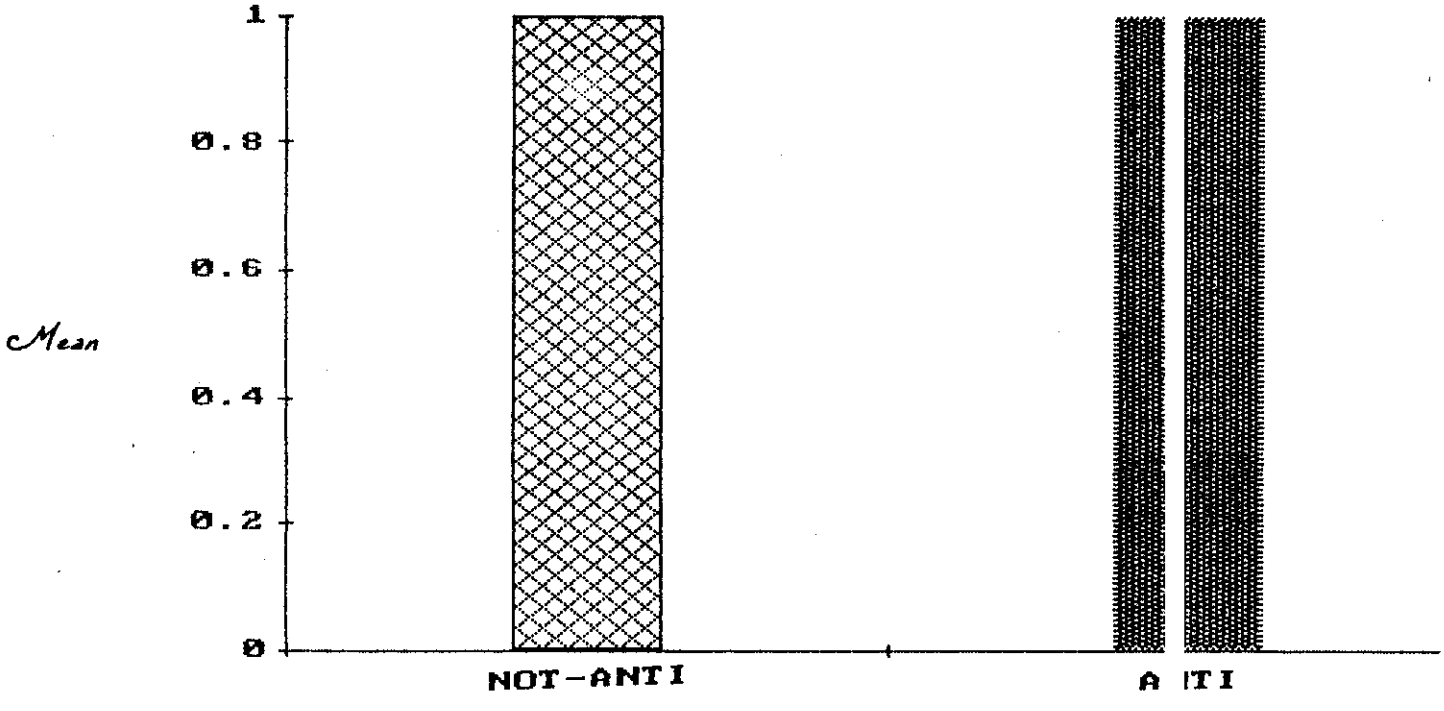

Fig (6): Comparison between the not-anticoagulated and anticoagulated groups as regards the early complications of acute myocardial infarction and / or Anticoagulation.

DISCUSSION

The rationale for the administration of anticoagulants in acute myocardial infarction is based on a) The probability that thromboembolism is less likely to occur if clotting of the blood is imparied b) mural thrombi, extension of coronary thrombosis, and peripheral venous thrombosis and embolism may be prevented by anticoagulants; and c) statistical evidence of clinical series showing a significant reduction in the incidence of thromboembolism in acute myocardial infarction among patients treated with anticoagulants. However none of these studies is conclusive (Frideberg, 1966).

As regards our results, the overall mortality in acute myocardial infarction was found to be 20% in not-anticoagulated 10% in the anticoagulated group for the period of the study (Table 7). Although this difference is statistically insignificant for the sample size and for the level of significance used, these results were in agreemant with the following studies (Gross et al., 1972 found an overall mortality in the C.C.U. around 15%. In a prospective cooperative multicenter study in 1973, it was found that mortality in the control group was 11.2% as compared to 9.6% in the anticoagulated one (statistically insignificant difference), while in the M.R.C. report 1969 it was 18 and 16% respectively (still, insignificant difference). The greater differences

found previously (Modan et al., 1975. Tonascia et al ,1975) etc... were in the pre-C.C.U. era. Some of them were criticized by Rogel and Passan 1976 and by Rapaport (1969 , as being incredible since the reported moraltity with anticoagulants was even less sometimes than that encountered in the C.C.Us. These results, however were supported again by Modan in 1976 and attributed to a still unknown pharmacological action of anticoagulants (Modan et al., 1976) and (Mitchell 1981).

Another possible explanation for this apparent inconsistency may lie in the fact that most of the studies reporting benefit on mortality were carried out for the most part during the late 1940s and 1950s (Rapaport, 1969 when the policy of treatment (prolonged bed rest) was complicated by a high incidence of life threatening thromboembolic episodes. In contrast, and parallelling the observation that a significant decrease, independent of the use of anticoagulats, has taken place over the past two decade in the incidence of thromboembolism, the major studies denying any benefit on mortality have appeared in more recent years (Rapaport, 1969).

As regards the subgroups that benefited more from the administration of anticoagulants, Szklo et al., 1979 found in a retrospective study that both complicated and noncomplicated cases did benefit from the drug. The mortality

dropped by anticaogulation from 60% in the former gro p and from 14% to 4% in the latter ($P < 0.001$). Drapkin and Merskey (1972) found that mortality in females was decreased by anticoagulant treatment from 31 to 15% especially in he "above 55 years ". age group, having moderately sev re infarctions. In males, on the other hand, mortality was unchanged by therapy (16%) though those with moderat ly severe infarctions had a significantly lower mortali y. Although Tonascia et al., 1975 found a benefit in mos subgroups under study due to anticoagulation. this t end, was not of statistical significance, but according t Modan's terms (Modan et al., 1976) "Increasing the pa ients numbers would increase the significance of the result "In our study however, no special subgroup did get any appreciable benefit from anticoagulation as compared to the others.

Extension, on the other hand, was not altered in this study by anticoagulant therapy in our results, it occ rred in 10% of the not anticoagulated and in 20% of the tr ated patients (Table 7) Rappaport, in 1969 found also tha there was not solid evidence to support the concept hat local extension of athrombus in a coronary artery co ld be presented by anticaogulants. Ebert in 1969 from ast experience, supported the same idea. The M.R.C. re ort in 1969 showed an incidence of 6.5 and 8.4% in the an ico-agulated and control groups respectively, and Estes a d

Smith 1966 could not show a significant difference b tween the two groups as pooled from 26 previous studies. c- Michael in 1960 tried to explain this failure of an icoag-ulant therapy in prevention of extension, which was lso confirmed pathologically Glueck et al., 1956 and Mc Michell and Parry, 1960). He suggested that the atheromatou: plaques to adhere and aggregate, liberating local thrombopla tins in the blood stream and causing thrombosis in spite of anticaogulation.

The cornerstone for the use of anticoagulant the apy in acute myocardial infarction is the prevention of t romb-oelism. Our study revealed a beneficial statistically significant result in this respect, the incidence of thromboembolic episodes (coronaries excluded) being 1 % and 0% in not anticoagulated and anticoagulated groups re pect-ively. Table (7). Not a single report in the litera ure we have gone through could demonstrate andetrimental ffect of anticoagulation on the incidence of thromboembolism in acute myocardial infarction. Some, however, like Feld an et al., 1952 denied a beneficial effect. On the othe hand, the vast majority of reports could prove that the inc dence of thrombembolic episodes was diminished by anticaog lants. It is mainly the extent of this drop that varies from a report to another, depending on the timing of mobiliz tion, the presence of absence of risk factors the methods o: diagnosing thromboembolism.

As regards the sites of thromboembolism, we have found an incidencd of 0 and 1% of the venous and arterial s des respectively in the control patients and 0% in the an icoa-gulated. (Table 7). These findings may raise a lot f arguments. First of all, we could not detect a singl case of deep vein thrombosis, in our study, although other studies (may be because based on radioactive fibrinogen) coul show an incdence of from 22-38%) in the control) and 0-6.! % in the anticoagulated patients (Frishman and Ribner, 197'). However, because of early mobilization adopted in our unit, and because most deep vein thrombi are of minimal im ortance In 1972 Drapkin and Merskey could find clinically an : ncidence of 3.1% that diminished by prophylactic anticaogulan s to 1.5% in females and to 1.4% in males. The M.R.C. in 1954 being earlier and using rather lower levels of antico gula-tion found on overal incidence of 9.8 and 3.8% respec ivley. The incidence of pulmonary embolism on the other hand depends mainly on its method of diagnosis: While of the orde of 2.6% when diagnosed clinically in the control patient , and 0.2% in the anticaoagulated, it reached 16 and 10% re pect-ively by lung scan (Results of a cooperative clinica trial 1973) Other authorities found values ranging fr m 4.8 to 9.4% for the control and 2-5% for the anticoag lated. (Frishman and Ribner, 1979), Also our results were in agreement with results of Meede, 1980 and Mitchell, 1 31. As regard naemorrhage, it occurred in 1% of the antico gulated

patients but in none of the controls",not anticoagula ed (Table 7). Not only were the haemorrhagic episodes ather minor, but the difference between the 2 groups was st tistically insignificant.

So the original rational for the use of anticoag lation in patients with acute MI was to impede coronary thro bosis in order to prevent MI or to limit infarct size. The studies of anticoagulation in acute MI that have been report d were not designed to assess the impact of anticoagul ion on infarct size. Furthermore, the study designs do ot permit an assessment of whether anticoagulation prev nts MI. The primary end point that does relate to ne impact of anticoagulation on coronary thromobosis in these trials is the CFR? The results in this regard were d sapp-ointing. Although the CFR was lower in the anticoagu ated group in each of these three trials, the sole differe ce achieving statistical significance was reported in th Bronx Municipal Hospital Trial, and then only in wome .

Current knowlege of the role of coronary thrombo is in acute MI, as determined by the use of fibrinolytic agents, makes it clear that treatment to lyse coronary thrombi must occur within the first 6 to 12 hours to h ve a beneficial impact on infarct size, (Khaja et al., 1)83) (Swan, 1983).

SUMMARY AND CONCLUSION

Myocardial infarction has become one of the most important diseases in technically advanced countries. Anticoagulants have been used for more than thirty-fo r years in the management of the acute phase of the dis ase but whether routine anticoagulation should be adopted or not is still not settled. This is why we planned our study in a trial to re-evaluate the possible benefits and h zards of such therapy.

Twenty patients admitted to Tanta Coronary Care Unit, suffering from acute myocardial infarction have been tudied. Ten of them were on anticoagulation therapy and ten w re not, thus serving as controls.

The patients were studied as regards the relevan risk and prognostic factors available in their files: age and sex, site and size of the infarct, presence or ab ence of previous infarction, heart failure, hyperglycaemia hypercholesterolaemia, hypertension, arrhythmias and l aem-atocrit value, systolic time Interval, The control and anticoagulated groups were evaluated statistically an they proved to be comparable.

Both groups were studied to find statistically i any correlation existed between any complications and the individual risk and prognostic factors mentioned abov .

In conclusion, by using anticoagulants in the management of acute myocardial infarction, life long serious disability may be decreased or abolished through a beneficial influence on the incidence of arterial thromboemboli. This is accomplished without singificantly altering the mortality rate in either direction.

In other words, by using anticoagulants, we are trying to add "Life to the years, if not years to the life of our patients".

CONCLUSION :

1. Anticoagulation should be the therapy of choice for acute myocardial infarchen. Preventing or impeding the progression of coronary thromboisis could prevent infarction or limit infarct size, and prevent renfarction. Furthermore, anticoagulation should reduc the incidence of two major complications of myocardial infarction systemic embolism and pulmonary embolism.

2. Mortality from myocardial infarction has markedl diminished after the advent of the era of corona y care units.

3. The incidence of thromboembolism is diminshed by early mobilization.

continued until the patient is ambulatory. We believe that low-dose heparin, given its low morbidity, is appropriate to further decrease the low incidence of pulmonary embolism. We reserve the use of full-dose heparinization to those patients at increased risk of plumonary embolism. We reserve the use of ful-dose heparinization to those patients at increased risk of pulmonary embolism. Increased risk of symtemic embolism because of past history of systemic embolism or the presence of atrial fibrillation.