THE EFFECT OF STEROIDS IN THE TREATMENT OF ACUTE MYOCARDIAL INFARCTION WITH PARTICULAR REFERENCE TO HISTOLOGICAL CHANGES

- by -

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V O L U M E I.

I N D E X.

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O B J E C T S.

(1) To study the effect of steroid therapy in cases of severe myocardial infarction, with particular reference to any alteration induced in the normal healing process in the infarct.

(2) To elucidate the possible significance of any such effects.

Introduction.

Many recent reports show that steroid therapy can frequently reverse acute heart block associated with recent myocardial infarction. Numerous animal experiments, however, have shown that such treatment influences the mechanism of the repair of infarcts and there is the possibility that this may be an adverse process resulting in mechanical weakness. Although Anfossi (1963) has shown that steroid therapy can reduce the short term mortality rate in a series of routine cases of myocardial infarction showing normal conduction, very limited knowledge is available on the repair of such human infarcts and the long term prognosis.

In this study both the pathological and the clinical aspects of this problem are considered.

A C K N O W L E D G M E N T S .

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C H A P T E R O N E .

REVIEW of LITERATURE.

The steroid drugs have not been widely used in the treatment of acute myocardial infarction despite their value in combatting acute shock (Shlesser and Asher, 1942., Perera, 1951., Cole, Grove and Montgomery, 1953., Fiegel, 1958., Neil, 1962., and Anfossi, 1963), because of the fear that the complications of such therapy might outweigh any benefit. The effects attributable to the steroids which are of particular relevance to the treatment of myocardial infarction are:-

(1) Modification of the acute inflammatory process which occurs soon after infarction, and interference in the subsequent production of a collagenised scar replacing the infarct.

(2) The promotion of sodium, chloride, and water retention.

(3) The promotion of thromboembolic incidents.

Effect on inflammatory process and scar formation.

The effect of steroids on organising granulation tissue was studied intensely as early as 1949 when Ragan and Howes reported their study of the effect of cortisone on rabbit granulation tissue. They found that the extent of the initial inflammatory infiltrate was slightly suppressed, but more importantly, the production of fibrous tissue was greatly inhibited. Baker and Whitaker (1950) showed that the local application of steroids to a simple wound had an identical effect.

Rebuck and Mellinger (1953) were able to demonstrate similar changes following the local application of cortisone to wounds in humans. They found that capillary permeability was reduced so that the initial neutrophil infiltrate was diminished in intensity. The later migration of macrophages into the zone was slowed and phagocytosis appeared to be reduced. The

proliferation of fibroblasts and the subsequent formation of collagen was inhibited so that a substantial delay in healing was observed. Noble (1953) in his review of the physiology of the adrenal hormones confirmed the opinion that they diminished all aspects of the local inflammatory reaction. Creditor, Bevans and Mundy, along with Ragan (1950) were, however, unable to confirm these findings in their study of the effect of A.C.T.H. on wound healing in humans: no adverse effects on fibrosis or on strong scar formation was observed. This view is not supported by this study.

Recently Cottrell, Wiener and Spiro (1964) have studied with the electron microscope the effect of cortisone on the essentially similar process of organisation of thrombi. They found that the initial inflammatory infiltrate was diminished as was the phagocytosis of erythrocytes. Fibroblasts were seen to appear in normal numbers but subsequent collagenisation was virtually halted. The ultimate recanalisation of the thrombosed vascular channel was greatly delayed.

It was felt that similar alterations in the repair process might affect healing myocardial infarcts. If this were so, the delay in healing might produce a weak scar with a real danger of aneurysm formation due to stretching of an incompletely healed infarct.

In 1952 Chapman, Skaggs, Thomas and Greene studied the effect of cortisone on experimentally produced myocardial infarcts in dogs and with doses of 7 mg/kg. body weight, they found no delay in phagocytosis, fibrosis, or subsequent collagen formation.

Shortly afterwards Johnson et al (1953) reported their extremely carefully controlled study of the effect of steroids on experimentally produced infarcts. This work suggested that in comparing steroid treated

T A B L E 1.

Effect of steroids on myocardial infarction in dogs produced by high and low ligation of coronary artery.

(Johnson et al 1953).

	Low Ligation		High Ligation	
	Control	Steroid	Control	Steroid
Average volume of Infarcts (c.c.)	7.8	0.96	18.8	0.14
Average body weight (Kg)	30	33	36	36
Average Heart weight (g)	104	109	117	119

animals with controls, the infarct was smaller, capillary anastomoses were better developed, and the mortality rate was reduced. No delay in repair of the infarct was seen.

This work was divided into several parts. In the first group 7 pairs of dogs were matched for weight, and in all an infarct was induced by ligating the left coronary artery 15mm. below the origin of the circumflex branch. One dog from each pair received cortisone in daily doses of 12.5 - 20.0 mg. The average body and heart weights of these two groups of animals was similar but in the steroid group the average size of the infarct was much smaller than in the controls (Table I). The results were similar in a second group of 9 pairs of dogs in which infarction was induced by ligating the left coronary artery at a higher point, 5mm. below the origin of the circumflex branch (Table I). Barium X-ray studies on these two groups showed that the intercoronary anastomoses were better established in the steroid treated animals.

A third group of 12 dogs was divided into subgroups of 4; one received A.C.T.H. for a month before high ligation of the coronary artery, the second subgroup received cortisone for one month before an identical procedure, while the third acted as controls. Both steroid subgroups showed better intercoronary anastomoses than in the control dogs.

A fourth and final group of 57 dogs received an infarct by high ligation; 23 were given cortisone and 34 acted as controls. The survival rate after 72 hours was 78% in the steroid treated dogs and 46% in the controls. In the experimental group phagocytosis of the dead myocardium was sometimes rather poor but in those that survived to the 30th. day, there was no evidence of delayed healing.

This work was challenged immediately by Opdyke et al (1953) who

performed similar experiments on 3 pairs of dogs. Infarcts were induced by tying the left coronary artery at a point varying between 9 and 34 mm. from its origin. One pair received cortisone in doses of 10 mg./kg. body weight, a further pair received 3 mg./kg. body weight, while the third group acted as controls. The animals were not paired for weight. No correlation was found between the treatment and the size of the infarct and no delay in fibrosis was reported. Hoover and Manning (1954) also using dogs, showed that the rate of development of coronary occlusion affected the prognosis. Complete sudden occlusion of the left circumflex branch was associated with a higher mortality than where the initial occlusion was partial, but was made complete 10 days later. The effect of steroids in dogs subjected to this two-stage ligation was not apparent histologically; the number studied was too small to permit comment on the mortality rates.

Hepper, Pruitt, Donald and Edwards (1955) in a study similar to that of Opdyke, claimed that while there was no apparent delay in the eventual healing of infarcts in those receiving high or low dosage steroid regimes, there was a narrowing of the zone of "tissue reaction" in the contiguous myocardium, and phagocytosis of the dead myocardium was delayed. By the 30th day healing appeared to be as advanced as in a control group.

Currently, Wexler and his associates in Cincinatti are studying myocardial infarction, both spontaneous and chemically induced in virgin and a repeatedly bred strain of Sprague-Dawley rats. Although this study has not yet been completed, several preliminary reports have been published (Wexler, 1964, a,b,c., Wexler and Kittinger, 1963).

It was observed that breeder, but not virgin, animals developed gross coronary atherosclerosis and many subsequently suffered spontaneous

myocardial infarcts. Within a few days of the subcutaneous injection of "isoproterenol" in both virgin and breeder animals there appeared areas of myocardial necrosis histologically identical with spontaneous infarcts. In rats killed at different times after receiving "Isoproternal", the infarcts underwent repair process identical to that of spontaneous infarcts.

A study was made on chemically induced infarcts in animals which also received hydrocortisone and in others who received no steroids. Marked differences were seen between the two groups. Although animals rarely succumbed to the injection, many showed evidence of severe shock; this, however, was less marked and less prolonged in the steroid treated animals. Rats were killed on different days after injection and no significant difference between the two groups was observed in the size or general appearance of the infarcts. Many showed aneurysmal bulging irrespective of treatment but there was no instance of rupture. Microscopical examination showed marked differences between the two groups. In the steroid treated infarcts the initial neutrophil infiltrate was much less than in the controls. While phagocytosis and fibrosis of the infarct proceeded well in both groups, subsequent collagen formation was delayed in the steroid animals, although eventual healing was adequate.

In both groups large amounts of a mucopolysaccharide were seen within the healing infarct, but in the controls this material was present also between the adjacent, surviving and apparently normal myocardial bundles; there was no such material in this position in steroid treated animals. The serum glutamic oxalacetic transaminase (S.G.O.T.) and serum glutamic pyruvic transaminase (S.G.P.T.) were estimated daily in all animals and S.G.O.T. levels attained a lower average peak in the steroid treated rats than in the controls; no difference was seen in S.G.P.T. levels.

The histological and biochemical results are closely similar to those of Frew and Dall (1962) and Broustet and Renner (1963, a, b), who in their review of human infarcts treated with hydrocortisone, stressed the possible danger of aneurysm formation or of late heart rupture.

These animal experiments aroused considerable interest, and Hopper's observation that steroids diminished the reaction around an infarct, together with the clinical observations of Sommerville et al (1951), and Lown et al (1955), raised hopes that steroids might be of value in the treatment of acute heart block complicating recent myocardial infarction. Sommerville noted that the atrioventricular conduction time (P-R interval) on E. C. G. tracings was higher in Addison's disease than in normal people. Lown studied 50 cases of Addison's disease, 39 of Cushing's Syndrome, and 53 of "normals" and found that the average P-R intervals in each were 0.176, 0.136, 0.158 seconds, respectively. It seemed that the amount of circulating adrenocortical hormone had some influence on the P-R interval.

Complete heart block is seen in about 4% of those with an acute myocardial infarct (Master et al 1938, Condry and Thomson 1957, Dimond, Dunn and Brosius, 1960, Lenegre, Blondeau and Rizzon (1962). Although normal rhythm may return after several days, there is an immediate mortality rate of 50% (Mintz and Katz, 1947, Penton et al 1956, Rowe and White 1958, Gilchrist, 1958, Dimond et al 1960, Lenegre et al 1962). The value of steroids in such a case was shown quickly by Prinzmetal and Kennamer (1954) who reversed complete block on two occasions in a 60 year old man, using A.C.T.H. By 1960 these authors had experience of 60 cases of complete block treated with prednisolone and they reported "good results" although they did not state the proportion in which block was reversed. There have been many

T A B L E 2.

Reports of use of steroids in Treatment of Heart Block complicating acute myocardial infarction.

AUTHOR	NO. OF CASES	BLOCK REVERSED.
Prinzmetal & Kennamer (1954)	1	1
Wohlrabe (1956)	1	1
Phelps and Lindsay (1952)	1	1
Aber and Wynjoner (1960)	2	2
Caramelli and Tellini (1960)	7	5
Friedberg *et al.* (1960)	3	3
Pay and Waverley (1961)	2	2
Dall and Peel (1962)	15	14
Grieco and Andreae (1963)	1	1
Total...	33	30

subsequent successes with steroid therapy in similar patients (Table II) and usually the restoration of normal conduction was achieved within a few hours. Successes have also been reported in the treatment of chronic block with Stoke-Adams Seizures (Litchifield et al 1958, Friedberg et al 1960, Fay and Waverley, 1961).

In few of these reports have there been any comments on delay in the healing of the infarct. Grioco and Andrea (1963) gave a detailed description of their patient who died 2 months after a myocardial infarct complicated by heart block and treated successfully with massive doses of hydrocortisone. Although the authors claimed that healing of the infarct was completely normal, they described aneurysmal dilatation of the recent infarct and the affected left ventricular wall was only 3 mm. thick.

Several reports stress the value of steroids in acute heart block with acute shock, and state that no complications were encountered (Gerish and Campeau, 1958, Fiegal, 1958, Kaiser, 1960). Anfossi (1963) studied two groups each of 50 patients all with recent infarcts but with normal conductions; one group received prednisolone and the other acted as controls. In the first day only one steroid treated patient died compared with 11 controls, but thereafter the death rates were more closely similar, a further 8 steroid treated and 5 controls dying by the end of the third month. This difference Anfossi attributed to the control of severe shock by steroids; he encountered no complications but did not report pathological findings.

Weil (1962 and Broustet and Remmer (1963 a and b) admitted that steroid treatment was of value in acute shock and conduction abnormalities but mentioned the danger of fluid retention and warned that such therapy was still experimental in nature.

Effect on Electrolytes.

Sprague et al (1950) found that while cortisone could cause sodium, chloride and water retention, this effect was seen only where large doses were administered. 100 mg. of cortisone acetate given daily produced no such effect, but when this dose was doubled there was an immediate slight retention of sodium, chloride and water, together with a minimally negative potassium balance. Luft and Sjorgen (1951) confirmed this and noted that the retention of salt and water was dependent on the continued administration of cortisone. Withdrawal of the steroid resulted in an immediate salt and water diuresis with retention of potassium.

These findings suggest that if steroids were used following an infarct there might be a danger of promoting fluid retention and precipitating cardiac failure. Camara and Schemm (1955), however, found that the administration of A.C.T.H. to patients already in congestive failure resulted in a diuresis of salt and water. Gutner et al (1957) confirmed this and showed that this action was potentiated by the administration of mercurial diuretics. Dall (1962) found no evidence to suggest that steroids increased the incidence of severity of congestive failure; steroids were given in large doses for a short time only and diuretics were used immediately failure became apparent.

Thromboembolic effects.

Cosgriff et al (1950), and Cosgriff (1951) noted thromboembolic incidents, usually thrombophlebitis of leg veins, in patients receiving A.C.T.H. and cortisone. The widespread use of anticoagulants in cases of myocardial infarction has now largely overshadowed this complication, although McMichael (1964) now doubts the value of anticoagulants owing to the risks of haemorrhagic complications and lack of evidence that they confer any long

term benefit.

The general consensus of world opinion, based on these animal experiments and on clinical and pathological observations in man, is that while steroids will very often reverse acute heart block and acute shock, and as a result reduce the immediate mortality rate, there is also a considerable modification in the healing process of the infarct. In particular, the possible delay in the formation of a densely collagenised scar has raised doubts about the mechanical strength of the repair with the theoretical danger of subsequent aneurysm formation and late cardiac rupture.

C H A P T E R T W O .

MATERIAL and METHODS.

Selection of patients by coronary prognostic index.

Standard treatment regimes.

Steroid regimes.

Investigations and points noted during admission.

Follow-up.

Postmortem Examination - Naked eye and Microscopic procedures.

Additional control groups.

Detailed counts of cell population within infarcts.

INTRODUCTION.

Following an earlier trial in which the efficacy of steroids in cases of acute heart block associated with myocardial infarction was examined, Dall and Buchanan (1962), it was decided to undertake a more prolonged investigation to determine whether the steroid group of drugs was of any value in cases of severe myocardial infarction, whether or not associated with heart block. This investigation was undertaken in Wards 2 and 9 (Dr. Peel's Unit) of the Victoria Infirmary, during 1961-1963 inclusive.

SELECTION of PATIENTS.

Because a high proportion of fatalities associated with acute myocardial infarct occur during the first two days of the illness, Peel et al (1962) it was decided that the investigation would be limited to patients admitted to hospital within twenty-four hours of the incident. Patients suffering episodes of severe chest pain during the week preceding admission were also excluded even if the final episode suggestive of an infarct had taken place within twenty-four hours of admission; in such cases an infarct might have occurred during an earlier episode of pain and the episode occurring just prior to admission might not, in fact, have been associated with a further infarct. Mild angina during the period before admission did not exclude the patient.

All patients who gave a history acceptable by the above criteria were included in the investigation.

ASSESSMENT of SEVERITY of INFARCT.

Since the study was to be limited to "SEVERE" cases of myocardial infarction only, a method both clear cut and simple to use had to be selected to determine the severity of each case. Assessment of the CORONARY PROGNOSTIC INDEX, Peel et al (1962) was the method chosen; this is claimed to give a guide to (1) the severity of the infarct, (2) the prognosis up to the 28th. day, and (3) a less definite indication of the longer term outcome. Over many years the importance of various factors in prognosis have been stressed. Master et al (1939), Baer and Frankal (1944), Sigler (1951), Barr (1955) and Peel (1955) all show that the prognosis is less good in older patients. Master, Dack and Jaffe (1937), Chambers (1946) and Billings et al (1949), have discussed the grave prognosis associated with congestive cardiac failure in the early post infarct period. That heart block associated with a recent infarct carries an immediate 50% mortality rate has been recognised by Kerr (1937), Master, Dack and Jaffe (1938), Mintz and Katz (1947), Renton, Miller and Levine (1956), Condry and Thomson (1957), Plotz (1957), Gilchrist (1958), Rowe and White (1958), Dimond, Dunn and Brosius (1960), and Lenegre, Blondeau and Rizzon (1962).

In the past various methods of assessing the prognostic significance of different clinical and electrocardiographic features of recent myocardial infarcts have been devised by Rosenbaum and Levine (1941), Rathe (1942), Woods and Barnes (1942), Master, Jaffe, Dack and Grishman (1944), Jacobs (1951) Schnur (1953) and Wooten and Kyser (1953). Many of the methods are complica-:ted and difficult to perform, and are quite unsuited to mass routine use.

To meet the need for a simple method, the Coronary Prognostic Index was devised over a period of years in Dr. Peel's wards. The Index

TABLE 3.

Components of the Coronary Prognostic Index.

SEX and AGE:	
M. 54 or under	0
55 - 59	1
60 - 64	2
65 or over	3
F. 64 or under	2
65 or over	3
PREVIOUS HISTORY:	
Previous cardiac infarct, established or strongly suspected, (Prodromal symptoms within 4 weeks of the onset are excluded).	6
Other cardiovascular diseases or history of exertional dyspnoea. History of "coronary insufficiency".	3
Effort angina only.	1
No cardiovascular disease.	0
SHOCK:	
Absent.	0
Mild - transient at onset.	1
Moderate - present on admission but subsiding with rest and sedation.	5
Severe - persisting despite rest and sedation.	7
FAILURE:	
Absent.	0
Few basal râles only.	1
Any one or more of the following:- Breathlessness; acute pulmonary oedema; orthopnoea or dyspnoea; gallop rhythm; liver enlargement; oedema or jug. vein distension.	4
RHYTHM:	
Sinus.	0
Any one or more of the following:- Atrial fibrillation; flutter; paroxy tachycardia; Simple tachycardia (110 or more); extrasystoles; nodal rhythm or any grade of heart block.	4
E.C.G:	
Normal QRS, changes confined to RT or T.	1
QR complexes.	3
QS complexes or bundle-branch block.	4
(If no E.C.G. obtained before death, assume a score of 4).	

takes account of the following factors:-

(1) The age and sex of the patient.

(2) The previous cardiac history.

(3) The degree of shock present on admission.

(4) The degree of cardiac failure present on admission.

(5) The nature of the cardiac rhythm.

(6) The nature of the electrocardiographic changes.

Each of these six factors is subdivided into clearly defined subgroups which give a score for severity; by trial and error a system was devised whereby a number of points was awarded for each subgroup. Definitions of each subgroup and of the appropriate score are shown in Table 3.

METHOD OF USING INDEX.

ITS USE IN SELECTION OF PATIENTS.

All patients considered for possible inclusion in the investigation were examined immediately after admission by at least two clinicians who were familiar with the method of scoring. Generally it was possible to complete the necessary clinical examination in under two minutes thus causing the minimum disturbance to the patient. As soon as possible a 12 lead electrocardiograph tracing was made (the standard technique used is described later in this chapter) and the tracings were examined independently by the same two clinicians.

Nearly always the scores were identical, but whenever a difference occurred this was resolved by re-examination of the patient.

During 1961-63 inclusive the index was used routinely in Dr. Peel's

FIG. 1.

MORTALITY RATE AT 28 DAYS RELATED TO PROGNOSTIC INDEX SCORES.

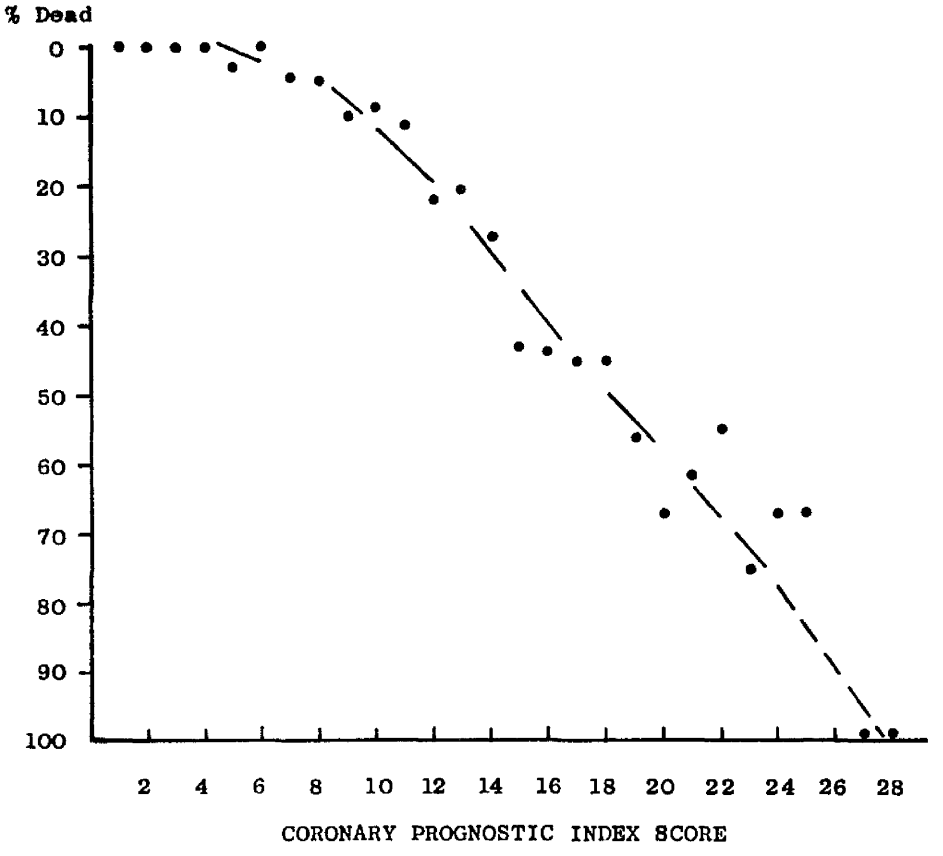


TABLE 4.

Reasons for failure to assess coronary prognostic index in 13 patients admitted with a recent myocardial infarction.

REASON.	NUMBER
Died before assessment was completed	8
Presence of severe co-existant disease:-	
(1) Shocked with severe haemolytic anaemia	1
(2) Advanced bronchial carcinoma	2
(3) Acute exacerbation of chronic lymphatic leukaemia	1
(4) Severe renal failure	1

wards. Over half of the patients with a score of 17 or above died during the first 28 days while in those with a score below 17 the mortality rate during the first 28 days was less than 25%. Figure 1 shows the percentage mortality rates at 28 days after infarction, related to the index score.

On the basis of this information it was decided that a "SEVERE" infarct would be defined as one in which the coronary prognostic index score, assessed within 24 hours of onset, was 17 or above.

During 1961-63 inclusive, 616 patients were admitted to Dr. Peel's Unit with a myocardial infarct of under 24 hours' duration and the index was assessed in 603; of the remaining 13 patients, 8 died before the assessment could be completed, and 5 were excluded because of the presence of severe intercurrent disease. Details of these are given in Table 4. A further 3 patients were excluded because the case records could not be found.

The distribution of the scores in the remaining 600 patients is given in Table 37, Vol. II; 149 had a score of 17 or above and these were accepted for this investigation. A summary of the criteria used in the selection of these 149 patients is:-

(1) The patients must have been accepted as a definite case of myocardial infarction of less than 24 hours' duration.

(2) The coronary prognostic index must have been assessed within 24 hours of the onset of the infarct.

(3) The index score must have been 17 or above.

TREATMENT.

The routine regime in all 149 cases was:-

(1) Bed rest: complete initially; later gradual mobilisation so that the patient became ambulant after about 4 weeks.

(2) Diet: light during the first week; no restriction thereafter except that added salt was prohibited in congestive cardiac failure.

(3) Anticoagulant therapy commencing with an initial intravenous injection of 10,000 units of heparin followed by Warfarin Sodium in doses determined by daily thrombotest estimations.

(4) Whenever indicated: morphine hydrochloride, omnopon, pethidine, digoxin (i. v. or oral), chlorothiazide, mercalyl, aminophylline, procaine amide, quinidine, oxygen, ferrous sulphate.

(5) In the event of sudden "death" external or internal cardiac massage was employed using a defibrillator, but such measures were attempted only in younger patients and were withheld where there was evidence of very extensive myocardial damage.

USE OF STEROID THERAPY.

In addition to the above standard regime certain patients received steroid therapy. It had been intended to use a double blind method of trial but this proved impracticable. In an earlier investigation using the double blind technique certain irregularities were discovered as a result of which patients in higher social groups received one particular form of treatment. It was considered that the danger of similar interference in this investigation could not be excluded and therefore a different method of selection was employed taking advantage of the following two circumstances:-

(1) It had been observed that the number of admissions of myocardial infarction was similar during - (a) the morning and afternoon and (b) the evening and night.

(2) It was considered desirable that this experimental use of steroid therapy should be commenced at a time when senior medical staff was readily available, i.e. during the daytime.

Thus steroid therapy was administered to those patients admitted between the hours of 7.00 a.m. and 5.00 p.m. approximately. Patients admitted outwith these hours did not receive steroids and formed a convenient control group. Thus during the 3 years of the investigation 74 patients received steroid therapy and 75 acted as controls.

STEROID REGIMES.

Steroid treatment was started within 4 hours of admission in all 74 patients; although most were managed under one regime using hydrocortisone (Series A), there were four distinct variations (Series B. C. D. and E.) using steroids other than hydrocortisone alone, in approximately equivalent doses, Sopper, Dorfman and Gabrilove (1961). Thus the 74 patients cannot be considered as belonging to one series but instead are regarded as forming 5 separate though very closely related groups, designated A, B, C, D and E respectively. Details of each are given below.

SERIES A. 49 Patients. Hydrocortisone hemisuccinate was given in these doses:- Day 1, 200 mg. by intravenous injection followed immediately by 200 mg. by intramuscular injection. On days 2-12 inclusive treatment was slowly tailed off with intramuscular doses of 300, 300, 200, 200, 150, 100, 100, 100, 50, 30 mg. On days 13 and 14 intramuscular A. C. T. H. 80 and 40 units respectively

was given.

SERIES B. 4 patients. Hydrocortisone hemisuccinate was given in doses larger than in Series A: First day - 300 mg. intravenously, soon after admission, repeated after 6-8 hours: During the following 14 days diminishing intramuscular doses of 400, 300, 300, 300, 200, 150, 150, 150, 100, 100, 100, 50, 50 and 50 mg., on the next two days, as in Series A, A. C. T. H. 80 and 40 units.

SERIES C. 12 patients. The steroid regime was started as in Series A with hydrocortisone hemisuccinate 200 mg. I. V. and 200 mg. I. M. During the next 11 days diminishing oral doses of prednisolone, 30, 30, 30, 25, 25, 20, 20, 15, 15, 10 and 10 mg. On the 13th and 14th days A. C. T. H. as before.

SERIES D. 5 patients. The steroid regime started as in Series A with hydrocortisone hemisuccinate 200 mg. I. V. and 200 mg. I. M. During the next 11 days treatment was tailed off with betamethasone ("Betnelan") intramuscularly, the daily amounts given being 8, 6, 6, 4, 4, 4, 3, 3, 2, 2, 2 mg. On the 13th and 14th days A. C. T. H. was given as before.

SERIES E. 4 patients. These received betamethasone ("Betnelan") only, 8 mg. intravenously soon after admission and on the following 11 days, intramuscular doses of 6, 6, 4, 4, 3, 3, 2, 2, 1½, 1 and 1 mg. On the next 2 days A. C. T. H. was administered intramuscularly in doses of 40 and 20 units.

CONTROL CASES.

The 75 patients admitted during the evening and night served as a control group and were designated Series F. These received the standard treatment already described. In 6 cases, however, following a marked sudden deterioration in their clinical condition occurring 3-6 days after admission, hydrocortisone was administered in large amounts as a possible emergency life saving measure. These 6 cases have been excluded from Series F. and are

designated separately as Series G. Certain laboratory results obtained BEFORE the administration of steroids are relevant and are recorded in Chapter 6.

INVESTIGATIONS AND POINTS NOTED ON ADMISSION.

The total duration of each admission was noted and the following investigations were performed routinely:-

(1) Serial electrocardiograph studies.

(2) Estimation of serum transaminases.

(3) Haematological examination of venous blood.

The systolic and diastolic blood pressures were recorded at least once each day and anticoagulant therapy was controlled by daily thrombotests estimations. Serum electrolytes were estimated in many patients at different times after infarction.

Details of the techniques used are given below.

ELECTROCARDIOGRAPH STUDIES.

As part of the initial assessment of the coronary prognostic index a 12 lead cardiogram was recorded soon after admission. Further tracings were taken later that day, on the following day and thereafter on alternate days until discharge or death. Additional tracings were taken whenever indicated by the patient's condition. So that different tracings from each patient could be compared with one another the following measures were taken:-

(1) Whenever possible all records were taken on identical Cambridge portable instruments which recorded by means of a heated stylus acting on moving sensitised paper. The stylus movement was carefully standardised before use.

(2) Whenever possible tracings from an individual patient were made by the same member of the cardiology department technical staff to ensure the greatest possible uniformity in the positioning of the chest leads and standardisation of the machines. Records taken outwith the normal working hours, however, were usually made by members of the medical staff, and changes of the cardiology technical staff occurred from time to time.

(3) Since even a small positional change in the chest leads can affect the tracing, the position of each lead was marked on the chest wall with carmine as a guide for further tracings. While this was of considerable value in male patients, the normal hygiene procedures in females resulted in obliteration of the marks within a short time.

The cardiograph tracings were examined in detail and the following features noted:-

(1) The presence of an infarct pattern, recent or old, was sought - (a) as an aid to diagnosis: (b) as a guide to the position and size of the infarct.

(2) Details of the infarct pattern were noted where the maximum abnormality on admission involved the Q. S., Q. R., or merely the T waves and the appropriate number of points for the coronary prognostic index was awarded.

(3) The P.-R. interval was measured in all cases by dividers so that the distance on the tracing could be expressed up to 1/100 second. The P.-R. interval on admission, on the 8th. day, and on the 28th. day, or on discharge, if sooner, was noted and the changes in the average values in the steroid treated and control groups were compared. The

presence of bundle branch block, or of interventricular block (complete, partial or latent,) was noted and any changes in the degree of block assessed.

(4) The nature of the rhythm was noted, and the result scored. The study of the electrocardiograph records was regarded as the only reliable method of distinguishing between arrythmias, e. g., differentiating between sinus bradycardia and heart block, or between certain tachycardias.

SERUM TRANSAMINASES.

The transaminases (S. G. O. T. and S. G. P. T.) were assessed on venous blood specimens obtained from 110 patients. Those estimations were performed in the Department of Biochemistry in the Victoria Infirmary using the method of Cabaud, Leeper and Wroblewski (1956), as modified by Mohun and Cook (1957). The accepted normal valves in Cabaud Units were S. G. O. T. 15-40, S. G. P. T. 10-30.

During 1951 those estimations were of limited availability and therefore in 10 patients they were performed once only; in 6 others with undoubted infarcts no estimations were made. In the remaining patients estimations were performed at least twice during the first 3 days unless death supervened. In many cases additional estimations were performed on the 4th. and 5th. days; in all, 279 results were obtained for each enzyme.

These results have been averaged and related to the treatment given.

HAEMATOLOGICAL EXAMINATION OF VENOUS BLOOD.

In all cases the haemoglobin was estimated by the clinical staff using a Sahli haemoglobinometer, and a blood film examined. In only 35 patients was a venous blood sample submitted to the haematology department as an additional check. In the laboratory a cyan-photoelectric method of haemoglobin estimation was used. The haemoglobin levels in the steroid treated and the control patients have been averaged and compared.

SERUM ELECTROLYTES:

This was not undertaken routinely. In view of the observations of Sprague et al (1950), Luft and Sjorgen (1951) and Sjorgen (1952) who noted that there was a negative potassium balance when large doses of cortisone were administered; and of Camara and Schemm (1955), and Gutner et al (1957), who showed that the administration of A. C. T. H. to patients in congestive failure resulted in a salt and water diuresis, it was felt that thos estima-tions which were performed should be assessed to determine whether any difference existed between steroid treated and control patients.

The serum electrolytes were estimated on 82 occasions by those methods:-

(1) Sodium and Potassium - by flame photometry.

(2) Chloride - by titration with silver using a meter to detect the end point.

(3) CO_2 - by Van Slyke gas analysis.

The normal range of values accepted for this study (in m. eq./litre) are Sodium 137-149, Potassium 4.0-5.4, Chloride 96-106, CO_2 24-29.

78 of those estimations were made during the undernoted periods:-

Time (Days)	Number of estimations.
2-3	21
6-8	33
13-15	13
21-28	11

The results for the steroid treated and the control patients have been averaged for each time period, and compared with each other.

FOLLOW-UP OF DISCHARGED PATIENTS.

55 patients were discharged from hospital: 33 steroid treated and 22 controls.

The sources of subsequent information include:

(1) The hospital anticoagulant clinic.

(2) Various hospital outpatient and inpatient departments.

(3) The hospital social service department.

(4) The family doctor of each patient.

(5) The appropriate Registrars of births, marriages and deaths.

A standard method of collating this information was devised. It is designed to reflect the overall condition of the patients, and comprises the following five grades:-

GRADE I. Patient clinically entirely well and without complaints or disabilities referable to the cardiovascular system.

GRADE II. Patient generally fit and able to undertake most normal activities although at times there might be slight dyspnoea

TABLE 5.

QUESTIONNAIRE SENT TO FAMILY DOCTORS OF PATIENTS DISCHARGED FROM HOSPITAL.

Please mark with a cross the category which most accurately describes the patient's present condition. If the patient has died please indicate the date and cause of death.

NAME ADDRESS		SERIES NO. DATES IN HOSPITAL
CATEGORY I	Symptom free and apparently entirely well.	
CATEGORY II	Relatively well; able to undertake normal activities; slight cardiac symptoms but no C. C. F.	
CATEGORY III	Troubled by dyspnoea, angina of more than slight degree, or by C. C. F.	
CATEGORY IV	Cardiac cripple.	
CATEGORY V	Patient dead.	

and/or angina of effort. Congestive cardiac failure not present.

GRADE III. Patient in fair or poor health. Activities restricted significantly by exertional dyspnoea, angina, congestive cardiac failure, or other cardiac abnormality producing significant symptoms.

GRADE IV. Patient a cardiac cripple, due to severe dyspnoea, angina and/or cardiac failure.

GRADE V. Patient dead.

Follow-up during the first 2 years after discharge was easily achieved in most patients since with 7 exceptions all received long term anticoagulant therapy and returned to the appropriate clinic at about monthly intervals. It was thus possible to see and examine the patients at regular intervals and to reassess the clinical grade. If a patient failed to appear at the clinic as arranged enquiries were made from the relevant general practitioner. Sometimes such enquiries revealed that non-attendance was due to deterioration in condition or to death of the subject.

In those patients who never received anticoagulants, and in those from whom treatment had been withdrawn, the principal source of follow-up was the relevant family doctor. At about 3 monthly intervals a questionnaire (Table 5) was sent to each practitioner. This gave details of each of the 5 clinical grades and the practitioner was requested to indicate which grade was appropriate to the patient's state and to give details of any disability present. Direct comparison of gradings obtained at the anticoagulant clinic and those provided by the family doctors was possible in 15 cases. Often it was known in advance when anticoagulants were to be discontinued and in such cases questionnaires were sent to the practitioners at approximately the same time as a visit to the clinic so

T A B L E 6.

Number of Postmortem examinations on different series.

	Series	No. of Examinations.
STEROID TREATED	A	15
	B	1
	C	5
	D	2
	E	-
	Total - A - E	23
CONTROLS	F	22

that direct comparison would be possible; it was found that the gradings made at the clinic and by the family doctor were almost always the same.

Sometimes a reply from a practitioner indicated the death of a patient. Although the cause was always available, the exact date was seldom known since the appropriate records had usually been returned to the Executive Council. Enquiries were then made from the appropriate Registrar from whom details were obtained in all cases.

Occasionally information was supplied by other departments. Eleven patients were readmitted for treatment of cardiovascular complications. Three were readmitted with further infarcts so severe that they were reincluded in the investigation. Two patients returned to other departments, the eye and dental outpatient clinics, where they were re-assessed. One patient was admitted to Hawkhead Hospital soon after discharge from the Victoria Infirmary, remaining there until he died 10 months later; adequate clinical information was available.

No other patients were admitted to other hospitals after their discharge from the Victoria Infirmary.

POST-MORTEM EXAMINATION.

A total of 88 patients in series A - F inclusive died in hospital; permission for post-mortem examination was requested in all but was granted in 45 only - 23 steroid treated and 22 controls. The distribution of those between the individual treatment groups is shown in Table 6.

The following additional material was examined:-

(1) A series of 19 post-mortems, designated SERIES H to act as controls for the control series.

(2) Histological material from 37 routine autopsies where death was due to myocardial infarction (Series J).

(3) Histological material from 11 autopsies performed in hospitals other than the Victoria Infirmary on patients dying of a myocardial infarct and treated with steroids (Series K).

Fuller details of those three additional groups are:-

(1) SERIES H. This comprised 19 patients admitted during 1961-63 to another medical unit in the Victoria Infirmary because of an acute myocardial infarct of less than 24 hours' duration and treated in a manner identical to that in Series F. All were considered to be severe and autopsy was requested in order that the presence of gross myocardial damage could be confirmed. The coronary prognostic index was not employed routinely in this unit but from the information in the admission note it is apparent that all would have had a high index score.

They constitute a group which can be compared with the main control group (Series F), although some doubt must remain as to their exact severity.

(2) SERIES J. This group constitutes all cases dying of a myocardial infarct and coming to routine post-mortem examination in the Victoria Infirmary during 1958-63, omitting those already included in Series A - F inclusive and in Series H. All cases were excluded in which there was a history of steroid therapy during the preceding year. The 37 cases remaining constitute a very varied group exhibiting all degrees of clinical severity and all received treatment similar to that given to those in the

main control group, Series F. Since the post-mortems were performed by several operators, the naked eye appearances cannot be compared in detail but the blocks of tissue preserved from each case have been processed in an identical manner and the sections thus prepared can be compared one with another and with the sections from cases in other groups.

The histological material obtained from Series F, Series H, and Series J, was examined and each series compared with the other two. In the absence of any significant difference in the repair process in the three groups, it could be deduced that variations in the clinical severity did not influence the histological picture, and therefore the histological aspects of Series F and H could be compared more readily since doubts about the clinical assessment in Series H would be less important.

(3) SERIES K. This comprises histological material from 11 autopsies performed in hospitals other than the Victoria Infirmary. These patients had been admitted to hospital within 24 hours of the onset of infarction and had been assessed by the coronary prognostic index on admission. All had scores of 17 or above and had been treated with hydrocortisone in doses similar to those used in Series A. The hospitals concerned and the number of patients are:-

Dumfries Infirmary.....	2
Gartloch Hospital.....	4
Western Infirmary, Glasgow.....	4
City Hospital, Aberdeen.....	1

This material has been examined to determine if the general appearances are similar to those in Series A; differences in fixation and processing methods would vitiate a closer comparison.

ROUTINE POST-MORTEM TECHNIQUE.

NAKED EYE EXAMINATION OF HEARTS.

The heart and pericardial sac were removed intact and the pericardial sac was opened over a container to collect any fluid present, the nature and volume of which was noted. The intact heart was examined externally and the size of the chambers in relation to one another was noted. The presence and position of any fibrin exudate, congested or haemorrhagic area, aneurysmal bulging or wall rupture was noted. Incisions about 30mm. long were then made (1) through the wall of the right ventricle at a point 25 mm. from the anterior end of the interventricular septum and 30 mm. from the apex, and (2) through the wall of the left ventricle through the mid point of the lateral wall, 30mm. from the apex. If any gross abnormality was noted at those points, e.g., marked thinning or the wall or rupture, the incision was moved to the nearest normal point. The auricles and auricular appendages were opened along their anterior aspects and the heart valves were then examined in detail. The patency was checked by establishing how many fingers could be passed into each valve; the degree of competency was checked by visual inspection of the cusps in situ, and in the case of the aortic and pulmonary valves, by the water test.

Next the ventricles were opened up more fully by extending the earlier incisions upwards through the aortic valve in the case of the left ventricle, and through the pulmonary valve in the case of the right ventricle, passing between cusps in each case. The incisions in the auricles were then extended downwards through the tricuspid and mitral valves, again between cusps to join the earlier incision through the ventricles at a point 15-20mm. below the level of the valves. Each valve was then opened out, and the cusps inspected for

any abnormality.

Thus all four chambers were opened up fully to permit wide inspection and at this time all blood clot was removed from within the chambers and the presence, site and amount of any antemortem mural thrombus was recorded. When all clot was removed the heart was weighed.

The coronary arteries and their principal branches were opened using blunt pointed, coronary artery scissors, and the presence, position and nature of any atheromatous plaques were noted. Particular attention was paid to the degree of narrowing of the vessels and the presence and position of any antemortem thrombi. Where the vessels were too narrow to be opened with scissors, they were cut across by a knife at 5 mm. intervals. The heart was now divided horizontally across both ventricles at the mid point between apex and base, care being taken to ensure that the incision through the left ventricular wall was at right angles to the surface. The thickness of the lateral wall of the left ventricle was measured with dividers at a point on the horizontal incision close to its junction with the original vertical incision; the dividers were measured against a ruler to give the ventricular wall thickness to the nearest millimetre. In virtually all cases a recent infarct was seen at some point along the horizontal incision. The thinnest point of any infarct seen was then measured with dividers.

Further horizontal incisions were then made in strips of 10 mm. from the first horizontal incision until the entire myocardium of both ventricles had been sliced. Any abnormalities in the myocardium, including new and old infarcts and areas of diffuse fibrosis, were examined in detail and the exact position, nature and size of any infarct noted. The thinnest

part of any recent infarct revealed by the further horizontal incisions was measured with dividers as before.

HISTOLOGICAL EXAMINATION OF MYOCARDIUM.

8-15 blocks of myocardium were taken for histological examination, at least six including both infarcted and adjacent apparently normal myocardium. Blocks were also taken of old scars and of any other lesion present.

PROCESSING METHODS.

In 30 cases two blocks were prepared for and cut on a cryostat; all blocks underwent paraffin processing, including those cut earlier on the cryostat. Full details of the processing and section cutting methods used are given in an appendix, (Volume II).

STAINING METHODS.

(a) Cryostat Sections:-

1. Haematoxylin and Eosin.
2. Oil red O.
3. Sudan Black B.
4. Gomori method for acid phosphotase.

(b) Paraffin Sections:-

1. Haematoxylin and Eosin.
2. Masson Goldner Trichrome.
3. Prussian Blue (Perls' reaction).
4. Reticulin Impregnation (Lendrum's picric acid modification of Gordon and Sweet method).
5. Periodic Acid-Schiff (P.A.S.)
6. P.A.S. after diastase.
7. P.A.S. after hyaluronidase.
8. P.A.S. - Alcian blue.

(b) Paraffin Sections, (Continued).

9. Alcian blue (Johnson 1960).
10. Alcian green (Putt and Mukill, 1962)
11. Sulphation Induced Metachromasia (Schrauth 1932)
12. Tolvidine blue metachromasin (Moore, Schoenborg and Koletsky, 1963).
13. Methylene blue metachromasia (Pearce, 1960).
14. Martius Scarlet Blue (Lendrum et al 1962).
15. Fuchsin Miller, (Lendrum et al 1962).
16. Masson 44/41. (Lendrum et al 1962).
17. Feulgen Reaction.

Those methods were employed for the following reasons:-

(1) Cryostat Sections. Sections from each block were stained by haematoxylin and eosin to reveal the general pattern of the tissues and con-:firm the presence of an infarct. Oli Red O and Sudan Black showed any fat or fatty change. Attempts to demonstrate acid phosphatase were limited to those cases where tissue could be obtained within 6 hours of death and so were made in only 5 cases, 3 steroid treated and 2 controls.

(2) Paraffin Sections: The general layout of the haematoxylin and eosin was investigated by Masson Goldner trichrome methods. In these sections the process of the formation of any cellular infiltrate and the gradual develop-:ment of scar formation in the infarct were studied. In addition H. & E. sections were used in a detailed study of the number and types of cells present within the infarct (method: later in this chapter). Prussian blue preparations were used to demonstrate haemosiderin within macrophages in the infarct; this was of importance in the countinf od the cell population.

Reticulin impregnation cast further light on the repair purposes in the infarct, and differences in the quantity and quality of reticulin fibres in the various treatment groups were studied.

Around the margin of many infarcts, lying between the surviving apparently normal myocardial fibres, were large amounts of a finely granular basophilic material which sometimes resembled fibrin. Its nature was investigated using the methods listed as Nos. 5-17 inclusive.

DETAILED EXAMINATION OF THE NUMBER AND TYPE OF CELLS PRESENT WITHIN THE INFARCTS.

Histological examination showed that several well defined stages could be seen in the process of repair of the infarcts. These are:-

(1) Formation of an inflammatory infiltrate.
(2) Phagocytosis of dead cells.
(3) Replacement of infarcted areas by vascular cellular fibrous tissue.
(4) Conversion of this fibrous tissue into a dense collagenous scar.

In order that any differences in this pattern between the various groups of cases might be elucidated an estimation of the number of cells within the infarcts was made by the following method:-

Equipment used and its calibration:

Sections of myocardium 6μ thick were stained by haematoxylin and eosin. These were then examined through a monocular "Bactil" (Watson & Co.) microscope employing a X8 eyepiece and a 4 mm. objective. The eyepiece was fitted with a graticule ruled in squares with sides measuring 2 mm. to superimpose a grid of small squares on the section. To establish the size of each square of tissue thus delineated, a slide micrometer was used and the tube length of microscope was adjusted so that one slide micrometer division (0.1 mm.) coincided with the combined length of the sides of three of the

superimposed small squares. This appropriate tube length was 171 mm. and the magnification x 342.

By this means it was established that with a tube length of 171 mm. and the lens system detailed, the area of section enclosed by 9 graticule squares was 0.01 sq. mm. It was decided that counts would be taken over an area of infarct measuring 2.0 sq. mm., i.e. an area enclosed by 200 x 9 squares = 1800 squares. It will be appreciated that 2.0 sq. mm. of section did not represent 2.0 sq. mm. of actual infarct since the processing methods used resulted in shrinkage of the tissue. Since all blocks (other than those from Series K) were processed by the same technique the degree of shrinkage was considered to be fairly uniform so that counts taken over a fixed area were regarded as comparable. Counts were made on material from Series K but owing to possible differences in processing, direct comparison of the results with those from the other cases was not made.

Method of Counting:

A rectangular block of squares in the centre of the field was used comprising 7 rows each of 8 squares, i.e. 56 squares in all. Thus to cover a total area of 2.0 sq. mm. if was necessary to count the cells within 32 of the 56 square units plus a single unit of 8 squares; the top row was always taken.

In the course of the examination it was noted that the number of cells of all types within an infarct varied with the position of the area examined. Generally, a narrow zone at the margin was the most cellular. Also the inflammatory cells invading the infarct during the initial few days appeared to spread inwards from the margins and thus a new type of cell could

be identified in the more peripheral zone before this type reached the central area. Later, when the infarct had become replaced by vascular fibrous tissue, this migration was much less in evidence. These observations indicated that the detailed counting should be undertaken at a position constant to the margin of the infarct. Initially it was decided to exclude a zone of infarct 0.1 mm. thick and to conduct the count in the area immediately deep to this. In theory a strip 7 graticule squares wide should have been counted but in practice marked variations in the contour of the infarct frequently made it impossible to obtain 56 squares composed entirely of infarct and thus the count was extended to a zone of up to 0.5 mm. in width.

CELL TYPES COUNTED:

The following cell types were counted and the total number of each recorded (1) neutrophils, (2) lymphocytes, (3) eosinophils, (4) plasma cells, (5) macrophages with no obvious haemosiderin, (6) macrophages containing haemosiderin, (7) fibroblasts (nuclei). The separate counting of haemosiderin laden macrophages (6) requires explanation. The total number would be dependent on the amount of haemorrhage which had taken place earlier into the infarct and the separate count made should show any differences in the amount of haemosiderin, and therefore in presumed earlier haemorrhage, between steroid treated and non steroid treated groups. It would also show if the process of repair was in any way disturbed where large amounts of haemosiderin were deposited.

The count of fibroblasts was affected by their loss of nuclei as they became converted into collagen. It was decided therefore to count nuclei specifically so that a rising count would be obtained in the earlier

stages of resolution and a falling count as collagen was formed.

SUMMARY OF CHAPTER TWO.

Patients with a very recent myocardial infarct were assessed by the Coronary Prognostic Index and those with a score of 17 or over were admitted to the investigation. 74 were treated with steroids and 69 received routine therapy only and acted as controls. Clinical and laboratory procedures have been detailed.

In fatal cases autopsy examination was undertaken when permitted and the methods adopted for naked eye and microscopic investigations have been described. Two additional control groups of autopsies have been included to test the validity of the control group.

C H A P T E R T H R E E .

RESULTS BASED ON CORONARY PROGNOSTIC INDEX.

(i) Mortality Rates : Effect of treatment.

(ii) Effect of Steroids on death rates related to individual prognostic index components.

(a) Age and Sex.

(b) Previous history.

(c) Shock : Study of serial systolic blood pressure readings.

(d) Failure.

(e) E. C. G. Changes.

(d) Rhythm: Detailed study of effect of steroids on various arrythmias and on heart block.

TABLE 7.

Hospital Discharge and Death Rates.

	Series	Discharged from Hospital Number	%	Died in Hospital Number	%
	A	19	37	30	63
Steroid	B	2	50	2	50
	C	7	58	5	42
Treated	D	1	20	4	80
	E	3	73	1	25
Total A - E		32	44	42	56
Controls	F	22	32	47	68

FIG. 2.

DEATH RATE OF HOSPITAL PATIENTS AT VARIOUS TIMES AFTER INFARCTION.

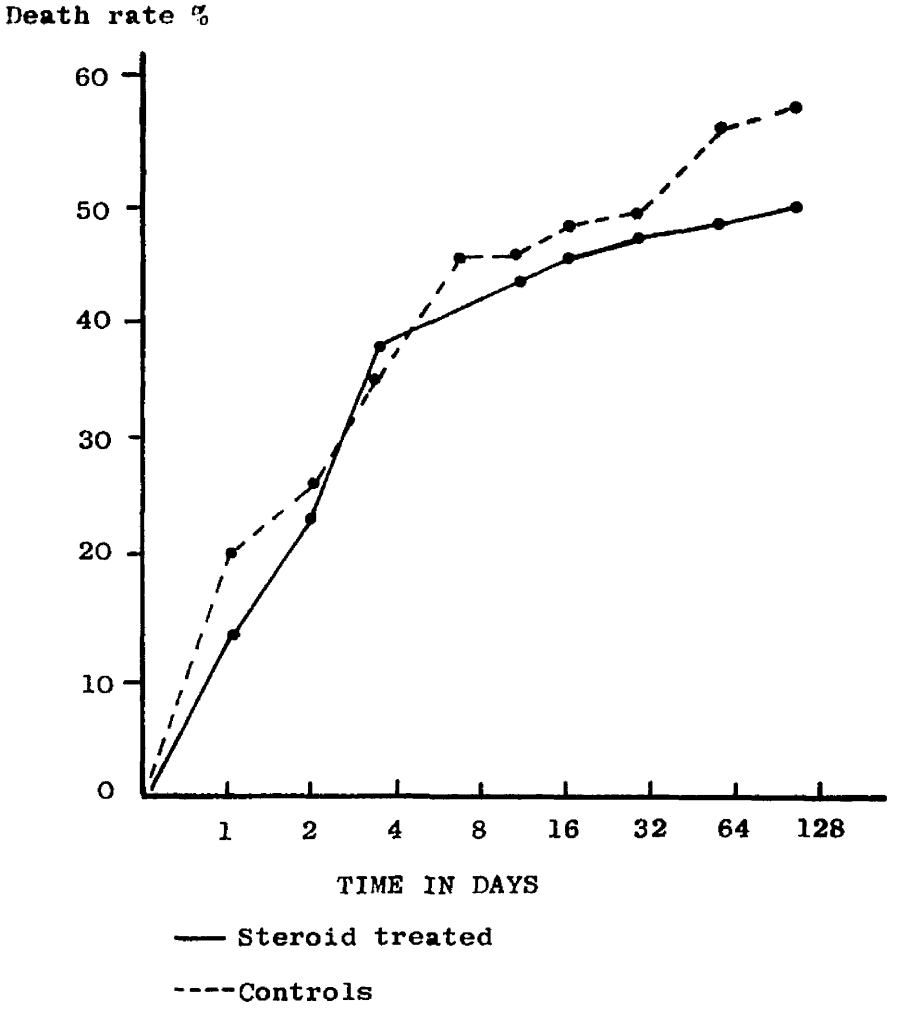
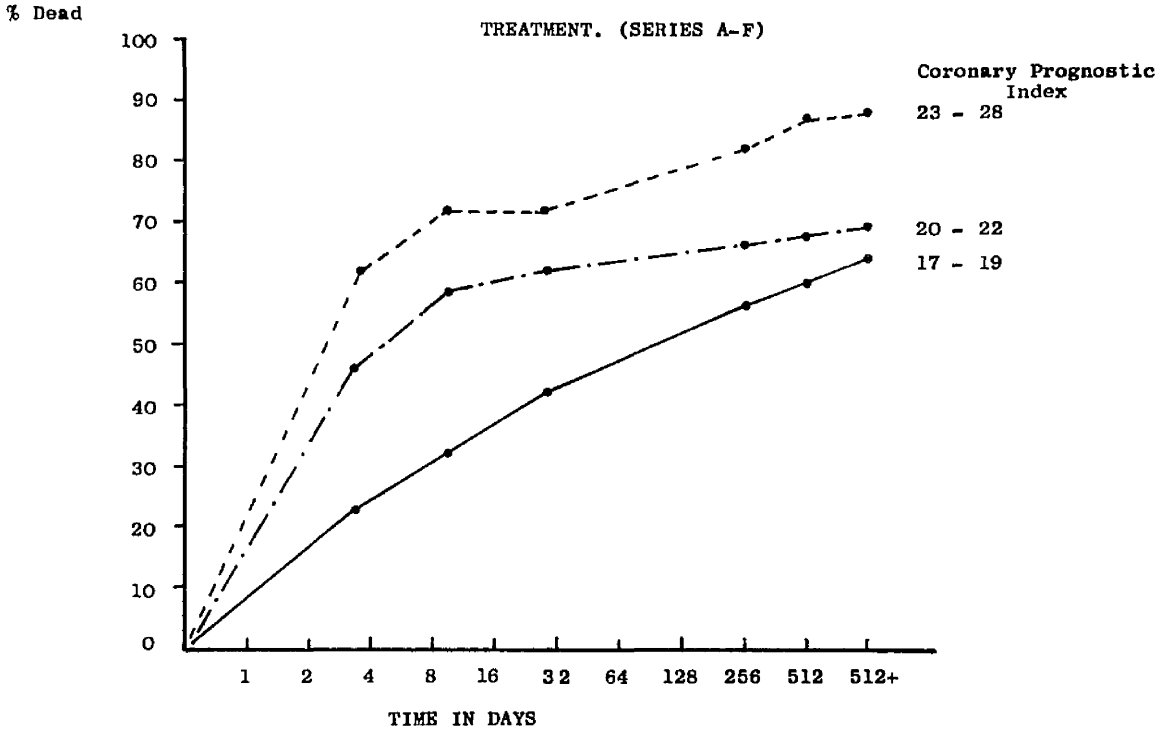


FIG. 3.

RELATIONSHIP BETWEEN PROGNOSTIC INDEX SCORE AND DEATH RATE AT VARIOUS TIMES AFTER INFARCTION REGARDLESS OF TREATMENT. (SERIES A-F)



RESULTS BASED ON PROGNOSTIC INDEX.

MORTALITY RATES.

Of the 143 patients comprising Series A-F inclusive, 89 (62%) died in hospital; 54 (38%) recovered sufficiently to be discharged home and of those 15 (11%) died subsequently. Although the survival rate is slightly higher in the steroid treated patients (Table 7), the difference is not significant at only 1½ times the standard error (8.06%). More than half the deaths occurred during the first three days and the mortality rates in this period were not affected by steroids, (Fig. 2). Of those patients who died after discharge 9 (28%) were steroid treated and 6 (27%) were controls. The follow-up of patients is discussed more fully in Chapter 7.

RELATIONSHIP BETWEEN DEATH RATE AND PROGNOSTIC INDEX SCORES.

The prognostic index is claimed to give a reliable guide to the prognosis at 28 days, and a rough guide to the longer term outlook (Peel et al 1962). Study of (a) the patients included in this study (Fig. 3) and (b) all admissions to Dr. Peel's Unit with a myocardial infarct during 1961-3 inclusive (Table 37, Vol. II) confirms that higher scores are associated with higher death rates. This association was more obvious at periods of up to 28 days after infarction than in later periods (Fig. 3). In cases with a relatively lower score, (17-19) three-quarters survived the first few days while most of those with higher scores, (20 and above) died during this period. By the end of one year about two-thirds of the lower score groups had died while

TABLE 8.

Time (In Days) Between Infarction and Death in Hospital Related to Coronary Prognostic Index.

Index Score	17 - 19	20 - 28
Steroid Treated	11.6	3.4
Controls	16.3	4.7

TABLE 9.

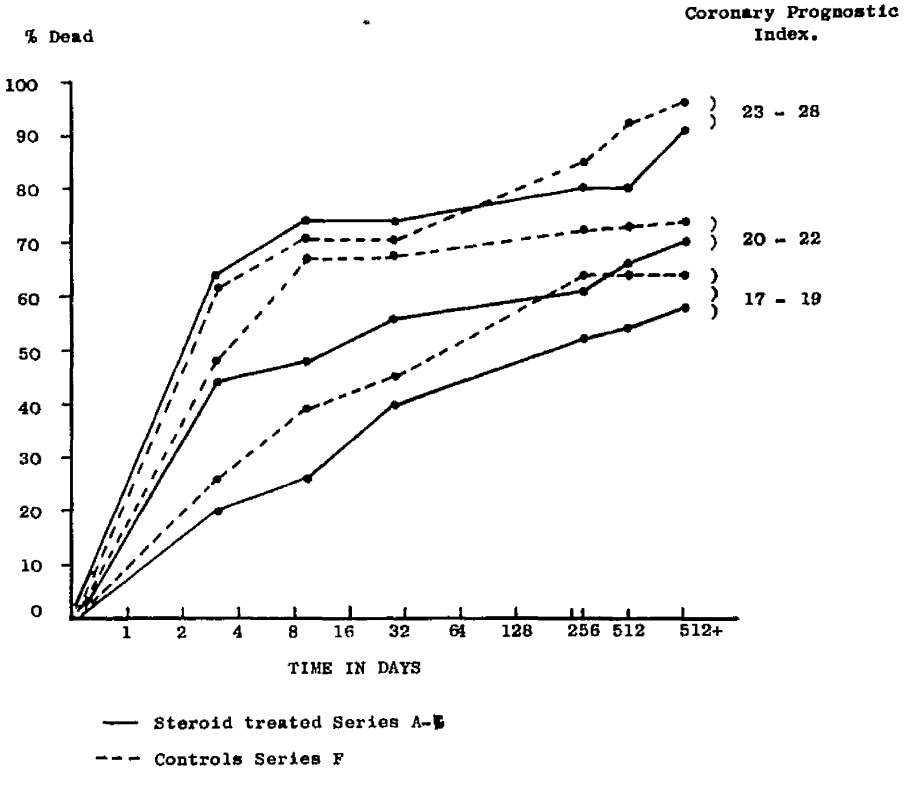
Mode of Death of Patients Dying in Hospital

Mode of Death	Steroid Treated Number of Patients.	Controls Number of Patients.
Speedy Deterioration After Admission	8	12
Never Satisfactory but Survived First Few Hours	15	14
Unremitting Congestive Failure	14	13
"Sudden" Death Cause Not Certain	2	5
Associated With Onset of Arrhythmia	2	2
Cerebral Embolus	1	2

FIG. 4.

EFFECT OF STEROID THERAPY ON DEATH RATES

AT VARIOUS TIMES AFTER INFARCTION.



in the higher score group there were few additional deaths; the difference between the lower and higher score groups thus became narrowed. This point can be amplified by comparing the time interval between infarction and death in patients with scores of 17-19, and 20-28; regardless of treatment those with the lower scores survived longer than those with higher scores (Table 8).

The cause of death of all those dying in hospital was related to the cardiac condition in all cases. Most were either hypotensive throughout the admission or were in unremitting congestive failure. The mode of death shows a similar pattern irrespective of therapy. (Table 9).

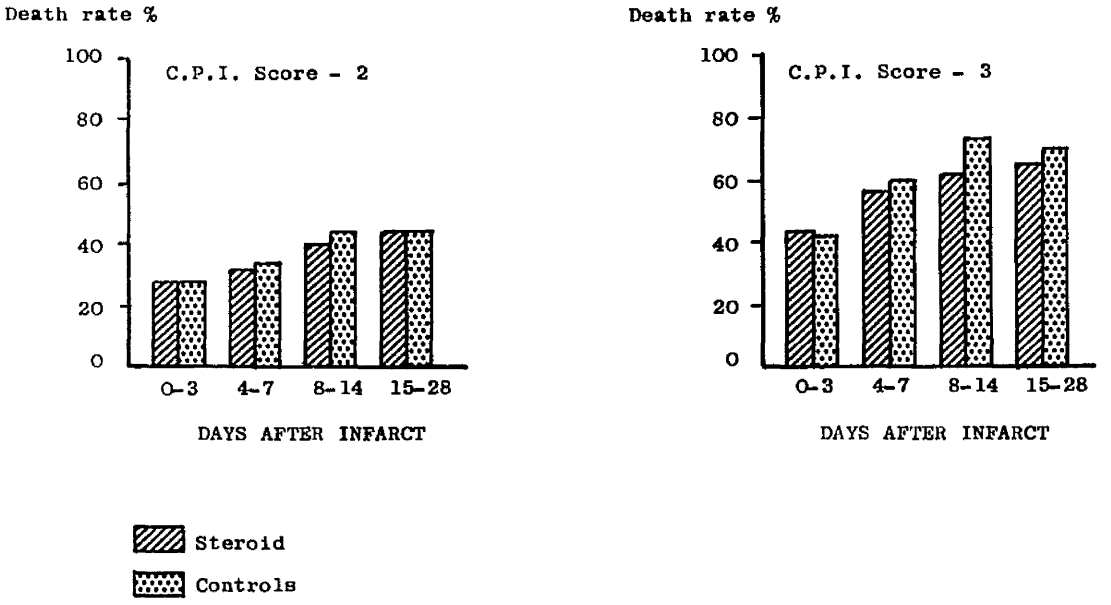
EFFECT OF STEROIDS ON DEATH RATE:

Comparing the steroid treated and control patients, steroids had a slight but not significant advantage where the prognostic index was 22 or less, but in patients scoring 23-28 there was the same high mortality rate irrespective of treatment, (Fig. 4). Where the index was 17-19 the mortality rate in the steroid group was 8 - 10% less than in the controls (Standard error of the difference = 11.8%). With scores of 20-22 the difference is greater (18.2% at 7 days) but is still not statistically significant (standard error of the difference = 14%). Most of the patients with the highest score (23-28) were admitted in a moribund state and died so rapidly that no form of treatment could have had time to be effective. Most of these patients were considered to have suffered a very large infarct, often superimposed on a previous infarct, and it was assumed that so little viable myocardium remained that no form of therapy could be expected to be of lasting value. Post-mortem examination of certain of those patients confirmed this view.

These results indicate that the death rate in the steroid group is not significantly less than in the controls, (Observed difference 12½%, Standard

FIG. 5.

MORTALITY RATES (%) AT VARIOUS TIMES AFTER INFARCTION RELATED TO CERTAIN C.P.I. SCORES FOR SEX AND AGE.



error of difference 9%).

EFFECT OF STEROIDS ON DEATH RATES RELATED TO PROGNOSTIC INDEX COMPONENTS.

The six factors comprising the index were examined individually to see whether any difference in the mortality rates between steroid treated and control patients could be related to any particular factor. The factors are considered in this order: (1) Age and Sex, (2) Past History, (3) Shock, (4) Cardiac Failure, (5) Electrocardiographic changes, (6) Rhythm. The distribution of scores within each factor is shown in Tables 38-43 (Vol. II). The mortality rates at various times after infarction in steroid treated and control patients, related to certain prognostic index scores for each factor is shown in Figs. 5, 6, 7, 11, 12, 13. Initial examination suggests that:-

(1) Mortality rates rise with the passage of time and increase with increasing prognostic scores.

(2) The mortality rate is lower in steroid treated than in control cases when related to scores for (a) past history, (b) shock, and (c) electrocardiographic changes.

(3) The mortality rates in steroid and in control cases when related to scores for (a) age and sex, (b) failure, and (c) rhythm, show no difference referable to treatment.

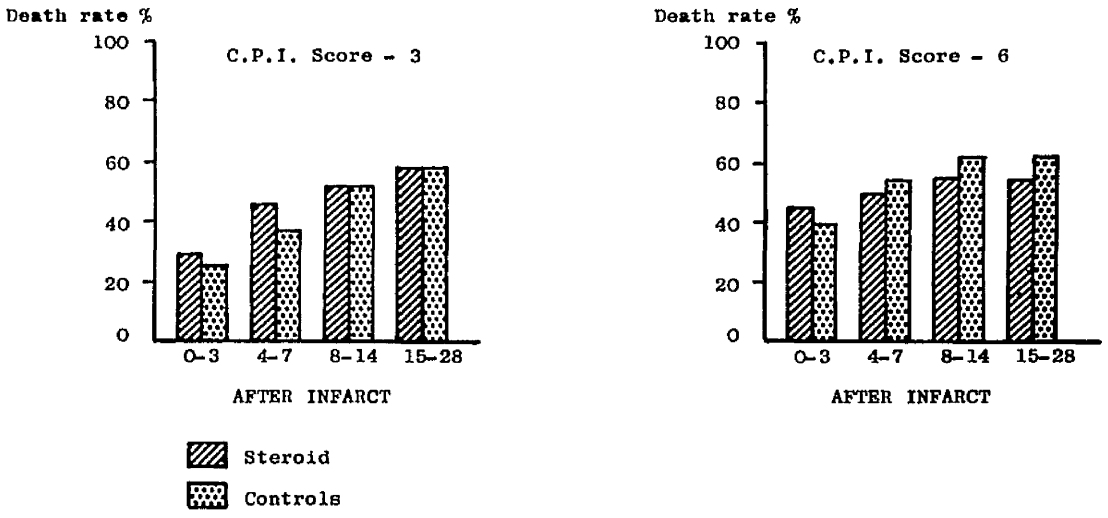
In the following sections each factor is re-examined and in certain cases is investigated in additional ways:-

AGE and SEX.

Few patients scored 0 or 1 and therefore only the mortality rates of those scoring 2 or 3 are suitable for comparison. There is no appreciable difference between steroid treated and control patients, older patients showing a higher mortality rate regardless of treatment (Fig. 5). This agrees

FIG. 6.

MORTALITY RATES (%) AT VARIOUS TIMES AFTER INFARCTION RELATED TO CERTAIN PROGNOSTIC INDEX SCORES FOR PREVIOUS HISTORY.



with the findings of Master et al (1939), Baer and Frankal (1944), Jacobs (1951), Sigler (1951), Barr (1955), and Pool (1955), all of whom have pointed out that after a myocardial infarct, older patients have a poorer prognosis.

2) PREVIOUS HISTORY.

Only with scores of 3 or 6 were there sufficient cases to permit of comparison (Fig. 6). Patients with a score of 3, i.e. those with a history of previous cardiovascular disease but NOT a previous infarct, showed similar mortality rates irrespective of treatment. Those scoring 6, i.e. those with a history of a previous myocardial infarct, showed a slight difference in favour of steroid treatment but this was not significant (Observed difference 8%: Standard error of the means (13%).

3) SHOCK.

The assessment of the degree of shock is subjective and is an expression of the opinion of the individual clinician. The other factors in the prognostic index have definite criteria, and although the opinion of the examining clinician is important in, for instance, the assessment of electrocardiograph tracings showing equivocal changes, in this study usually little doubt existed as to the appropriate score. In the assessment of shock, however, there is no simple answer as to how long shock must persist after sedation before it becomes "unremitting", or how to differentiate between mild shock which will prove to be transient, or moderate shock which will be relieved by rest and sedation. Furthermore, the assessment of shock was often made by consultant staff during the daytime (i.e. on steroid treated patients) and almost always by junior staff at night, (control cases). Although the mortality rate, when related to these scores, was advantageous

FIG. 7.

MORTALITY RATES (%) AT VARIOUS TIMES AFTER INFARCTION

RELATED TO PROGNOSTIC INDEX SCORES FOR SHOCK.

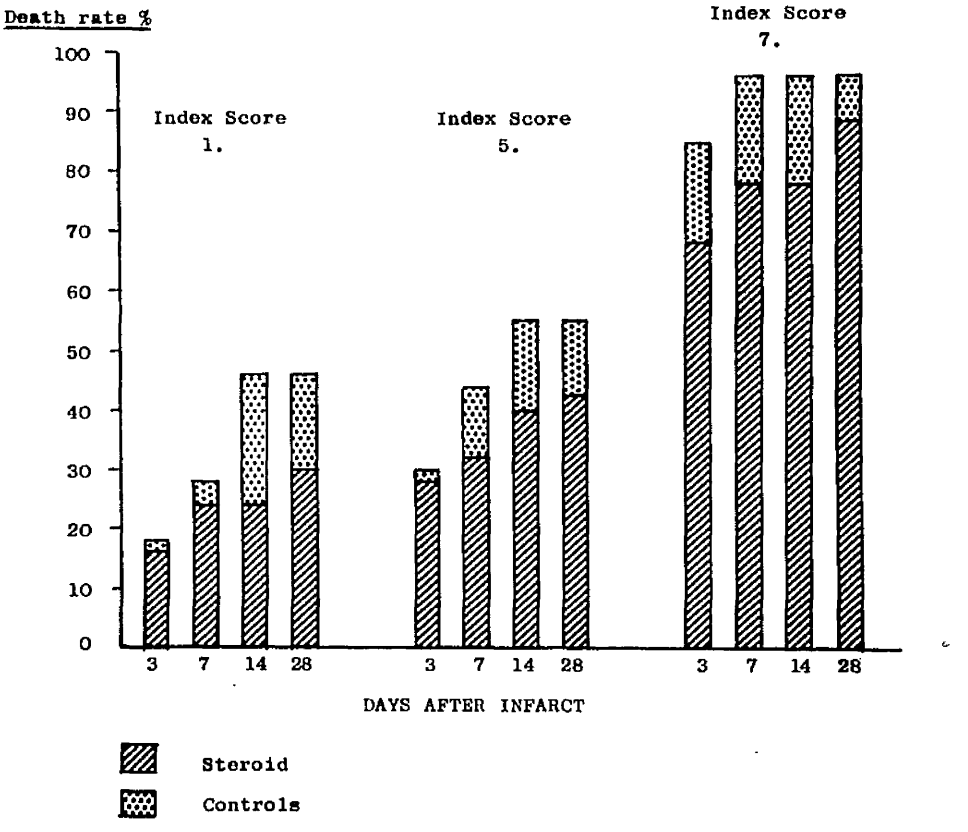


FIG. 8.

DISTRIBUTION OF SYSTOLIC BLOOD PRESSURES ON ADMISSION.

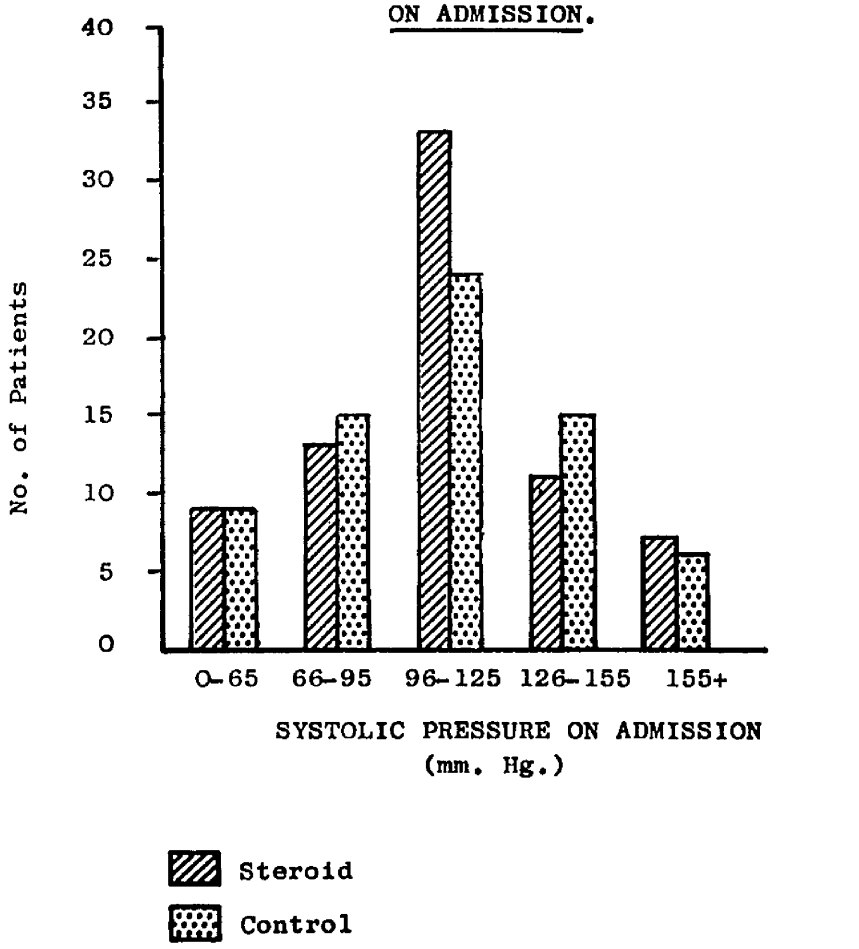
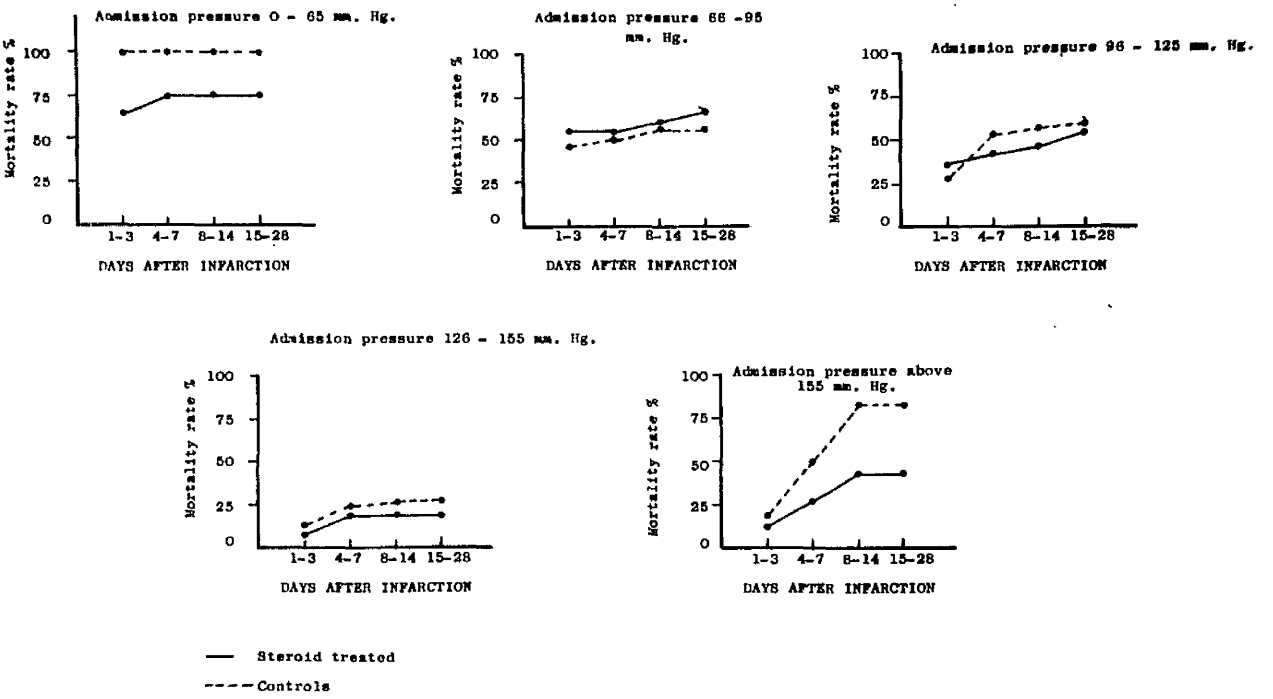


FIG. 9.

MORTALITY RATES (%) DURING FIRST MONTH RELATED TO SYSTOLIC PRESSURE ON ADMISSION.



to steroids by as much as 20% (Fig. 7), it cannot be accepted without closer examination, especially when it is remembered that the actual death rates in the two groups during the first three days were identical, (Fig. 2).

Serial systolic blood pressure readings were analysed to assess the degree of initial shock and rate of recovery, the distribution of admission pressures is given in Fig. 8. Irrespective of treatment the lowest mortality rate was seen in those who were normotensive (125-155 mm. Hg. systolic) and higher death rates were found in those with higher or lower pressures on admission. Comparing the steroid treated groups with the controls, steroids favoured those with pressures of below 66 mm. Hg. (Fig. 9); the difference in the mortality rates is significant at 2.6 times the standard error of the difference. With patients admitted with a systolic pressure of above 155 mm. Hg., there is an apparent advantage with steroids but this is not significant (Standard error is 1.5 times the standard error of the difference). Similarly there is no significant difference with treatment in the immediate mortality rates in those with admission pressures of 66-155 mm. Hg. In some cases the mortality rates showed an apparent advantage to steroids after 7 days but this cannot be related directly to the degree of initial shock. Experience has shown that if recovery from shock is not complete in the first few days then death is likely to occur within the following few days. On the other hand if recovery is complete it seems unlikely that the initial degree of shock can then influence the prognosis.

As a measure of the recovery from shock the average alteration of systolic blood pressure between the first reading after admission and that at the end of the third day has been noted. Irrespective of the initial

FIG. 10.

AVERAGE CHANGE IN SYSTOLIC BLOOD PRESSURE

BETWEEN ADMISSION AND END OF 3rd. DAY

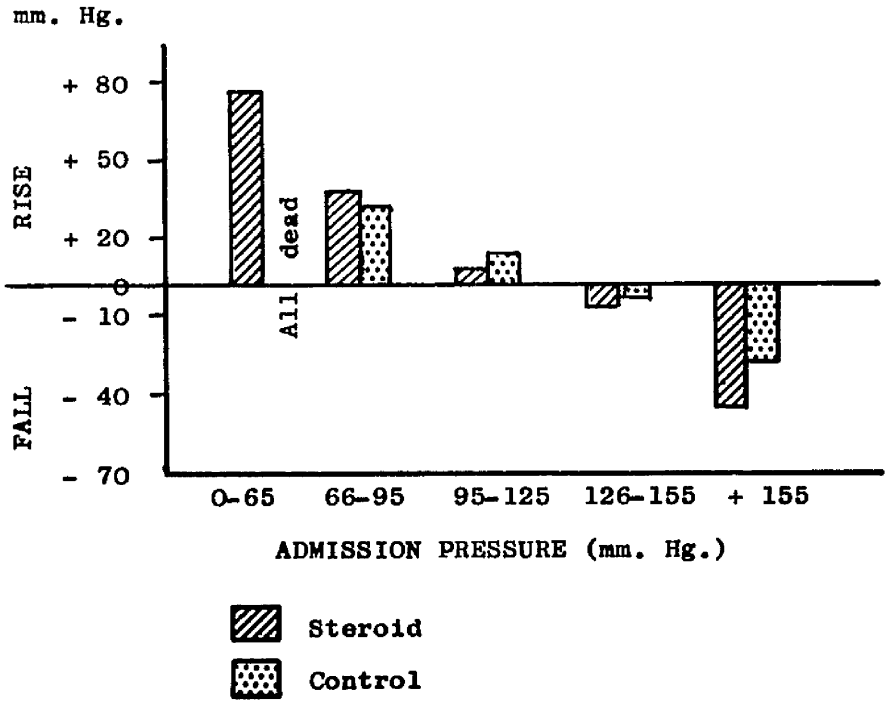


TABLE 10.

Change in average systolic pressure from admission to within 4 - 6 hours of death where death occurred in first three days.

Series	No. of Patients	Initial Pressure (mm Hg)	Pressure before death (mm Hg)	% Fall
Steroid Treated	27	87	56	35
Control	26	82	52	36

pressure and of treatment, the blood pressure tended to stabilise around 110-130 mm. Hg. (Fig. 10). The only exception was where the initial pressure was under 66 mm. Hg; the pressure rose to about 120 mm. Hg. in the steroid treated group (9 patients) but all the controls died, (9 patients).

Study of the systolic pressures in those patients dying during the first three days showed that steroid treatment made no difference. In the majority pressure readings were recorded hourly, and in all at least one reading was available during the 4-6 hours period prior to death. Readings nearer to death were disregarded since they often showed a very sharp fall as a terminal event. By 4-6 hours before death the pressure showed an average fall to about 60% of the admission level, regardless of treatment. (Table 10).

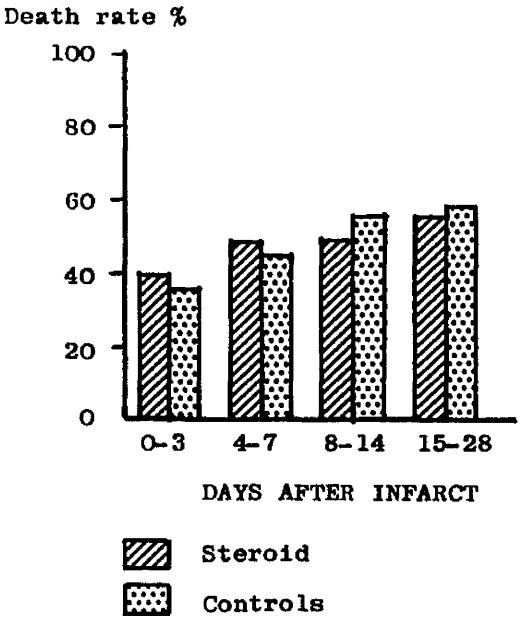
Although the mortality rates related to the prognostic scores for shock showed an advantage with steroids, particularly where there was slight or moderate shock (Fig. 7), examination of several blood pressure readings reveals that, in fact, there was an advantage only in severely shocked patients.

) FAILURE.

The prognostic significance of cardiac failure in cases of recent myocardial infarction has long been recognised. Master, Dack and Jaffe (1937) pointed out that failure was associated with no less than 72.5% of the fatal cases. Chambers (1946) found that failure was present in 48% of all admissions with a recent infarct while Billings et al (1949) found that 52% of patients showing basal rales or heptomegaly on admission died in hospital. A theoretical danger in the use of steroids in patients with congestive failure is that there might be retention of sodium and chloride thus aggravating fluid retention (Sprague et al, 1950, Luft and Sjorgan, 1951:

FIG. 11.

MORTALITY RATES AT VARIOUS TIMES AFTER INFARCTION RELATED TO THE PROGNOSTIC INDEX SCORE OF 4 FOR FAILURE.



Sjorgan, 1952). On the other hand Camara and Schemm (1951), and Gutner et al (1957) have shown that in the presence of congestive failure steroids in fact produce a salt and water diuresis.

In the present study most patients showed some degree of failure on admission, the majority scoring 4 points while the number scoring 0 or 1 is too small to permit comparison. Study of the mortality rates indicates that more than half of those scoring 4 points died during the first month irrespective of treatment (Fig. 11), a result which is closely similar to that of Billings et al (1949). All patients with evidence of failure were treated with mersalyl or chlorothiazide with digoxin also in many cases. As such treatment is potent against congestive failure any minor effect of steroid therapy would be masked. Large doses of steroids were given for only the first few days, the doses thereafter being so small that any adverse effect on the congestive failure would be unlikely.

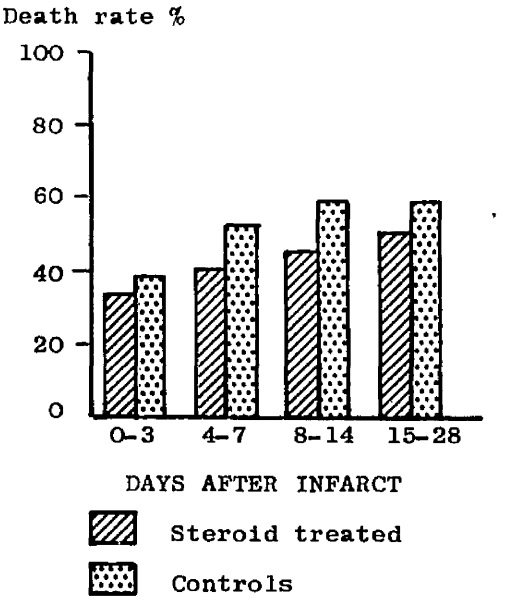
Steroid treatment did not influence the later appearance of failure in those patients with no failure on admission. Three steroid treated patients (Nos. A3, A12, and A23), went into failure after 5, 10 and 2 days, all dying 1-2 days later. Two controls (F25 and F41) went into failure on the second day and died later on the same day.

) ELECTROCARDIOGRAPHIC CHANGES.

Electrocardiographic tracings were available in all but 9 patients, 4 steroid treated and 5 controls, all of whom died before a useful tracing could be obtained. In the remaining 134 cases adequate tracings were obtained on at least one occasion and in all but two (Cases A3 and F25) the first tracing revealed a definite infarct pattern, usually involving the Q S waves. In the two exceptions persistent auricular fibrillation masked the infarct

FIG. 12.

MORTALITY RATES AT VARIOUS TIMES AFTER INFARCTION RELATED TO THE PROGNOSTIC INDEX SCORE OF 4 FOR E.C.G. CHANGES.



pattern initially although a typical Q S infarct pattern was evident in tracings made later on the first day. Only cases scoring 4 points (Q S pattern) are sufficiently numerous to permit comparison. The mortality rates show a slight advantage with steroids (Fig. 12) but this is not significant since the difference is only 0.8 of the standard error of the means.

The predominance of cases showing Q S changes is not surprising since all cases in this study were clinically severe. Q S wave changes are usually associated with the largest, most severe transmural infarcts (Peel 1955), while Q R waves are thought to be associated with smaller transmural lesions, and T waves with infarcts affecting only part of the thickness of the wall (Peel et al 1962).

Changes in the initial infarct pattern were observed in several cases, and during the second week some improvements in the basic pattern were seen in both steroid treated and control patients. Two patients showing initial Q R patterns later developed Q S changes (Cases A30 and E1, 5 and 4 days after admission respectively), thus indicating a possible flaw in the scoring system. It is well recognised that the initial cardiographs may not show the full extent of an infarct pattern, and days or even weeks may pass before these changes become evident. In this study, however, most cases showed initial abnormalities justifying a maximum score. In a group of patients with less severe infarcts, however, assessment based on the initial E.C.G. appearance could be a significant source of error.

5) RHYTHM.

Disorders of rhythm, including upsets in atrioventricular conduction were seen in a high proportion of cases. Where rhythm and conduction were normal the mortality rates for steroid treated and control patients were

FIG. 13.

MORTALITY RATES AT VARIOUS TIMES AFTER INFARCTION RELATED TO CORONARY PROGNOSTIC INDEX SCORES FOR RYTHM.

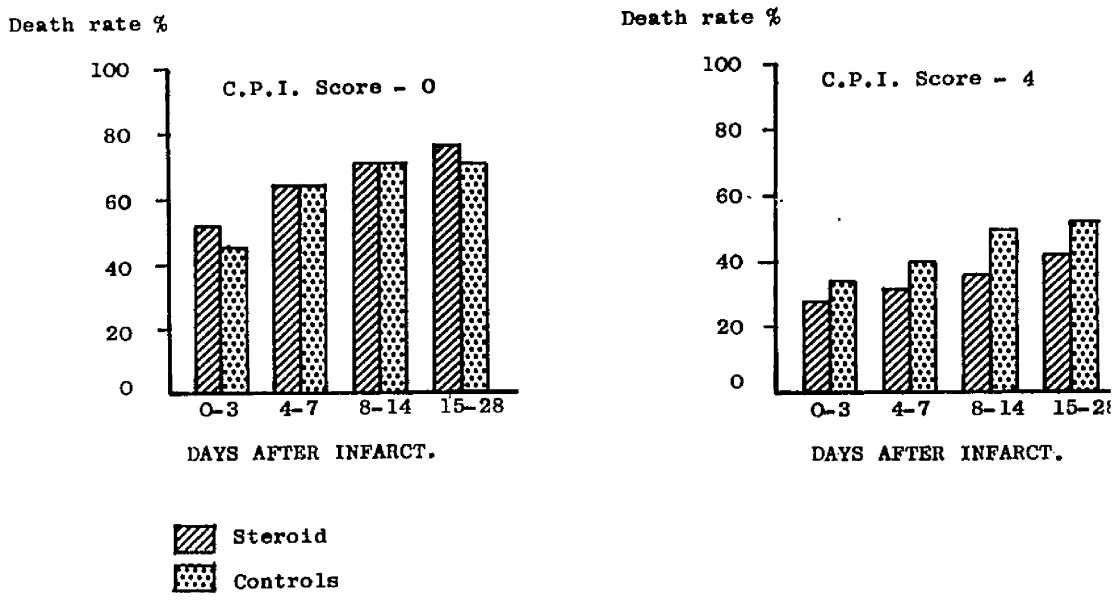


TABLE 11.

Incidence of Arrythmias and Conduction Defects.

	No. of Patients	
	Steroid Treated	Controls
Arrythmias - All Cases	38	25
Sinus Tachycardia	9	10
Auricular fibrillation	9	7
Ventricular Extrasystoles	12	5
Other arrythmias	8	3
Conduction Defects - All Cases	18	15
Potential Block	6	5
Partial Block	4	5
Complete Block	8	5

TABLE 12.

Mortality rate (%) in patients showing any arrythmia on admission, at various times after infarction.

	Mortality Rate (%)			
Days after Infarction	3	7	14	28
Steroid Treated	34	42	47	53
Controls	33	38	46	50

similar. Where an abnormality of rhythm or conduction was present the mortality rate was slightly lower in the steroid group (Fig. 13). Little information is available in the literature regarding the effect of steroids on the arrythmias but the efficacy of steroid therapy in the treatment of conduction defects associated with a recent infarct has been reported by many observers (Prinzmetal and Kennamar, 1954 and 1960; Phelps and Lindsay, 1957; Fiegel, 1958; Kaiser, 1960: Friedberg et al 1960; Caramelli and Tellini, 1960; Aber and Wyn Jones, 1960; Pay and Viscount Waverley, 1961; Dall and Peel, 1962; Broustet and Renner, 1963a, 1963b; Grieco and Andreae, 1963).

Cases scoring 4 because of an abnormality of rhythm or conduction have been separated into those showing (a) an arrythmia on admission, and (b) a conduction defect on admission, and the effect of steroid therapy has been studied. The distribution of arrythmia and conduction defects is shown in Table 11.

(a) ARRYTHMIAS.

Throughout the first month after infarction, the percentage mortality rate in patients showing any arrythmia on admission was closely similar in the steroid treated and control patients, (Table 12). The commonest arrythmias were ventricular extrasystoles, sinus and tachcardia and auricular fibrillation. These three types are considered separately, and the other arrythmias, seen only infrequently have been taken as a fourth group.

(i) VENTRICULAR EXTRASYSTOLES:

These were seen on admission in 12 steroid treated and 5 control patients and were associated with death during the first few days in about half, irrespective of treatment. No significant difference in mortality

rate was evident up to the end of the first month after infarction. Of the 12 steroid cases ventricular extrasystoles persisted until death in seven, four dying on the first day, one on the second day and two on the third. In the five survivors the arrythmia subsided spontaneously in two during the first day, while in one case it persisted for five days before normal rhythm reappeared. In a further case the extrasystoles persisted for three days despite the administration of quinidine sulphate but they ceased early on the fourth day. In the remaining case extrasystoles occurred intermittently throughout the period of admission despite the repeated administration of procaine amide, and they were still present nine months after infarction.

Of the five controls showing ventricular extrasystoles on admission, the arrythmia persisted until death in three, death occurring on the first day in two and the fourth day in the third. In a fourth patient normal rhythm returned spontaneously on the second day but death occurred on the eighth day following the onset of congestive failure. In the fifth patient the arrythmia persisted for five days and subsequently recurred intermittently, being present 15 months after infarction.

(ii) SINUS TACHYCARDIA.

Sinus tachycardia, defined as a pulse rate of over 120/minute in the presence of sinus rhythm and the absence of other arrythmias, was present on admission in nine steroid patients, and in ten controls; only two survived to be discharged from hospital. During the first week approximately half died irrespective of treatment but in the subsequent three weeks a greater, though not significant, proportion of steroid cases died. Sinus tachycardia persisted until death in five steroid cases, death occurring on the first, third (two cases), fourth and sixth days. In two other cases the tachycardia

subsided spontaneously in three and five days, but these patients both died in congestive failure after 22 and 16 days respectively. In the two survivors the tachycardia had subsided spontaneously after one and three days.

In four of the controls the tachycardia persisted until death, which occurred on the first and second days (two each day). In a further case normal rhythm was restored spontaneously on the second day but this patient died in congestive failure on the eighth day. One patient exhibited intermittent tachycardia throughout the admission and died in failure after 36 days. In the remaining four patients the arrythmia subsided spontaneously on the second day and all recovered sufficiently to be discharged.

(iii) AURICULAR FIBRILLATION.

This occurred in nine steroid treated and seven control patients, with a death rate of about 25% during the first month, regardless of treatment. By the end of the first year, however, nearly three-quarters of the controls had died compared with one third of the steroid group.

The three deaths in the steroid group occurred at one day (shock), eleven days (cerebral embolism) and 39 days (congestive failure) after infarction; in all three fibrillation had been present for most of the admission period. In the six survivors, fibrillation disappeared spontaneously in three soon after admission and had not recurred at 15, 24 and 24 months respectively. In three others fibrillation recurred intermittently throughout the admission period despite digitalisation and in one the use of a defibrillator. Eighteen months after infarction one case still had fibrillation, while in the other two normal rhythm was restored spontaneously

two months after infarction.

Of the seven controls only two survived. Fibrillation persisted until death in three; two did not recover from the initial shock and died on the second and third day but the third lived 93 days before dying in congestive failure. One patient showed fibrillation for the first four days and sinus rhythm was restored after the administration of quinidine sulphate; although the rhythm remained normal thereafter the patient died in congestive failure after 29 days. Three patients recovered sufficiently to be discharged but only one remained well. In one, fibrillation persisted for several weeks after infarction and then recurred intermittently, the patients dying at 14 months in congestive failure. A further patient showed persistent fibrillation throughout the admission and the arrythmia was still present 15 months after infarction. In the last case normal rhythm was regained on the second day and the patient remained well two years later.

(iv) MISCELLANEOUS GROUP.

In this group of 8 steroid treated and four control patients, the mortality rates showed no significant difference. The steroid cases comprised four of sinus tachycardia, two of supraventricular tachycardia and one each of auricular extrasystoles and auricular flutter. The controls included one example each of sinus bradycardia, supraventricular tachycardia and auricular extrasystoles.

Of the steroid cases with sinus bradycardia, two died one and three days after admission without improvement in the arrythmia, but in the other two normal rhythm returned spontaneously on the second day and both were eventually discharged from hospital. In one patient with supraventricular tachycardia, the arrythmia persisted for three days alternating at times with

auricular flutter despite the administration of procaine amide, digoxin and mersalyl, and the patient died in congestive failure 39 days after the infarct. Sinus rhythm was restored spontaneously on the first day in the remaining three cases who both improved sufficiently to be discharged home.

Of the controls the patients with sinus bradycardia and auricular extrasystoles showed a spontaneous return to normal rhythm after 36 hours and both were discharged home still with sinus rhythm. The patient with supraventricular rhythm died in congestive failure after 22 days; after the administration of procaine amide his rhythm had altered to auricular fibrillation and this persisted until death. The patient with a nodal rhythm received procaine amide, sinus rhythm returned within ten hours, and eventual recovery was uneventful.

The later development of an arrythmia in patients with a previously normal rhythm was unaffected by treatment. Two steroid patients died on the second day following the onset of auricular fibrillation (A20) and ventricular fibrillation (A43). Two controls developed ventricular fibrillation on the second and sixth days (F33 and F39) and, in both, this rapidly proved fatal.

(b) DISTURBANCE OF ATRIOVENTRICULAR CONDUCTION.

A total of 33 patients (detailed in Table 44, Vol. II) showed a disturbance of atrioventricular conduction on admission, 18 steroid treated and 15 controls. Three degrees of disturbance were recognised; (Table 12).

POTENTIAL HEART BLOCK. This was considered to be present where the P-R interval was above 0.20 seconds but where no ventricular beats were dropped.

PARTIAL HEART BLOCK. This was considered to be present where the P-R interval was abnormally long and where ventricular beats were dropped, either in a regular fashion (e.g. 2 : 1 block) or irregularly (e.g. Wenkebach

FIG. 14.

MORTALITY RATES (%) IN PATIENTS WITH HEART BLOCK ON ADMISSION.

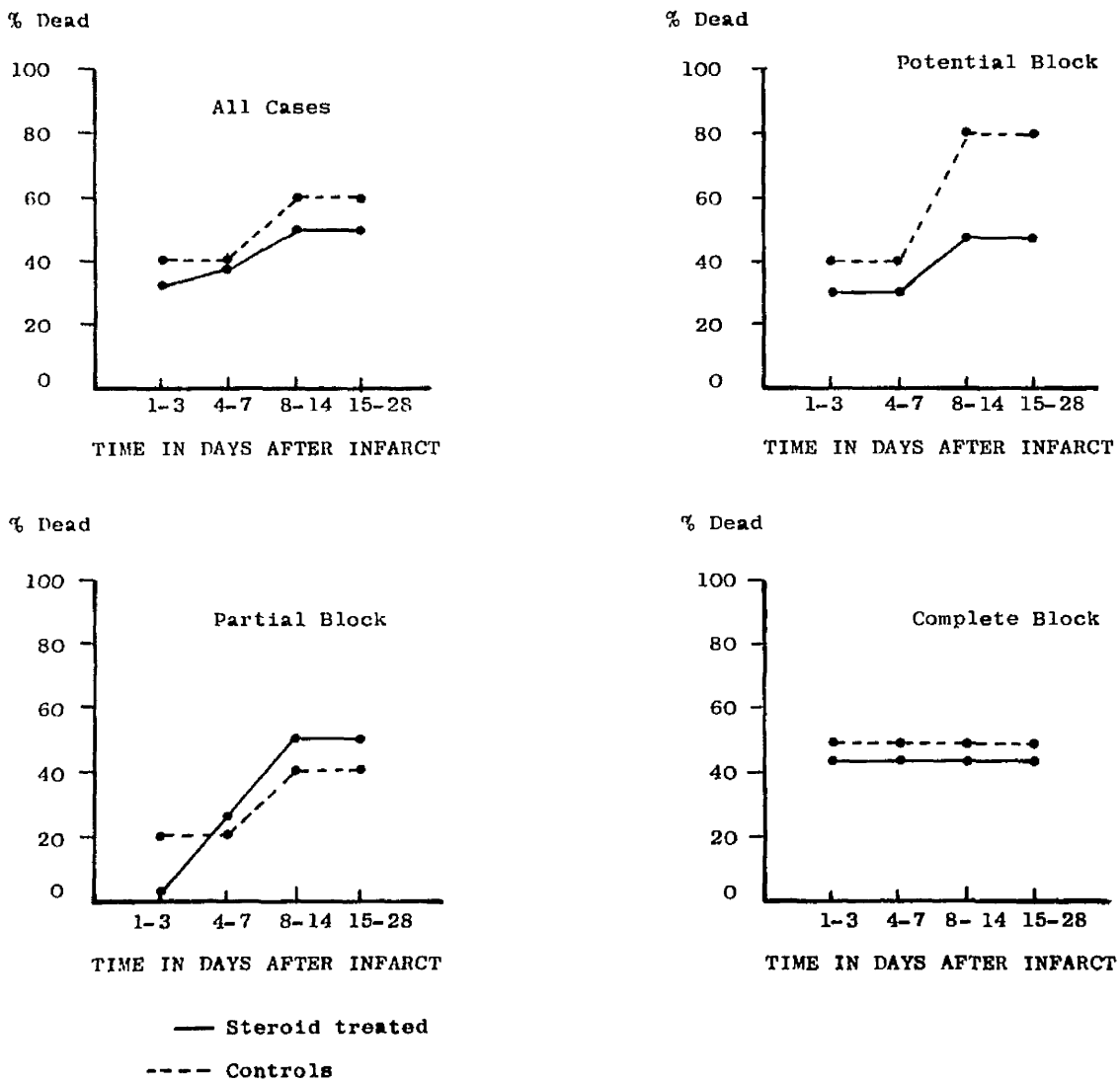


TABLE 13.

Number of patients dying within 28 days showing a persistant A - V conduction defect.

	Total	Degree of Persistant Block		
		Potential	Partial	Complete
Steroid treated patients	8	3	2	3
Control patients	8	4	1	3

TABLE 14.

Number of patients surviving 28 days showing a persistant A - V conduction defect.

	Total	Degree of Persistant Block		
		Potential	Partial	Complete
Steroid treated patients	1	1	-	-
Control patients	5	1	3	1

phenomenon).

COMPLETE HEART BLOCK. This was considered to be present when complete atrioventricular dissociation was noted, the auricles and ventricles beating independently at their own separate rates.

There was a striking difference in the behaviour of cases of heart block in the steroid treated and control series although there was no significant difference in the mortality rates taking all the cases together, or considering the individual degree of block (Fig. 14). Over one third of the patients died within the first week and about one half by the end of the first month regardless of the degree of block or of the treatment. Normal conduction was restored in ten steroid treated patients in from two hours to five days, with an average of twenty eight hours, but despite this eight of them died within twenty eight days. The block was over-:come in only two of the controls, in two and five days, and of the nine who died within twenty eight days block persisted in eight. Of the sixteen patients who survived this period normal rhythm had been restored in nine out of ten treated with steroids, but only in one of the six surviving controls.

In those patients in whom some conduction defect remained, slight improvement was seen in a minority of those who died and in half of those who survived for twenty eight days. The distribution of patients showing a persistent defect who died within twenty eight days, and who lived for twenty eight days after infarction, is shown in Tables 13 and 14 respectively.

Of those who died within 28 days, the only steroid treated case to show improvement (A35) was admitted with complete block; sinus rhythm was restored on the second day but by the time death occurred on the third day the P-R interval had not been recorded at less than 0.24 seconds. The only corresponding control case (F61) was admitted with partial block which reverted to potential block after 12 hours; the P-R interval was never less than 0.24 seconds and death occurred on the third day.

Of those who lived for 28 days, the only steroid treated patient was admitted with a P-R interval of 0.24 seconds which remained unaltered during the next eight months. Two of the control patients who were admitted with partial block (F26 and F63) showed a spontaneous change to potential block, the P-R interval six months later being 0.24 seconds and 0.26 seconds respectively. A third patient (F16) was admitted with atrio-ventricular dissociation which altered spontaneously to partial block soon after admission; complete block recurred on the 13th. day but sinus rhythm returned shortly afterwards with a P-R interval of 0.24 seconds and this remained unaltered three months later.

Details of all patients admitted with a conduction defect are listed in Table 44 (Vol. II) and are described in the clinical summaries (Vol. II). The significance of the response to steroid therapy will be discussed in Chapter 8.

Only one patient (D2) developed a conduction abnormality after admission; complete block appeared suddenly on the second day and death occurred one day later.

S U M M A R Y .

44% of the steroid treated patients were discharged from hospital compared with 32% of the controls. Higher prognostic index scores were associated with higher mortality rates in both groups. The beneficial influence of steroids was most evident where the score was 19 or under; with the highest scores (23 and above), there were no differences referable to treatment.

The six factors comprising the prognostic index were examined individually to determine whether an improved survival rate associated with steroids could be ascribed to a particular factor or factors. This revealed a striking advantage in favour of steroids in the presence of severe shock on admission. None of the other factors was influenced significantly by steroids. Although there was a marked difference in the clinical behaviour of heart block, the speedy restoration of normal rhythm in many steroid treated patients did not affect the prognosis; approximately half of all admissions died within three days irrespective of treatment.

C H A P T E R F O U R

AUTOPSY - NAKED EYE APPEARANCES

Weight of hearts; presence of left ventricular hypertrophy.

Prensence of Infarcts, recent and old.

Septal damage; relationship to heart block.

Thickness of infarct and normal wall; relationship between treatment and aneurysm or rupture.

TABLE 15.

Weights of hearts related to sex, and sex ratio of cases coming to autopsy.

	MALES		FEMALES		SEX RATIO
	No.	Avge Wt. (G)	No.	Avge wt (G)	M : F
Steroid treated (Series A – E	14	478	9	383	1.75 : 1
Controls (Series F)	14	465	8	382	1.75 : 1
Additional Control Group (Series H)	13	513	9	408	144 : 1

NAKED EYE EXAMINATION OF HEARTS.

Autopsies were performed on twenty-three patients treated with steroids (Series A-E) and on twenty-two controls (Series F).

In addition a group of nineteen routine postmortems performed on patients from the other medical unit in the hospital (Series H) were considered; all were admitted during 1961-3 and received treatment identical to that of Series F. Although these patients were not selected by the coronary prognostic index, all were clinically severe. Autopsies were not normally requested by this unit on cases of myocardial infarction unless they were clinically severe, postmortem being performed to confirm the extent of the myocardial damage. The patients constituting series F and H are thus essentially similar and it would be expected that the mode of resolution and repair of the infarcts in both groups would follow the pattern described by Mallory, White, and Salcedo-Sagar (1939) and Lodge-Patch (1951).

Naked Eye Examination of the Hearts.

WEIGHT. The average weight of the hearts, related to sex, is similar in each series, as is the sex ratio (table 15). Left ventricular hypertrophy was present in some degree, in 80% cases; there were no congenital abnormalities though the following valvular defects were present: rheumatic valve deformity, 1 case (A.21); aortic incompetence due to calcification of the cusps, 1 case (F 7); slight stretching of the aortic ring, 8 cases.

TABLE 16.

Position of recent infarcts seen at autopsy.

Position	No. of Cases in each position		
	Series A-E	Series F	Series H
Anteroseptal	10	9	8
Anterior	-	2	2
Lateral	1	-	1
Posterior	5	2	4
Posteroseptal	7	9	4

TABLE 17.

Position of old infarcts seen at autopsy.

Position	No. of Cases in each position		
	Series A-E	Series F	Series H
Anteroseptal	2	3	1
Anterior	-	1	-
Lateral	1	-	1
Posterior	-	-	-
Posteroseptal	3	3	2

TABLE 18.

Distribution of Cases showing heart block on whom autopsies were performed.

Degree of Block	Steroid Treated	Controls
Potential	4	2
Partial	2	2
Complete	1	2

INFARCTS: A large recent infarct was found in all 45 hearts examined from series A - F. In 42 the infarct was in the position indicated by the electrocardiograph tracings; no tracings were available from the other three. The distribution was similar in each series (table 16). In a quarter of the cases an old well fibrosed infarct was also present and these also had a similar distribution in each group (table 17).

Examination of the recent infarcts showed that all were of considerable size, none being less than 30 mm. in diameter. The coronary arteries showed moderate to severe atheroma in all cases and an antemortem thrombus was found in 8 steroid treated and 7 control hearts. Detailed description of each heart are given in the clinical summaries (Vol. 11).

RELATIONSHIP BETWEEN AUTOPSY FINDINGS AND HEART BLOCK.

Autopsy was undertaken in thirteen patients who had had some degree of heart block; the distribution of these patients is shown in table 18. Evidence of recent infarction was seen in the interventricular septum of all but one case (A 16) who had shown potential block. The amount of damage was not related to the degree of block present; in most cases the infarct involved $\frac{1}{3}$ - $\frac{1}{2}$ of the interventricular septum. In one patient (F.68) who showed complete block more severe damage seen; a recent infarct affected the posterior half of the septum while an old fibrosed infarct was present in the anterior quarter.

FIG. 15.

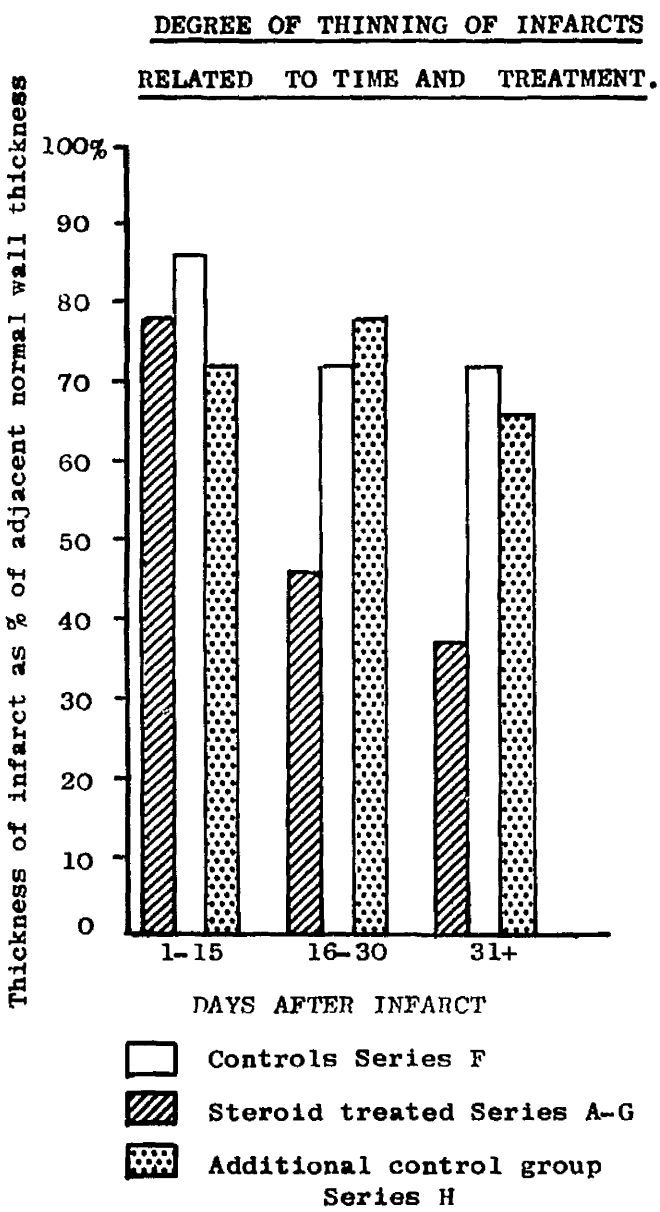

DEGREE OF THINNING OF INFARCTS
RELATED TO TIME AND TREATMENT.
Thickness of infarct as % of adjacent normal wall thickness
100%
90
80
70
60
50
40
30
20
10
0
1-15
16-30
31+
DAYS AFTER INFARCT
Controls Series F
Steroid treated Series A-G
Additional control group
Series H

TABLE 19.

Thickness of infarcted wall (expressed as % of normal wall thickness) in patients dying before the 29th. day.

Thickness of infarct %	No. of CASES		
	Series A-E	Series F	Series H
100-71	13	15	18
70-41	4	1	1
40-0	4	1	5

TABLE 20.

Incidence of thinning of infarct to under 70% of adjacent wall thickness in patients dying after the 15th. day.

SERIES	PROPORTION(%) SHOWING thinning to under 70%
A-E (Steroid)	83
F (Controls)	20
H (Extra Control Group)	33

TABLE 21.

Statistical Assessment of Table 20.

Groups Compared	Standard Error of difference of Means (%)	Minimum significant Difference (%)	Observed Difference
A-E and F	23.5	47	63
A-E and H	24	48	50
F and H	26	52	13

THICKNESS OF INFARCT AND LEFT VENTRICULAR WALL.

In all three groups a basically similar pattern was evident. Where death occurred within a day or two of infarction the infarct and adjacent healthy wall were of similar thickness. Where more time had elapsed the infarct was thinner than the adjacent wall and in general the degree of thinning became more marked with increase of time, (fig. 15). In cases dying within twenty-eight days of infarction, the infarct thickness was found to be less than 70% of that of the adjacent healthy wall in 29% of those given steroids (Series A-E), 12% of the controls (Series F), and 25% of the additional control group (Series H). The differences are not significant and reflect the small size of each group. (table 19). The degree of thinning was significantly more marked in the steroid treated cases dying after the 15th day (tables 20 and 21).

Only nine postmortems were undertaken in patients dying after the 28th day. In two of the three steroid treated cases there was marked thinning of the infarct; this was not observed in either of the other groups.

ANEURYSM AND RUPTURE.

Aneurysmal bulging of the recent infarct was seen in eight hearts, five steroid treated and three in the control groups (one in Series F and two in Series H). The combined incidence of aneurysm and rupture is similar in the steroid treated patients and those in the supplementary control group (Series H); it is not significantly

T A B L E 22.

Incidence of Aneurysm and Rupture of left ventricle.

SERIES	Number Observed		
	Aneurysm	Rupture	Total
A - E	5	0	5
F	1	0	1
H	2	2	4

FIG. 16

Case No. A10, steroid treated, 7th. day.

Rupture of right ventricle.

significantly different from that in Series F., the main control group (table 22).

Rupture of the left ventricle was not seen although in one steroid treated heart (No. A 10) there was rupture of the right ventricle (fig. 16). In Series G, however, rupture of the left ventricle had occurred in two hearts (11%). A search of the departmental records for the years 1951-60 inclusive revealed that death could be attributed to a recent myocardial infarction in 152 autopsies, and of these 25 (16%) had a rupture of some part of the heart. 18 (12%) showed rupture of the left ventricular wall, none being suspected clinically; this incidence is the same as that in series H and agrees with the findings of Levene (1960) who stated that the incidence of rupture in his P.M. series was 12.5%.

SUMMARY

The hearts of twenty-three steroid treated and twenty-two control patients were examined together with twenty-two hearts from an additional control group. A recent infarct was found in all and showed a similar anatomical distribution in each group. Of thirteen patients who had heart block, damage to the interventricular septum was found in twelve. The infarcted zone of the left ventricular wall was usually thinner than the adjacent wall; marked thinning, aneurysm formation, or rupture occurred in one quarter. The incidence of these changes bore no relationship to treatment.

C H A P T E R F I V E .

AUTOPSY - MICROSCOPIC APPEARANCES.

The four stages of the repair process.

Cell Counts on healing infarcts.

Effect of steroids on initial neutrophil infiltrate, and on scar formation.

Relationship between fibroblast count and aneurysm formation.

Effect of steroids on formation of peri-infarct basophilic material.

HISTOLOGICAL EXAMINATION OF HEARTS.

As detailed in Chapter 2 histological examination was undertaken on material from five groups of hearts:-

(1) 23 hearts from Series A-F (steroid treated).

(2) 22 hearts from Series F (main control group)

(3) 19 hearts from Series H (supplementary control group)

(4) 37 hearts from Series J (routine autopsy group of 1958-63).

(5) 11 hearts from other hospitals, Series K, (steroid treated).

Examination of this material shows that in all groups the process of resolution and repair follows an identical pattern. With steroid treatment the initial inflammatory response is markedly depressed and the conversion of young fibrous tissue into dense scar tissue is delayed although the general pattern of resolution and repair of the infarct follows that of the control cases.

The repair process can be divided into four distinct stages:-

I. STAGE OF INFILTRATION. During the first few hours after infarction there is slight pyknosis of the nuclei and minimal fatty change in the cytoplasm. Soon the infarct is invaded by vast numbers of neutrophils and these reach a peak after 2-3 days. Haemorrhage may occur during the first day but this is quite variable in extent.

II. STAGE OF PHAGOCYTOSIS. The neutrophil infiltrate disappears 5-6 days after infarction and is replaced by a less intense infiltrate of lymphocytes and macrophages. Phagocytosis of the dead myocardium begins

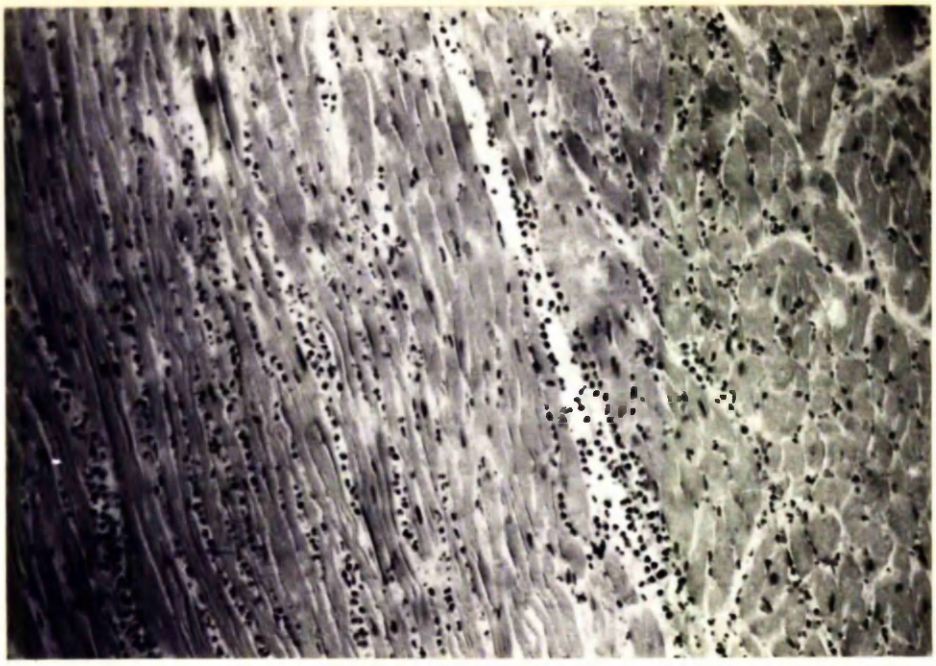


FIG. 17

Case No. A35, steroid treated, 3rd. day.

(H. & E. x 150)

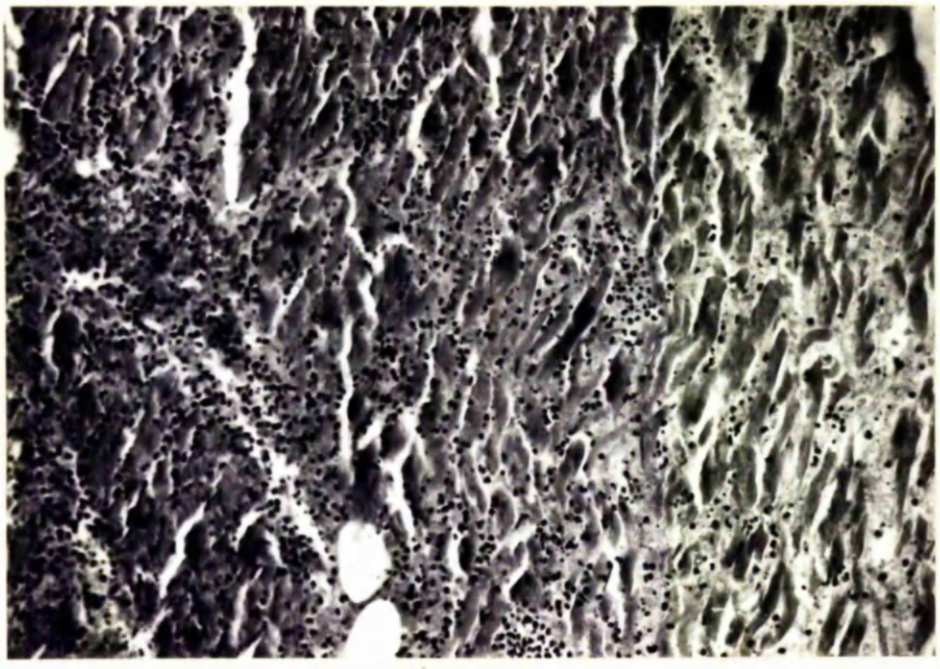


FIG. 18

Case No. F32, control, 3rd. day.

(H. & E. x 150)

The neutrophil infiltrate is much less intense in the steroid treated case compared with the control.

FIG. 19

Case No. C1, steroid treated, 16th. day.

(H. & E. x 320)

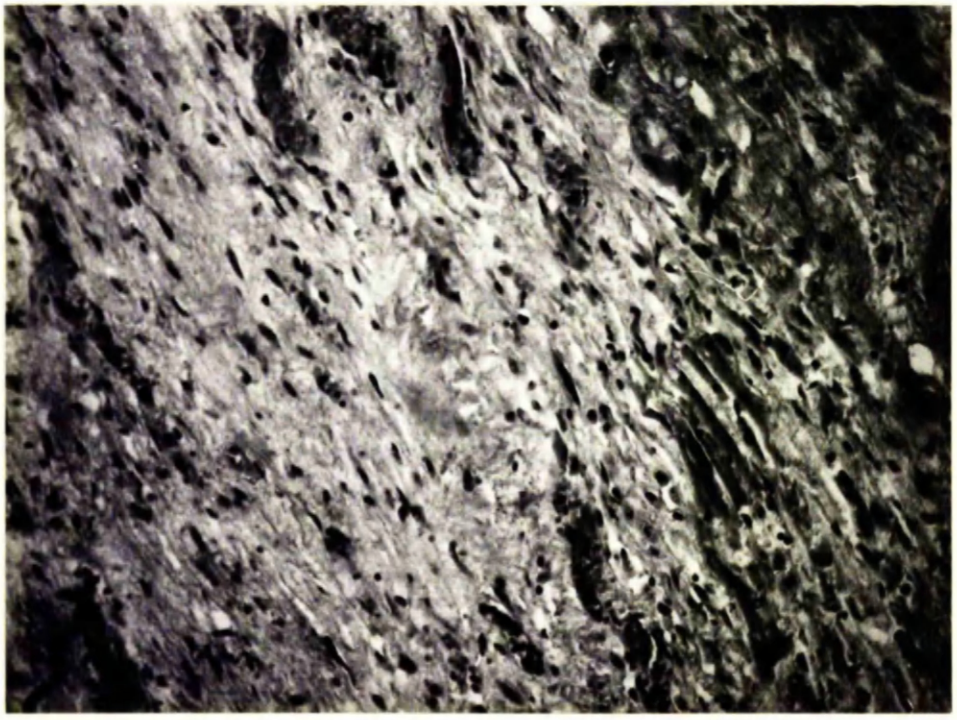


FIG. 20

Case No. H15, Additional control group, 15th. day.

(H. & E. x 320)

There are more fibroblasts and less evidence of hyalinisation in the healing infarct treated with steroids, compared with the control.

3-4 days after infarction and reaches a peak of 6-9 days, but may not be complete for a further week.

III. STAGE OF FIBROSIS. As the dead myocardial fibres are removed their place is taken by fibroblasts spreading into the infarct from the contiguous viable myocardium and at 10-14 days the infarct is largely replaced by cellular highly vascular fibrous tissue which is infiltrated diffusely by lymphocytes and by macrophages laden with haemosiderin or debris.

IV. STAGE OF HYALINISATION. The fibrous tissue slowly becomes less vascular and less cellular so that eventually it is relatively avascular, acellular and hyalinised. As these changes progress the inflammatory infil-:trate regresses slowly. This process is not complete until 2-3 months after infarction; at one month, when most patients are discharged from hospital, the infarct is still moderately cellular, though most of the large capillaries have disappeared.

CELL COUNTS WITHIN INFARCTS.

Histological sections stained by haemotoxylin and eosin were prepared from all hearts examined and the cells within 2 sq. mm. of infarct were counted as detailed in Chapter 2. The results are given in Tables 45-49 (Vol. II) inclusive. The counts on the steroid treated hearts differ from those in the other groups in two ways. (1) The reduced intensity of the initial neutrophils infiltrate, (Figs. 17-18) and (2) the increase in nucleated fibro-:blasts during the second and third week after infarction, (Figs. 19, 20).

In the steroid treated hearts the following pattern was apparent. During the first few days the infarct contains a dense neutrophil infiltrate which is at its most intense at the second day and thereafter regresses

slowly although many cells persist after one week. The neutrophils are replaced by a less intense infiltrate of lymphocytes and macrophages in approximately equal numbers; phagocytosis starts on the third day and takes up to three weeks to be completed. The phagocytosed muscle is replaced by young fibroblasts so that by the end of the second week the infarct is replaced almost entirely by tissue containing plump fibroblasts and numerous wide capillaries. Only after the end of the second week can significant degrees of hyalinisation be seen and in cases dying on the 39th. and 107th. days hyalinisation was incomplete; many nucleated fibroblasts persisted and some capillaries remained throughout the infarct.

In contrast, the hearts from Control Series F. H. and J. showed the following changes. The initial neutrophil infiltrate was more intense than in the steroid treated hearts, with counts up to double the steroid levels. This more intense reaction regressed more rapidly and most of the neutrophils had disappeared by the sixth day. As in the steroid treated hearts the neutrophil infiltrate is replaced by lymphocytes and macrophages; the intensity of the infiltrate is the same in steroid treated and non-steroid hearts. Phagocytosis proceeds with equal speed irrespective of treatment and the phagocytosed muscle is replaced by young fibroblasts. By the 12th. day the replacement fibrous tissue in the controls begins to show slight hyalinisation and this is reflected in a gradual drop in the count of fibroblast nuclei. In the steroid treated cases hyalinisation proceeds more slowly and therefore fibroblasts remain more numerous. At the end of the first month hyalinisation is well advanced in the controls; more so than in the two steroid treated patients dying after three months.

These serial changes are illustrated by figures 22-35 (Vol. II).

RELATIONSHIP BETWEEN FIBROBLAST COUNT AND ANEURYSM FORMATION.

Despite the differences in the fibroblast nuclei counts and the degree of hyalinisation no connection could be established between the count or degree of hyalinisation and the extent of thinning or of aneurysmal dilatation.

OTHER STAINING METHODS USED.

(1) Gordon and Sweet silver impregnation.

Preparations were made from old cases to demonstrate reticulin fibres using the Gordon and Sweet method. These reflect the changes already described in the routine sections; as hyalinisation of the fibrous tissue proceeds there is a reduction in the number of reticulin fibres. In steroid treated hearts this reduction proceeded more slowly than in controls, (Figs. 36-45, Vol. II).

During the first three days after infarction the pattern remains the same in all groups. Fairly thick reticulin surrounds each myocardial fibre and each small vessel. As phagocytosis proceeds this pattern is disrupted and is replaced gradually by a meshwork of very slender reticulin fibres so that after 10 days most of the original network has been obliterated. Thereafter the steroid treated and non steroid treated hearts show differences. In the non steroid groups the reticulin fibres become thicker and begin to lie parallel to one another around the circumference of the ventricle. Subsequently these fibres thicken further and become progressively fewer in number as the formation of collagen proceeds. At three months virtually no reticulin fibres remain within the infarct. In contrast, in steroid treated hearts the process of thickening of the fibres and the reduction in their

number is slowed down. At three months fairly numerous broad fibres remain in the infarct, an observation in keeping with the appearances in haematoxylin and eosin preparations which show that hyalinisation in the steroid treated infarcts is less than that in appropriate controls. In one steroid treated case dying after eight months (C.6) a few reticulin fibres still survive although the infarct as a whole is well hyalinised.

(2) Masson Goldner Trichrome Method.

Sections from all cases were stained by this method.

These confirmed the differences in degree of the hyalinisation of the steroid treated and control cases.

(3) Prussian Blue (Perls').

In haematoxylin and eosin preparations numerous macrophages laden with golden yellow pigment were seen in infarcts in all treatment groups, particularly in those dying at 2-3 weeks. The Prussian blue reaction confirmed that this pigment was haemosiderin, presumably derived from erythrocytes extravasated into the myocardium during the first day after infarction.

It was considered that the number of haemosiderin laden cells would be related directly to the variable amount of initial haemorrhage and not to the treatment given. For this reason separate counts were made of the pigmented and nonpigmented macrophages; counts of pigment laden cells in haematoxylin and eosin and in Prussian blue preparations were closely similar.

While haemosiderin laden cells are numerous during the second and third weeks there is often a considerable variation between infarcts of

TABLE 23.

Range and average number of haemosiderin laden macrophages seen in different groups if infarcts of 10 - 17 days' duration.

INFARCT GROUP	RANGE per sq. mm. of section.	AVERAGE NUMBER per sq. mm. of section
Steroid treated		
Series A-E	6 - 87	48.5
Non Steroid treated		
Series F	30 - 68	47.0
Series H	31 - 103	55.7
Series J	9 - 153	58.7

similar age, as would be expected if the number were related to the earlier variable amount of haemorrhage. In infarcts of 10-17 days the average number of pigment laden cells is similar irrespective of treatment (Table 23).

S E R I E S K.

This comprised histological material showing myocardial infarcts of steroid treated patients obtained from four different hospitals (Chapter 2).

The general appearances of those infarcts were similar to that seen in corresponding hearts in Series A-E. Detailed cell counts were performed and the results are similar to those in Series A-B (Table 49, Vol. 11), showing the same marked difference to the figures from Series F, the main control group. Because of possible differences in tissue shrinkage due to variation in the processing techniques used in each hospital, however, the counts in Series K were not compared directly with those from the other Series.

CHANGES AROUND THE INFARCT.

The appearances of the myocardium immediately around the infarct were studied in all cases.

In the steroid treated patients this remained almost entirely normal in appearance and was sharply demarcated from the infarct. In the controls the tissue spaces around the infarct became filled with a basophilic granular material and subsequent diffuse fibrosis occurred in the myocardium around the infarct so that the infarct appeared to merge with the contiguous tissue.

By study of all the cases the following serial picture was obtained:-

(a) NON-STEROID TREATED CASES.

Non steroid treated cases from all sources showed a similar picture. In haematoxylin and eosin stained sections the surrounding myocardium remained

apparently normal for the first day after infarction. Thereafter a very finely granular basophilic material appeared between the surviving apparently normal fibres in a zone 0.1-0.3 mm. wide around the edge of the infarct. This material was greatest at the 4-7 days when it filled the tissue spaces completely (Figs. 46 and 48, Vol. II), and thereafter it became infiltrated by small numbers of neutrophils, lymphocytes, macrophages and fibroblasts; it was gradually reabsorbed and replaced in part by fine fibrous tissue which surrounded the individual myocardial fibres close to the infarct (Figs. 45 and 50, Vol. II). In general the individual fibres remained normal in appearance but occasional fibres showed necrotic changes and were removed by macrophages.

Sections stained to demonstrate fat (Oil red O, Sudan black B methods), showed fatty changes in the myocardial fibres adjacent to the infarct; fatty change was visible in a zone of fibres adjacent to the infarct up to 0.5 mm. in width developing within the first 24 hours and reaching apeak at three days. Thereafter the process was reversed and the cells had returned to normal by two weeks.

In three cases acid phosphatase was demonstrated in moderate amounts in the infarct and between the adjacent surviving myocardial fibres, (Fig. 52, Vol. II); there was no obvious relationship to blood vessels within the infarct.

(b) STEROID TREATED CASES.

In haematoxylin and eosin preparations the myocardial fibres remained normal in appearance and by contrast with the controls, only small amounts of basophilic granular material was seen in only 5 (22%) hearts. The subsequent diffuse fibrosis between the myocardial fibres could not be seen around infarcts of more than 10 days' duration and as a result the demarcation between the

infarct and adjacent viable myocardium remained more obvious, (Fig. 47, Vol. II).

Fatty change in the surviving myocardium appeared to be present to the same extent as in the controls. Attempts to demonstrate acid phosphatase were made in only two cases; the amount of enzyme present within the infarcts was slightly less than in the controls while virtually none was visualised between the adjacent myocardial fibres (Fig. 51, Vol. II).

THE BASOPHILIC GRANULAR MATERIAL.

In haematoxylin and eosin sections this material was largely amorphous but it did include many tiny granular fragments and some groups of short fine fibrils. The material was amphophilic and in H. and E. sections appeared moderately basophilic. With the Masson Goldner trichrome technique it was blue-purple and resembled fibrin; more specific staining methods for fibrin, Masson 44/41, Fuchsin Miller and Martins Scarlet Blue (Lendrum et al 1963) however, were negative.

The material seemed to be composite. The Feulgen reaction was positive with many of the granular fragments but chromatin could not be identified in sections stained by other methods. The P.A.S. reaction was slightly positive in all areas of the material and a rather stronger reaction was present in similar areas using the alcian blue, P.A.S. - alcian blue, and alcian green reactions. The P.A.S. positive material was shown to be at least partially mucopolysaccharide in nature. The P.A.S. reaction was unaltered by prior treatment with diastase or hyaluronidase and the material gave a metacrhomatic reaction with toluidine blue and with methylene blue. Buffered solutions of toluidine blue in a pH range of 2.5 - 6.0 at intervals of0.5 pH showed that the metachromatic reaction began at pH 5.0 and was

extinguished at pH 2.5. Using methylene blue buffered at the same pHs. metachromasia was seen in the pH range 6.0 - 3.0. Pearce (1960) states that if nucleic acids can be excluded such a result confirms the presence of acid mucopolysaccharides. In this case the Feulgen reaction showed that nuclear material was present in relatively small amounts, most of the granular material being negative. The metachromasia demonstrated with both toluidine blue and methylene blue was seen in all areas of the granular material and appeared to be of uniform intensity. While a small part of the metachromasia might be due to the presence of nuclear material the greater part is considered to be due to acid mucopolysaccharides.

Although the P.A.S. reaction was at all times only weakly positive, the parallel method of sulphation-induced metachromasia using the method of Schrauth (1932) produced very intense metachromasia in all areas of the material confirming the widespread presence of polysaccharides.

As already recorded the Gomori method for acid phosphatase was positive in the area occupied by the granular material.

From the results it can be deduced that the basophilic material contains cellular debris comprising both nuclear and cytoplasmic elements. It is likely that most of this material is derived from the infarct, though some may be contributed by the plasma transuded into the infarct soon after its formation.

SUMMARY.

The general pattern of the resolution and repair of myocardial infarcts was the same irrespective of treatment.

There were four distinct stages:- I Stage of infiltration, II Stage of phagocytosis, III Stage of fibrosis, IV Stage of hyalinisation. In steroid treated patients the initial neutrophil infil-:trate was greatly diminished in intensity but it persisted longer than in controls. Phagocytosis and fibrosis of the infarct were not influenced by treatment but subsequent hyalinisation of the young fibrous tissue was greatly delayed in the steroid treated cases and was still incomplete in two patients dying at three months. The incidence of aneurysm and heart rupture was not related to treatment. A basophilic granular material was demonstrated between the myocardial fibres around the infarcts only in the non steroid patients and was shown to contain acid mucopolysaccharides and nuclear fragments.

C H A P T E R S I X .

ADDITIONAL LABORATORY AND CLINICAL RESULTS.

Serum Transaminases: Significance of effect of steroids on S.G.O.T. levels.

Serum Electrolytes: Importance of changes in potassium levels.

Haemoglobin levels:

Electrocardiograph Studies: The P-R interval; position of infarcts; onset of arrythmias after admission.

TABLE 24.

Average S.G.O.T. levels (Cabaud units) on first five days.

Series	Day after infarction					Average of highest levels regardless of Day
	1	2	3	4	5	
A	77	87	64	62	26	96
B	25	34	64	58	34	53
C	31	53	46	47	27	61
D	106	83	44	31	1	106
E	37	118	5	46	-	118
F	88	133	107	63	53	151
G	109	132	171	35	-	171
A-E	71	82	57	53	28	90
F-G	92	133	116	60	53	156

TABLE 25.

Average S.G.P.T. levels (Cabaud units) on first five days.

Series	Day after infarction					Average of highest levels regardless of Day
	1	2	3	4	5	
A	56	70	50	117	49	79
B	25	32	61	80	77	64
C	25	52	53	46	67	53
D	33	47	19	11	-	42
E	29	42	42	30	-	43
F	60	66	74	89	79	87
G	34	52	144	26	-	110
A-E	47	61	47	86	57	70
F-G	56	65	84	82	79	90

ADDITIONAL LABORATORY AND CLINICAL RESULTS.

(1) Serum Transaminases:

279 estimations each of serum glutamic oxylacetic transaminase (S.G.O.T.) and of serum glutanic pyruvic transaminases (S.G.P.T.) were available for analysis from 110 patients including six from Series G before they had received steroids, (Chapter 2).

Lower average S.G.O.T. levels were found in steroid treated patients than in controls and values above normal were found in only 82% of those given steroids compared with 98% of controls (Table 24). The corresponding proportion found in groups of routine myocardial infarct cases by other workers is 97% (Agress and Kim, 1960); 97% (Meyers and Evans 1964); 96.9% (Agress, 1959), and 95.5% (Wahlberg, 1963). The results did, however, show a similar pattern irrespective of treatment with levels usually above normal on the first day, rising to a peak on the second day and then falling slowly. The value on the second day in steroid cases was little over half that in the controls, the difference being statistically significant (3.4 times the standard error of the difference of the means). By the fifth day the average value in steroid cases was normal while that in controls was still above normal. It would seem that the rise in S.G.O.T. levels is suppressed in those given steroids and in some a diagnostic rise is prevented.

S.G.P.T. estimations show no such differences; the level often is normal on admission and rises to a similar peak after 3-4 days irrespective of treatment (Table 25). A rise above normal was seen in 73% of steroid treated and 79% of control patients. Since the S.G.P.T. level is

TABLE 26.

Average serum electrolyte levels in M.eq./l. at various times after infarction, in steroid treated and control patients.

Series	Time after infarction (Days)	No. of Estimations	Serum Electrolyte Levels						
			No.	K	(Sum of) (Anions)	Cl	CO_2	Protein	(Sum of) (Cations)
A-E	2-3	11	141.1	4.27	(145.4)	101.0	21.9	15.4	(138.3)
F		10	141.6	4.69	(146.2)	104.7	23.5	15.2	(143.4)
A-E	6-8	16	141.1	4.05	(145.6)	102.3	24.8	15.2	(142.3)
F		17	141.2	4.54	(145.7)	102.3	24.8	15.3	(142.4)
A-E	13-15	6	141.5	4.17	(145.7)	101.2	26.1	15.0	(142.3)
F		7	139.8	4.62	(144.4)	101.5	25.1	15.4	(142.0)
A-E	21-28	5	139.4	4.47	(143.9)	100.6	26.0	15.0	(141.6)
F		6	138.3	4.90	(143.2)	100.5	23.6	15.0	(139.1)

TABLE 27.

Examination of Serum Electrolyte Estimations to illustrate number outwith normal range. (Results in M.eq./l).

TIME (DAYS)	SERIES	No. of Estimations	No.			K			Cl			CO_2		
			Normal	High	Low	Normal	High	Low	Normal	High	Low	Normal	High	Low.
2-3	A-E	11	11	-	-	7	1	3	11	-	-	4	-	7
	F	10	10	-	-	10	-	-	10	-	-	6	-	4
6-8	A-E	16	14	1	1	7	-	9	14	1	1	10	1	5
	F	17	17	-	-	17	-	-	17	-	-	16	-	1
13-15	A-E	6	6	-	-	4	-	2	6	-	-	6	-	-
	F	7	7	-	-	7	-	-	7	-	-	5	-	2
21-28	A-E	5	5	-	-	5	-	-	5	-	-	5	-	-
	F	6	6	-	-	5	1	-	6	-	-	4	-	2

considered to reflect the degree of hepatocellular damage associated with cardiac failure (Wilkinson 1962), these results suggest that steroid therapy had no effect on the degree of failure.

(2) Serum Electrolytes:

These form a small part of the study, 78 estimations from 45 patients made during the following periods of time after infarction being available.

(1) 2 or 3 days (21 estimations)

(2) 6-8 days (33 estimations)

(3) 13.15 days (13 estimations)

(4) 3-4 weeks. (11 estimations)

The averaged results shown in Table 26 show little difference between steroid treated (Series A-E) and control (Series F) patients, but more detailed examination of the individual figures reveals that in the steroid treated cases the serum potassium and carbon dioxide levels were frequently subnormal; this was not so in the controls. Serum sodium and chloride levels were almost always normal at all times irrespective of treatment (Table 27).

The serum potassium levels show the most important departure from normal. In steroid treated patients almost half of the estimations performed up to the eighth day gave subnormal results. Of the nine low values at 6-8 days, six were minimally lowered (3.9 m.eq./l.) but the others had values of 3.1, 3.6 and 3.7 m. eq./l. The two subnormal values at 13-15 days were only 3.1 and 3.4 m. eq./l. The levels were therefore at a potentially dangerously low level on five occasions though at no time did any arrythmia occur when a hypokalaemia was known to be present. It is evident

TABLE 28.

Average haemoglobin (%) on admission (100% = 14.80).

	Males	Females	A C C
Steroid treated cases	98	91	95
Controls	95	91	94

that even with transient steroid therapy there is a significant effect on potassium levels and in any future similar study the need to check electrolyte levels during the first 2-3 weeks is stressed.

Carbon dioxide levels. These were often subnormal in both the steroid treated and control groups, and this was probably the result of a lactic acidosis secondary to cardiac failure and hypotension. One patient had a high level but was admitted in severe congestive failure; this patient also had high serum sodium and chloride levels and the high CO_2 level was therefore probably compensatory. Subnormal levels were found in 21 patients (12 steroid treated and 9 control) but in only three were valves found below 20.0 m.eq./l. These were 18.4, 14.2 and 15.5 m. eq./l. on the second, seventh and 28th. days respectively in cases Nos. F11, F60, and F27 respectively.

Haemoglobin Studies:

Average haemoglobin levels, estimated usually by a Sahli haemoglobinometer and sometimes by a cyan-photo-electric method (Chapter 2) were closely similar in the steroid treated and control groups (Table 28). Only two patients were anaemic, both being female controls with a haemoglobin of 76%; the film appearance in both showed slight hypochromia,

Additional Results from Electrocardiographic Studies.

(a) The P-R interval. Reference has been made to the abundant literature showing that the administration of steroids in acute heart block often results in a rapid improvement, with abolition of the block or shortening of the P-R interval and the results recorded in Chapter 3 have confirmed this.

The P-R interval has been studied in those who did not show heart

TABLE 29.

Average P-R intervals (No evidence of Heart Block on admission).

Series	P-R interval (seconds)		
	On admission	8th. Day	28th. Day
A	0.19	0.17	0.17
B	0.18	0.17	0.18
C	0.18	0.18	0.18
D	0.18	0.16	0.17
E	0.18	0.17	0.17
A-E	0.19	0.17	0.18
F	0.18	0.17	0.17

TABLE 30.

Average P-R interval (Excluding those with partial or complete Heart Block).

Series	P-R interval (seconds)		
	On admission	8th. Day	28th. Day
A	0.19	0.17	0.18
B	0.18	0.17	0.18
C	0.20	0.18	0.18
D	0.18	0.16	0.17
E	0.18	0.17	0.17
A-E	0.19	0.17	0.18
F	0.19	0.18	0.19

TABLE 31.

Number and position of infarcts as shown by electrocardiographic tracings.

SERIES		Position of Infarct				
		Ant. or A/S	AL	L	PL	Post or P/S
STEROID TREATED	A	17	5	2	3	18
	B	1	-	-	2	1
	C	6	-	-	2	4
	D	5	-	-	-	-
	E	2	1	-	-	1
	A-E	31	6	2	7	24
CONTROL	F	29	3	4	5	24

TABLE 32.

Site of infarct related to survival at 28 days.

(a) Steroid Treated Patients (Series A-E)

Position		Anterior	A-L	Lateral	P.L.	Posterior
No. of Cases	Alive	14	4	0	5	11
	Dead	17	2	2	2	13
%	Alive	45	67	0	61	46

(b) Control Patients (Series F)

Position		Anterior	A-L	Lateral	P.L.	Posterior
No. of Cases	Alive	12	2	3	2	9
	Dead	17	1	1	3	15
%	Alive	31	67	75	40	38

TABLE 33.

(a) Position of old infarcts seen on E. C. G.

	Steroid Treated	Controls
Anterior	9	7
Lateral	2	-
Posterior	6	2

(b) Position of unsuspected infarcts found at P. M.

	Steroid Treated	Controls
Anterior	-	-
Lateral	2	-
Posterior	4	1

(c) Position on E.C.G. tracing of old infarct in relation to new infarct.

	Steroid Treated	Controls
Adjacent	5	2
Separate	12	7

(d) Position of unsuspected infarcts found at autopsy related to recent infarct.

	Steroid Treated	Controls
Adjacent	2	-
Separate	4	1

block. The average P-R interval fell slightly between admission and the eighth day and usually rose again a little by the 28th. day irrespective of treatment (Table 29). No significant difference was found between the steroid treated and control groups. Inclusion of these cases of potential block having a constant P-R interval, made no significant difference to the overall figure (Table 30).

(b) Position of Recent Infarct. The distribution of the anatomical position of the recent infarcts as seen on E.C.G. tracings was closely similar (Table 31, and the position had no effect on mortality rate (Table 32).

(c) E.C.G. evidence of Old Infarction. Although there was a history suggestive of previous infarction in 22 steroid treated and in 32 control patients a definite old infarct was identified in only 17 steroid treated and 9 controls. In only one patient (A8) was an old infarct seen in E.C.G. tracings in the absence of a history of an old infarct. These infarcts showed a similar anatomical distribution in each group, (Table 33a). Seven old infarcts, suspected neither by the history nor by E.C.G. tracings, were found at autopsy, 6 in steroid treated patients and one in a control, (Table 33b). In most cases the new infarct was in a different position from the old (Tables 33c and 33d).

(d) Onset of arrythmias after admission. In Chapter 4 arrythmias present on admission were considered. Arrythmias appeared after the day of admission in only four steroid treated and four control cases.

Steroid treated cases. Ventricular extrasystoles occurred in two patients. In case C7 this was noted on the fifth day and was controlled partially by the administration of quinidine; however, congestive cardiac failure ensued and death occurred on the 11th. day. In case D1 ventricular

extrasystoles appeared on the 28th. day but subsided spontaneously within 24 hours. In case A20 auricular fibrillation appeared on the second day and death occurred soon afterwards. Ventricular fibrillation occurred in case D1 on the second day soon before death.

Control Cases. Case F18 had sinus tachycardia on the fifth day but this settled spontaneously within a few hours. Cases F33 and F39 both showed ventricular fibrillation shortly before death on the fourth and sixth day respectively. Case F63 developed a nodal rhythm on the third day but this subsided after the administration of quinidine.

5) Other Complications attributable to Steroid Therapy.

Apart from the alterations in the repair process of the infarct, and on the serum electrolyte levels, there were no side effects attributable to steroids. Many such side effects have been described (in Noble 1953) but in this study high dosage therapy was too transient to produce the classical "moon face" effect, or to result in significant osteoporosis. No patient was known to have a peptic ulcer, a potentially active tuberculous lesion, or to suffer from epilepsy.

SUMMARY.

S. G. O. T. levels were markedly lower in steroid treated patients than in controls although in both groups the levels frequently rose above normal to reach a peak on the second day. It is possible, however, that steroid therapy could lessen the diagnostic value of S. G. O. T. estimation. S. G. P. T. levels showed no such difference; peak levels occurred at 3-4 days.

Serum potassium levels were frequently subnormal during the first week in steroid treated patients, the results in controls being normal. Sodium and chloride levels were normal and patients in both groups showed low CO_2 levels particularly after one week. Haemoglobin levels were normal in most patients.

Examination of electrocardiograph tracings showed that the recent infarcts had a similar anatomical distribution in both groups. Where old infarcts were present they were generally in a position different from the new. The P-R interval in patients without heart block showed a similar pattern irrespective of treatment. The interval shortened during the first week after admission and then lengthened to near the initial level by the end of the first month.

C H A P T E R S E V E N.

FOLLOW-UP OF DISCHARGED PATIENTS.

TABLE 34.

Admission period (days) of discharged patients.

Series	No. of survivors	Average days in hospital
A	19	38.4
B	2	28.0
C	7	34.0
D	1	33.0
E	3	45.0
A–E	32	37.3
F	22	39.6

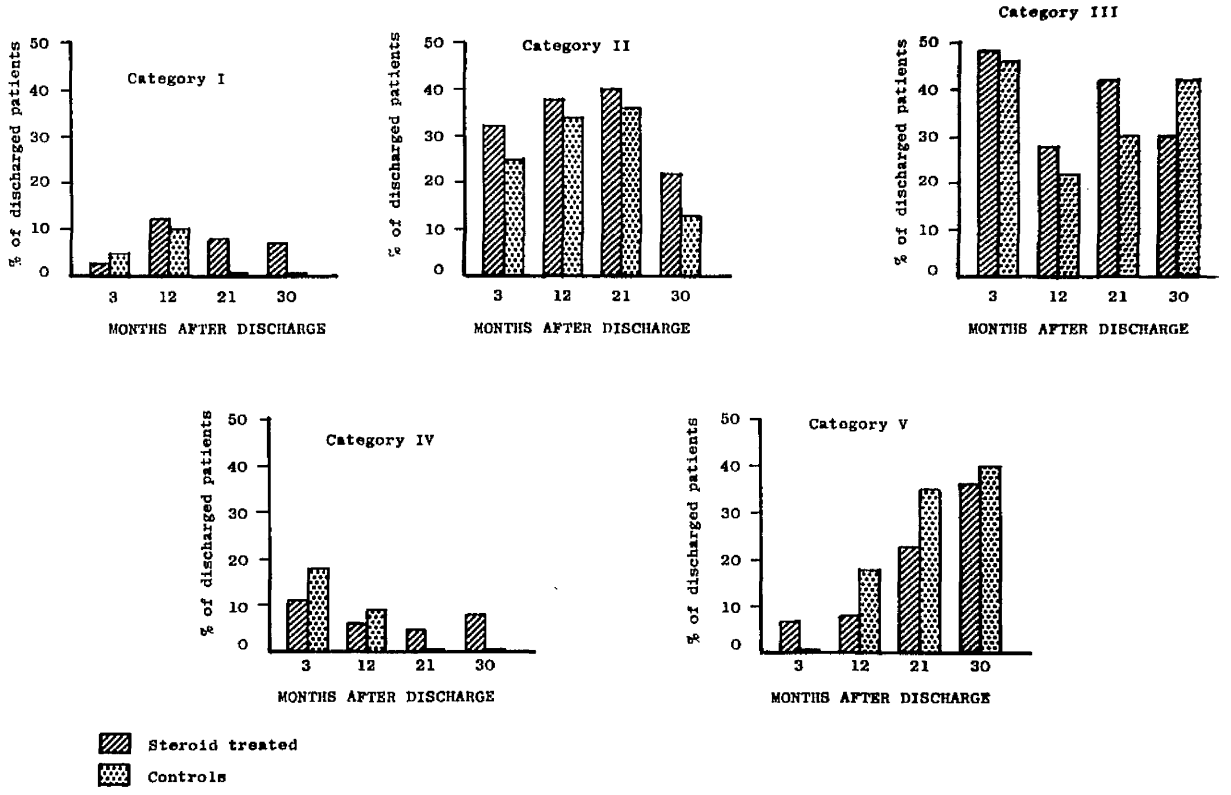
TABLE 35.

Sex distribution and average age of discharged patients.

Series	A–E	F
Number of Cases	32	22
Male : Female ratio	1.75 : 1	1.36 : 1
Average Age (All)	61.7	64.3
Do. (Males)	61.5	63.9
Do. (Females)	61.9	65.0

FIG. 21.

PROPORTION OF DISCHARGED PATIENTS IN EACH CLINICAL CATEGORY AT VARIOUS TIMES AFTER DISCHARGE.



FOLLOW-UP OF DISCHARGED PATIENTS.

54 patients were discharged from hospital; 32 steroid treated and 22 controls. The average days in hospital (Table 34), average age and sex ratio (Table 35) were similar. The follow-up period extended for 12-48 months with an average of 26 months.

Most patients were seen regularly during the first 1-2 years after discharge and thereafter frequent reports were received from the patients' family doctors, (Chapter 2). Eleven patients were re-admitted with cardiovascular complications and were then reassessed. One patient was admitted to another hospital soon after discharge and remained there until his death.

To condense this information a simple grading system was devised as described in Chapter 2.

Grade I Patient completely well - no disability.

Grade II Patient well - slight cardiac disability.

GRADE III Moderate cardiac disability.

GRADE IV Cardiac cripple.

GRADE V. Patient dead.

Comparing the gradings in all survivors, no significant difference was demonstrated between the steroid treated and the control groups. The categories for each patient at various times are given in Tables 50 and 51, Vol. II.

Most of the patients lay in categories II or III; there was no significant difference with treatment (Fig. 21). Few patients lay in categories I or IV but an increasing proportion died (category V). These

TABLE 36.

Details of Deaths occurring after discharge from hospital.

	Case No.	Time of Death after infarct (Months)	Ultimate Cause of Death
(a)	STEROID TREATED CASES		
	A3	1½	Congestive cardiac failure
	A5	14	Further myocardial infarct
	A16	26	Further myocardial infarct
	A30	8	Congestive cardiac failure
	A37	9	Generalised atherosclerosis
	A41	19	Further myocardial infarct
	A42	19	Congestive cardiac failure: Gangrene of Leg
	C6	9	Cerebral Haemorrhage
	D3	3	Further myocardial infarct
(b)	CONTROLS		
	F5	16	Cerebral haemorrhage
	F14	4	Further myocardial infarct
	F15	4	Further myocardial infarct
	F19	4	Congestive cardiac failure
	F28	4	Congestive cardiac failure
	F43	5	Further myocardial infarct

included nine steroid treated (28%) and six control (27%) patients; the cause of death in all was related directly to the cardiovascular system (Table 36).

S U M M A R Y .

There is no significant difference in subsequent progress in the steroid treated and control groups. Approximately one quarter died during the follow-up, due to a cardiovascular lesion in every case; of the remainder, one half were reasonably well and the other half were incapacitated to a significant degree.

C H A P T E R E I G H T.

DISCUSSION OF RESULTS.

DISCUSSION of RESULTS.

The results presented show that with the exception of a very few patients admitted in gross shock steroid therapy has no significant effect on the immediate prognosis; the longer term outlook also appears to be identical.

Steroid therapy has not, however, been without effect. Substantial modification of the initial inflammatory reaction and of the later scar formation have been demonstrated within the infarcts, the elevation of the serum glutamic oxalacetic transaminase has been partially suppressed, and a considerable depression in the serum potassium levels during the first week has been observed in several patients. It has been shown that steroids can bring about a rapid restoration of sinus rhythm in many cases of acute heart block, but this did not improve the prognosis, and steroids had no significant influence on the P-R interval in patients who had normal conduction.

While some of these effects may be beneficial to the patient, others must be regarded as being at least potentially dangerous.

The effects of steroids on the healing infarct and the surrounding area of surviving myocardium are of considerable importance, and three separate sections must be considered:

(1) The suppression of the acute inflammatory reaction within the infarct and the surrounding viable myocardium during the first few days.

(2) The alteration in the subsequent changes in the contiguous surviving myocardium.

(3) The delay in the conversion into dense collagen of the young fibrous tissue replacing the infarct as from about the 10th. day onwards.

(1) Plotz (1957) pointed out that around a recent myocardial infarct there was a zone of viable myocardium which, however, was anoxic because of oedema. Steroids are known to suppress acute inflammatory reactions in widely differing parts of the body and the marked inhibition of the neutrophil invasion into recent infarcts observed in the study in steroid treated patients is evidence that this same anti-inflammatory action occurs in the myocardium. This finding is in agreement with results of earlier work on humans (Rebuck and Mellinger, 1953, Cotterell, Wiener and Spiro, 1964), and on animals (Ragan and Howes, 1949; Baker and Whitaker, 1950; Chapman, et al 1952; Johnson, et al 1953; Wexler and Kittinger, 1963; Wexler, 1964 a, b, c).

The important point is that not only is the neutrophil infiltration modified, but the oedematous reaction in the surrounding myocardium is also limited.

This profound anti-inflammatory effect may explain the often dramatic restoration of sinus rhythm in instances of acute heart block complicating myocardial infarction. If part of the conduction system is actually infarcted then a permanent upset in conduction is to be expected (Rossi, 1962), but if the conduction tissue is merely involved in the peri-infarct reaction then any upset in conduction would be temporary and the return of normal conduction would be facilitated by the regression of the oedema. Thus, if steroids could limit the formation of the oedema fluid, or hasten its resorption, sinus rhythm might be restored more speedily than normal. Such an effect would certainly explain the extremely prompt restoration in seven cases in this study, as well as the reports of similar findings (Prinzmetal

and Kennamer, (1954), Wohlrabe, (1956), Phelps and Lindsay, (1957), Friedberg et al (1960), Aber and Wyn Jones (1960), Fay and Waverley, (1961), Dall, (1962), Dall and Buchanan, (1962), Grieco and Andreae, (1963). Dall, (1962) and Prinzmetal and Kennamer, (1954) have illustrated the remarkable way in which steroids can act. Both described patients with complete heart block following a recent myocardial infarct, in whom block was abolished temporarily within a few hours of receiving steroids; in both patients the block was again speedily but temporarily abolished by the administration of more steroids on two further occasions. These authors considered that this demonstrated the rapid anti-inflammatory action of steroids and the return of the block was thought to be a consequence of this action wearing off.

Not all writers accept that this effect of steroids is due to a direct anti-inflammatory action. Bellet, Wasserman and Brody, (1955), in their study of patients with chronic heart block and Stokes-Adams attacks, found that the intravenous infusion of half molar sodium lactate produced a hypo-:kalaemia which was thought to stimulate the ventricular pacemaker and improve atrioventricular conduction. Tobian (1961) considered that the abolition of block by steroids was due to the production of a hypokalaemia with similar effects. Although he describes an improvement in cases of chronic heart block, the change was more gradual and certainly was in no way as dramatic as that described by Prinzmetal and Kennamer or by Dall. In the present study low serum potassium levels were seen in several patients, usually about a week after infarction. In no patient could a significant alteration in the P-R interval be related to a low serum potassium level.

The observations of Lown (1955) on the P-R interval in normal individuals and in others with Addison's Disease and with Cushing's Syndrome

suggest that steroids may have an action which facilitates conduction. This view is supported by the observations of Prinzmetal and Kennamer (1960), and of Pay and Waverley (1961), in their studies of cases of long standing block with Stokes Adams seizures. These were improved by the administration of steroids; often sinus rhythm was not restored but the heart rate was increased and the Stokes Adams attacks abolished. This effect was considered to be due either to an improvement in conduction, or to the stimulation of the ventricular pacemaker. This suggestion is attractive since the anti-inflammatory theory cannot be applicable to cases of chronic block in which active inflammatory changes are absent.

In the present study autopsy was undertaken on 13 patients who had shown some degree of heart block. In 12 recent septal damage was demonstra-:ted at autopsy which supported the theory that the conduction disturbance was associated with inflammatory changes in the septum. The one exception (C.6) had potential block which did not improve with steroids, and there was no septal abnormality at autopsy performed eight months after the infarct.

It seems likely that the restoration of sinus rhythm is the result of both an anti-inflammatory action and a facilitation effect, and the importance of each varies in individual cases.

Whatever the mode of operation, the restoration of sinus rhythm had no significant effect on the prognosis. Unless Stokes Adams attacks were very frequent the presence of even complete block with a heart rate of about 30 per minute would seem to be less important prognostically than the total amount of myocardial damage that has been sustained. Complete heart block is certainly wholly compatible with life in a patient resting in bed but no treatment can help a patient who has suffered excessive myocardial

damage; thus those patients with extremely large infarcts must be expected to die whatever their rhythm might be. The continued presence of heart block is only of importance whenever the acute stage has passed and the patient becomes more active.

Apart from its effect on the surrounding myocardium the diminution of the initial inflammatory reaction seems to be of little importance. In both the steroid treated and control cases, tissue macrophages and lymphocytes appeared in approximately equal numbers at the same time, and there was nothing to suggest that subsequent phagocytosis was delayed by steroids.

(2) The changes in the myocardium surrounding the infarct after the first day are of considerable interest. In the controls a basophilic material, largely mucopolysaccharide in nature although containing some chromatin fragments and acid phosphatase, was seen to lie between the surviving muscle fibres. Subsequently this material underwent resolution and was replaced partially by fibrous tissue which was continuous with the fibrous tissue of the healing infarct. Thus the infarct appeared to merge with the surrounding myocardium and the peripheral fibrous meshwork seemed to fix the scar firmly into the adjacent viable muscle. In steroid treated patients only very small amounts of such material could be seen and the healed infarct seemed more sharply defined from the adjacent viable tissue. It would appear that steroids had interfered with the normal repair process to produce a scar which was less adequately fixed into the wall, thus creating the possible danger of later rupture of the heart through the edge of the infarct. In fact only one steroid treated patient (A10) is known to have died following cardiac rupture and in this instance the rupture occurred through recently necrotised myocardium. Of the other patients who died in hospital none was considered

to have suffered a rupture and in most of those who died suddenly or unexpectedly the absence of rupture was confirmed at autopsy.

Of those who died after discharge from hospital, death from a cardiac cause was sudden in two cases only (Nos. A3 and A41). In both there was a clinical history strongly suggestive of a further myocardial infarct and death did not occur for several hours, a period that is longer than would be expected if the preceding pain had been due to rupture of an earlier infarct.

Thus although there is nothing in the present study to suggest that the incidence of heart rupture would be increased by steroid therapy, a theoretical danger must remain and a larger series might produce a different conclusion.

It has not been possible to discover the precise reason for the difference in the amount of basophilic granular material in the two groups of cases but recent work on the nature of lysosomes suggests a possible explanation. De Duve (1963), Novikoff (1964), and Brandes (1964) showed that the lysosome is a tiny cytoplasmic organelle containing a wide variety of powerful enzymes, including acid phosphatase, enclosed within a very thin lipoprotein membrane. Anoxia causes a rapid breakdown of this membrane resulting in the escape of the enzymes into the cytoplasm and the autolysis of the cell. Weissmann and Thomas (1962) pointed out that the administration of steroids prevented, or at least greatly slowed down, the disruption of the membrane and thus limited the escape of the destructive enzymes.

In the present work free acid phosphatase has been demonstrated within several recent infarcts and also within the surrounding basophilic granular material, but the enzyme was less in the steroid treated cases.

When myocardial fibres were surrounded by the basophilic material a few showed necrotic changes, but where the material was absent no such change was seen. This slender evidence suggests that the basophilic material contains cytolytic enzymes, presumably derived from the infarct, and indicates that less enzyme is present if steroids are given. The protective action of steroids on the lysosome membrane may explain this.

(3) The delay in the collagenisation of young fibrous tissue is of practical importance; although a dense scar is probably formed eventually, healing is not complete after one month at the time most patients who have survived are discharged home. Since cellular fibrous tissue stretches more readily than dense scar tissue when subjected to the same degree of stress, the relatively prolonged persistence of cellular fibrous tissue in steroid treated infarcts would lead to a greater tendency to thinning of the fibrous wall, with ultimate aneurysm formation. Study of the hearts from patients dying more than 15 days after infarction confirms that there is a significant tendency to thinning and aneurysm formation where steroids are given, (Tables 19-21).

It is not known whether there is an increased tendency to aneurysm formation in those steroid treated patients who still survive. None has shown any evidence of significant enlargement of cardiac dullness since their infarction and a chest X-ray in three has not revealed an aneurysm. Nevertheless, since a small or early aneurysm would be unlikely to be dis-:covered clinically, rupture might be the first evidence of weakness. As stated above none of the patients who died after discharge was thought to have suffered a rupture, but the possibility of such a complication would seem to be increased in steroid treated patients.

In addition to this important histopathological evidence, several

clinical factors must be considered.

Although steroid treatment had little overall effect on the mortality rate it did have a significant effect in a small group of grossly shocked patients; this was much less marked than in the series reported by Anfossi (1963).

Although normal rhythm was restored speedily in several cases with heart block, administration of steroids did not alter the immediate 50% mortality rate. This was indeed disappointing since it had been anticipated that if the block could be overcome it was reasonable to hope that the mortality rate would fall. Complete block itself, however, is not incompatible with life and a patient lying sedated in bed can frequently survive without distress despite a low heart rate. A patient with heart block following a recent infarct dies not because of the block but because of the infarct; if too much myocardium has been destroyed then the patient will die whether or not block is present. If the block is accompanied by frequent prolonged Stoke-Adams attacks this would affect the short term prognosis and steroids might be life saving. In the present study, however, this situation was seen once only (No. A39) and the patient died despite hydrocortisone therapy.

By suppressing the rise in serum glutamic oxalacetic transaminase, steroids reduced the clinical value of this biochemical estimation. The peak levels in steroid treated patients were little over half those in controls and a diagnostic rise in S. G. O. T. levels was absent in 18% of those given steroids compared with only 2% of controls. This study was undertaken on clinically extremely severe cases and a diagnostic rise was to be expected in all; S. G. O. T. levels can be related to the infarct size, high enzyme

levels being associated with large infarcts (Nydick, Wroblewski and La Due, 1955). Steroids appear to have suppressed a diagnostic rise in a proportion of cases, the recorded levels remaining within normal limits. Had the patients been clinically less severe, with a correspondingly less marked S. G. O. T. elevation, steroids might well have caused a greater proportion of false negative results and thus seriously undermined the diagnostic value of the estimation. The reason for this action is difficult to find but the power of steroids to prevent the disruption of the slender lipoprotein membrane of the lysosome (De Duve, 1963, Brandes, 1964, Novikoff, 1964) and thus limit the escape of certain cytolytic enzymes from the dead myocardial cells, may offer an explanation.

That steroids can induce a hypokalaemia is well known (Sprague et al 1950, Luft and Sjorgen, 1951, Sjorgen, 1952) and is of particular importance in cases of cardiac disease in which the onset of an arrythmia due to hypokalaemia might be disastrous. In the present study serum potassium levels as low as 3.1 m. eq./litre were present at the end of the first week in nine steroid treated patients without the onset of abnormal rhythm; the corresponding controls had normal levels. Although oral potassium was not given such treatment could easily have been administered, and would appear to be indicated as a precautionary measure.

Apart from these drawbacks steroid treatment was remarkably free from complication, probably because the drug was used in high doses for only a few days and then rapidly tailed off. The treatment programme was too transient for significant osteoporosis to result and no gastrointestinal haemorrhages or other more immediate complications were encountered. In particular, the incidence of congestive cardiac failure was the same irrespective of treatment

and steroids did not appear to affect its progress. Presumably the routine administration of a diuretic preparation, usually with digoxin, to all patients in failure masked any effect which the steroid might have had on fluid balance.

R E S U M E .

An overall assessment of the usefulness of steroid therapy, as seen in this study, gives disappointing results. Beneficial effects are limited to those who are so shocked that they are virtually moribund and to those with heart block; this excludes the vast majority of cases.

The acute inflammatory response in the dead myocardium is diminished in intensity and later dense scar formation is delayed so that the wall is liable to become excessively thin leading to aneurysm formation with the possible risk of heart rupture. The formation of a basophilic granular material in the peri-infarct zone is inhibited and this may result in the infarct being less well fixed into the adjacent living myocardium. There is a danger of producing a significant hypokalaemia though there is little upset of fluid balance. Serum glutamic oxalacetic transaminase levels are suppressed, masking a diagnostic rise at times.

It is concluded that steroids are not indicated in the routine treatment of myocardial infarction. Although in the short term there are benefits in the presence of acute shock and of acute complete heart block, in the long term there is the danger of aneurysm formation and perhaps of heart rupture.

(c) CONCLUSIONS.

(1) (a) Steroid therapy did not influence significantly the mortality rate in severe myocardial infarction.

(b) The process of resolution and of repair in myocardial infarcts was modified, the formation of a strong scar was delayed, and the formation of a periinfarct fibrous meshwork was diminished.

(c) Steroids induce hypokalaemia in 56% of patients at the end of the first week though this was not associated with any arrythmia.

(d) The rise in serum glutamic oxalacetic transaminase was checked and sometimes a diagnostic rise was not attained.

(2) (a) The pathological changes give rise to fears that steroid therapy might lead to thinning of the healed infarct, aneurysm formation, or late cardiac rupture.

(b) The prevelance of hypokalaemia after a week underlines the importance of electrolyte estimation in such patients.

(c) If steroids were used in clinically mild cases, S. G. O. T. estimation might be rendered valueless since small rises in the serum enzyme levels are masked.

THE EFFECT OF STEROIDS IN THE TREATMENT OF ACUTE MYOCARDIAL INFARCTION WITH PARTICULAR REFERENCE TO HISTOLOGICAL CHANGES

- by -

IAIN DAVID OWEN FREW.

V O L U M E II.

I N D E X .

VOLUME TWO.

TABLE 37.

Relationship between coronary prognostic index and death rate at 28 days on all cases of myocardial infarction admitted during 1961-63.

C.P.I.	No. of Cases			Average Age	Survival at 28 days (%)
	Total	Male	Female		
1	3	3	-	45	100
2	9	9	-	50	100
3	13	9	4	54	100
4	26	22	2	52	100
5	30	24	6	55	97
6	21	15	6	57	100
7	43	37	6	59	95
8	37	25	12	57	95
9	40	31	9	61	90
10	34	21	13	60	91
11	37	31	6	59	89
12	37	23	14	63	78
13	29	21	8	58	79
14	41	28	13	65	73
15	28	16	12	64	57
16	32	26	6	63	56
17	20	9	11	62	55
18	27	18	9	64	56
19	25	9	16	68	44
20	18	11	7	63	34
21	18	12	6	64	39
22	11	9	2	71	46
23	12	10	2	66	25
24	3	2	1	68	33
25	6	3	3	60	33
26	-	-	-	-	-
27	1	-	1	60	0
28	3	3	-	67	0

TABLE 38.

Distribution of prognostic index scores for age and sex in steroid treated and in control patients.

Series	Scores			
	0	1	2	3
Steroid (A-E)	7	7	25	36
Control (F)	4	2	25	34

TABLE 39.

Distribution of prognostic index scores for previous history in steroid treated and in control patients.

Series	Scores			
	0	1	3	6
Steroid (A-E)	17	9	26	22
Control (F)	3	6	26	35

TABLE 40.

Distribution of prognostic index scores for shock in steroid treated and in control patients.

Series	Scores			
	0	1	5	7
Steroid (A-E)	0	13	41	19
Control (E)	9	17	27	16

T A B L E 4 1 .

Distribution of prognostic index scores for failure in steroid treated and control patients.

Series	Scores		
	0	1	4
Steroid (A-E)	4	8	62
Control (F)	4	3	62

T A B L E 4 2 .

Distribution of prognostic index scores for E.C.G. change in steroid treated and control patients.

Series	Scores			
	0	1	3	4
Steroid (A-E)	0	1	8	62
Control (F)	0	2	9	59

T A B L E 4 3 .

Distribution of prognostic index scores for rhythm in steroid treated and control patients.

Series	Scores		
	0	4	
Steroid (A-E)	17	54	
Controls (F)	27	42	

TABLE 44.

Case Numbers of patients showing heart block on admission.

PARTIAL BLOCK	(11 Cases)							
Steroid Treated.	A.1,	A.28,	A.41,	A.45,	C.6,	C.10.		
Controls.	F. 10,	F.23,	F.34,	F.37,	F.45.			
POTENTIAL BLOCK.	(9 Cases)							
Steroid Treated	A.17,	A.18,	A.35,	A.39,	A.48,	C.3,	C.4,	C.8.
Controls	F.4,	F.16,	F.53,	F.67,	F.68.			

TABLE 45.

CELL COUNTS (SERIES A - E).

Number of cells / 2 sq. mm. tissue section.

Day After Infarct	Case No.	Neut.	Eos.	Lympho.	Plas.	Mac.	Mac(H)	Fib.
1	A11	90	-	45	-	-	-	-
1	A32	230	-	20	-	-	-	-
1	A40	310	-	-	-	-	-	-
2	A 4	1660	-	55	-	30	-	30
2	A28	1670	-	80	-	50	-	15
2	A29	920	-	-	-	-	-	-
2	A45	950	-	60	-	-	-	-
3	A35	1290	-	80	-	50	-	30
3	A38	1350	-	100	-	75	4	40
3	D 4	990	-	60	-	-	-	-
3	D 2	1280	-	50	-	15	-	5
5	A27	830	-	160	-	120	9	105
6	A22	750	-	240	-	90	-	75
7	A10	640	-	150	-	150	-	230
11	A21	130	15	410	6	210	44	685
11	C 7	70	-	480	13	195	87	660
12	A12	80	-	410	-	180	44	740
13	C10	30	35	380	-	85	65	880
16	C 1	10	-	390	-	75	6	805
16	A31	20	20	340	-	80	45	870
22	C 2	-	-	170	-	10	87	605
39	B 2	-	-	30	-	-	6	230
107	D 4	-	-	-	-	-	-	190
240	C 6	-	-	-	-	-	-	140

TABLE 46.

CELL COUNTS (SERIES F).

Number of cells / 2 sq. mm. tissue sections.

Day after infarct	Case No.	Neut.	Eos.	Lymphs.	Plas.	Mac.	Mac(H)	Fib.
1	F24	860	-	30	-	-	-	-
1	F50	450	-	20	-	50	-	-
1	F68	50	-	40	-	-	-	-
2	F25	2950	-	145	-	70	-	-
2	F53	3050	-	45	-	15	-	30
2	F63	3250	-	155	-	50	-	-
2	F 9	3100	-	150	-	40	-	-
3	F32	2510	-	140	15	80	10	30
3	F49	2820	-	70	-	70	-	45
3	F55	2910	-	160	1	80	27	40
3	F61	2210	-	50	-	40	6	40
4	F48	1250	-	205	-	130	28	120
6	F39	140	-	290	-	150	14	345
6	F52	155	-	200	-	140	62	385
8	F23	30	-	345	-	230	59	460
8	F59	50	-	365	-	240	27	495
12	F35	40	35	345	-	80	68	540
14	F10	60	-	370	-	70	30	580
22	F13	30	-	85	-	30	21	435
29	F27	-	-	70	-	15	-	175
30	F 6	10	-	-	-	-	25	205
40	F 7	-	-	15	-	-	-	130

TABLE 47.

CELL COUNTS (SERIES H).

Number of cells / 2 sq. mm. of tissue section.

Day after infarct	Case No.	Neut.	Eos.	Lymphs.	Plas.	Mac.	Mac(H)	Fib.
1	H 1	600	-	20	-	45	-	-
1	H 2	80	-	-	-	-	-	-
1	H 3	430	-	-	-	-	-	-
1	H 4	750	-	40	-	40	-	-
2	H 5	2885	-	60	-	60	-	-
3	H 6	2385	-	120	-	80	-	10
3	H 7	2425	-	60	-	65	-	-
4	H 8	1845	-	80	-	80	-	-
4	H 9	1805	-	160	6	115	9	100
5	H10	850	-	215	4	60	-	245
6	H11	205	-	280	-	140	-	230
8	H12	70	8	340	-	215	10	510
10	H13	60	-	520	-	130	103	600
14	H14	10	30	420	16	90	28	575
15	H15	-	-	400	-	75	70	580
18	H16	50	-	230	-	45	40	560
19	H17	-	-	320	-	30	52	590
21	H18	25	-	315	-	20	158	490
27	H19	-	-	135	-	60	17	305

TABLE 48.

CELL COUNTS (SERIES J).

Number of cells / 2 sq. mm. tissue section.

Day after infarct	P.M. No.	Neuts.	Eos.	Lymphs.	Plas.	Mac.	Mac(H)	Fib.
1	4/58	395	-	50	15	30	-	-
1	210/58	45	-	-	-	-	-	-
1	31/62	35	-	-	-	-	-	-
1	104/63	910	-	55	-	35	-	-
1	155/63	65	-	-	-	-	-	-
1	156/63	115	-	15	-	-	-	-
1	243/63	15	-	-	-	-	-	-
2	97/59	3345	-	60	-	25	-	-
2	99/60	3005	-	155	-	50	-	-
3	179/58	2645	-	50	-	60	-	-
3	190/58	2830	-	125	-	40	-	-
3	21/59	2420	-	190	-	60	-	-
3	182/63	3020	-	40	-	50	-	-
4	206/63	1720	-	150	-	90	-	40
6	35/63	255	-	260	-	120	35	360
6	109/63	160	6	215	-	150	-	310
6	38/64	210	-	230	-	130	40	340
7	238/58	80	-	305	17	100	40	365
7	11/60	160	-	390	2	90	65	490
9	40/58	50	-	410	-	170	-	510
9	106/58	30	-	380	-	280	-	550
10	96/59	40	-	400	8	130	50	640
10	127/62	6	-	440	-	160	20	575
12	92/59	18	15	395	6	149	65	645
12	173/60	50	-	425	-	90	10	545
12	49/64	10	-	360	-	90	150	580
13	285/62	45	-	450	6	105	30	620

Day after infarct	P.M. No.	Neuts.	Eos.	Lymphs.	Plas.	Mac.	Mac(H)	Fib.
14	194/60	10	10	400	12	100	10	620
14	210/62	45	10	405	-	40	80	590
14	227/62	20	-	375	17	110	40	610
17	127/64	-	-	315	-	45	105	570
20	27/63	25	-	260	-	20	115	550
23	11/63	-	-	80	-	-	75	415
33	M/63	-	-	65	-	-	-	225
41	M/59	-	-	30	-	5	25	150
91	165/62	-	-	75	-	-	-	105
8 months	196/63	-	-	-	-	-	-	65

TABLE 49.

CELL COUNTS on SERIES K.

Number of cells / 2 sq. mm. tissue section.

Day after infarct	Hospital	P.M. No.	Neuts.	Lymph.	Plas.	Mac.	Mac(H)	Fib.
10	Glasgow Western	B3735	150	370	4	250	11	470
1	Gartloch	103/63	130	-	-	-	-	-
2	Gartloch	80/63	1215	60	-	-	-	-
3	Gartloch	97/63	1400	85	-	40	-	50
16	Gartloch	74/63	40	335	11	75	32	850
12	Dumfries	100/63	105	480	-	125	39	635
17	Dumfries	138/63	15	230	-	15	55	840
6	Aberdeen City	43/63	750	205	-	105	-	70

Table 50

FOLLOW UP CATEGORIES IN STEROID TREATED PATIENTS.

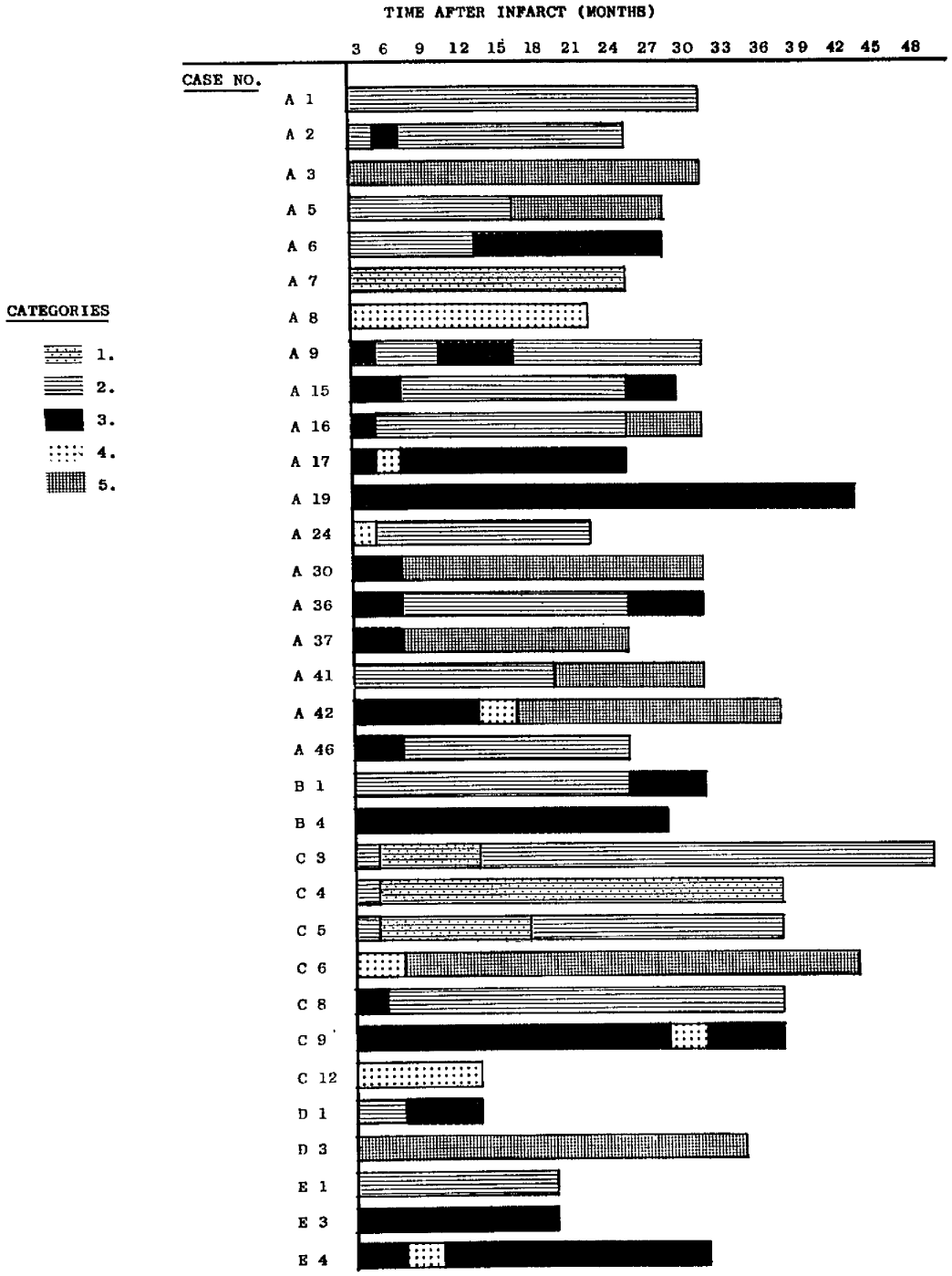
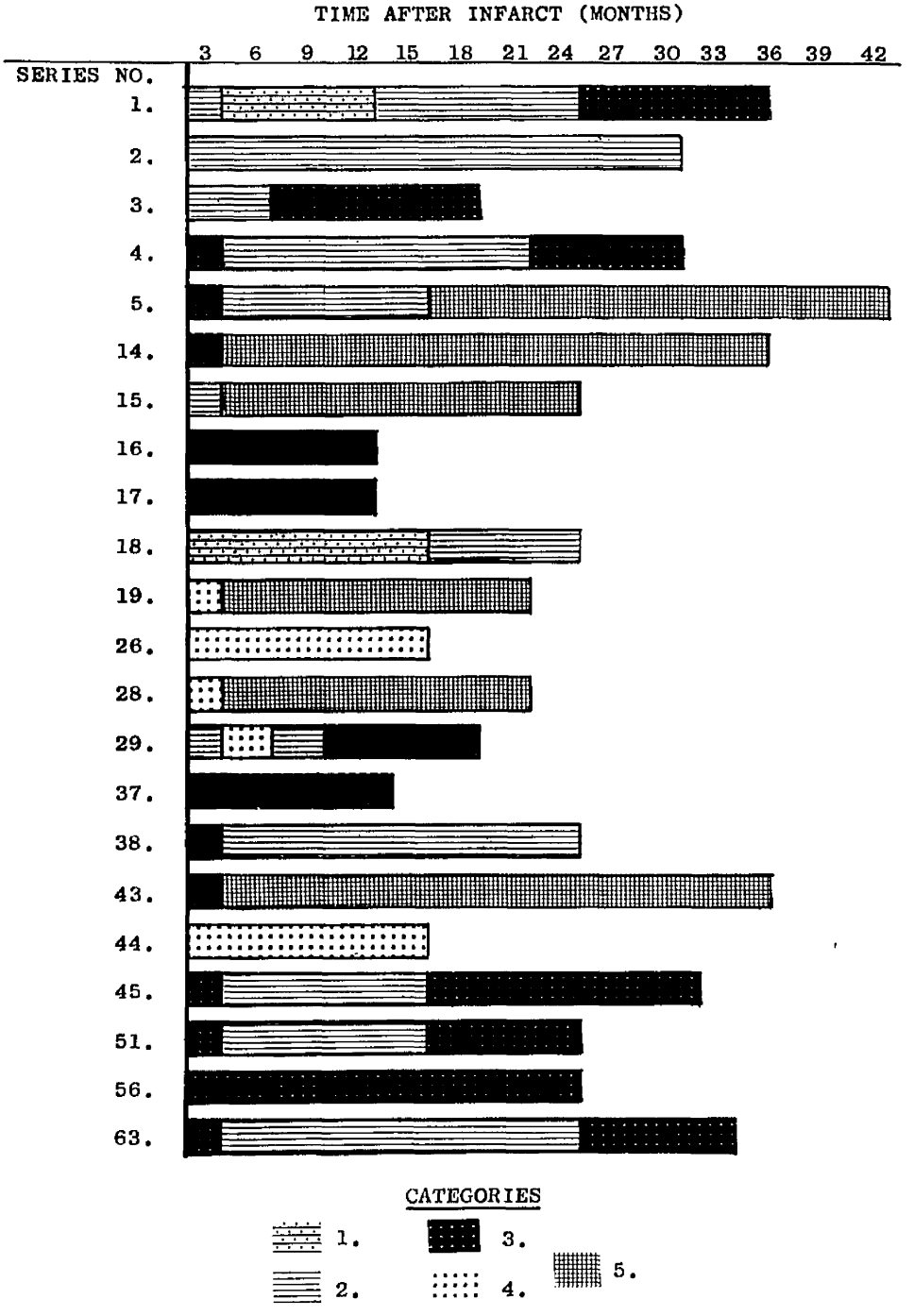


Table 51

FOLLOW UP CATEGORIES IN CONTROL PATIENTS (SERIES F).



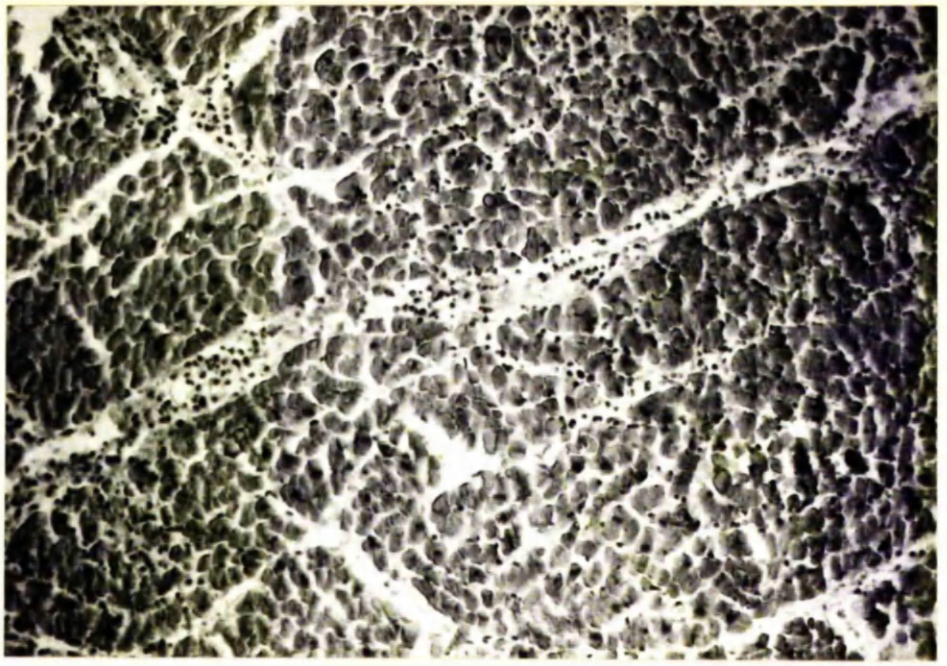


FIG. 22

Case No. A28, steroid treated, 2nd. day.
(H.& E. x 150)

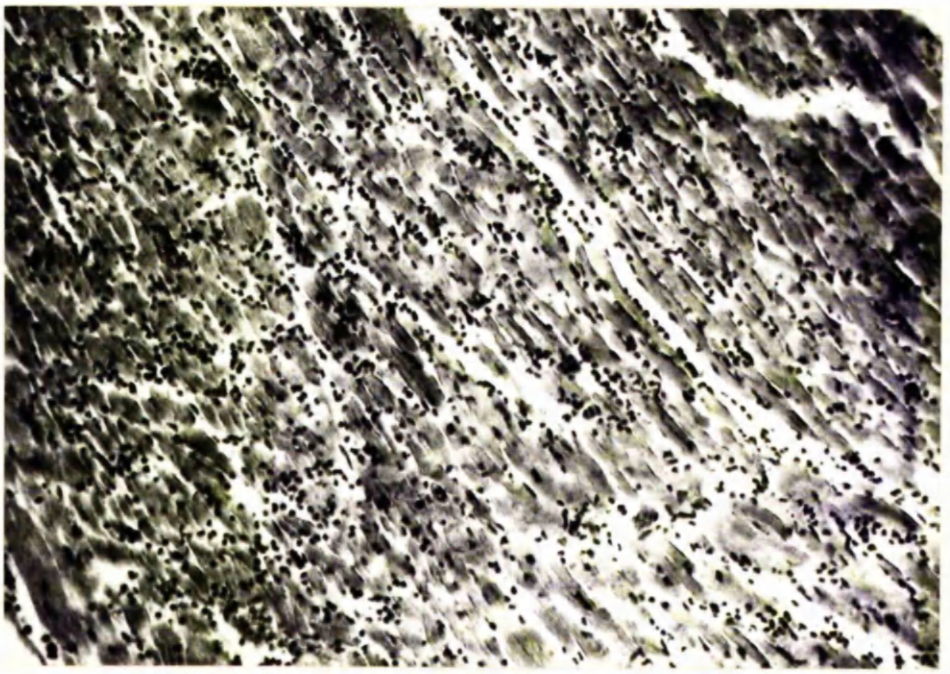


FIG. 23

Case No. F9, control, 2nd. day.

(H. & E. x 150)

The neutrophil infiltrate is much less intense in the steroid treated case compared with the control.

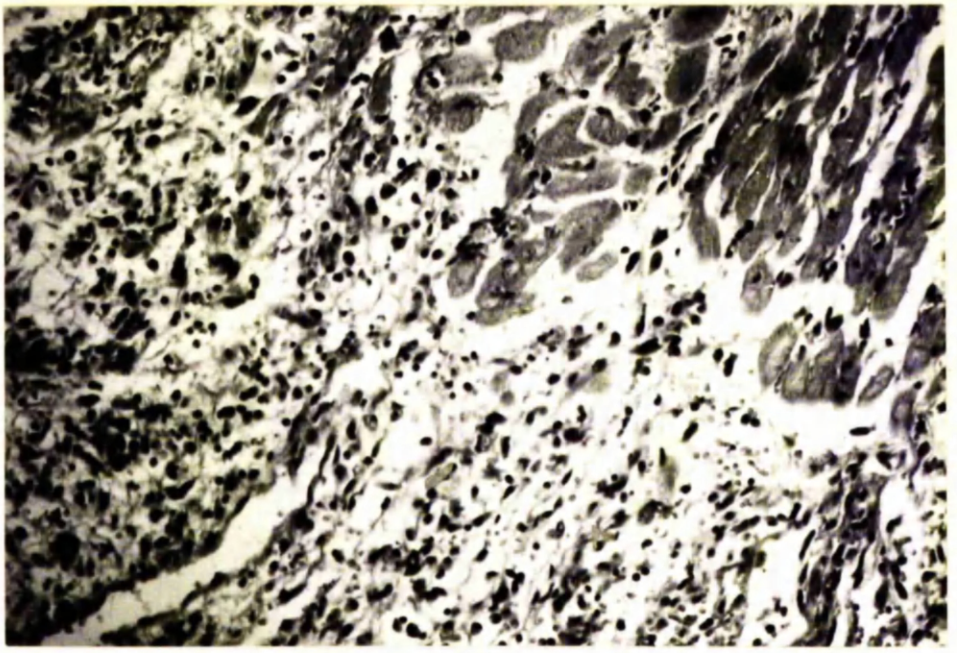


FIG. 24
Case No. A10, steroid treated, 7th. day.
(H. & E. x 320)

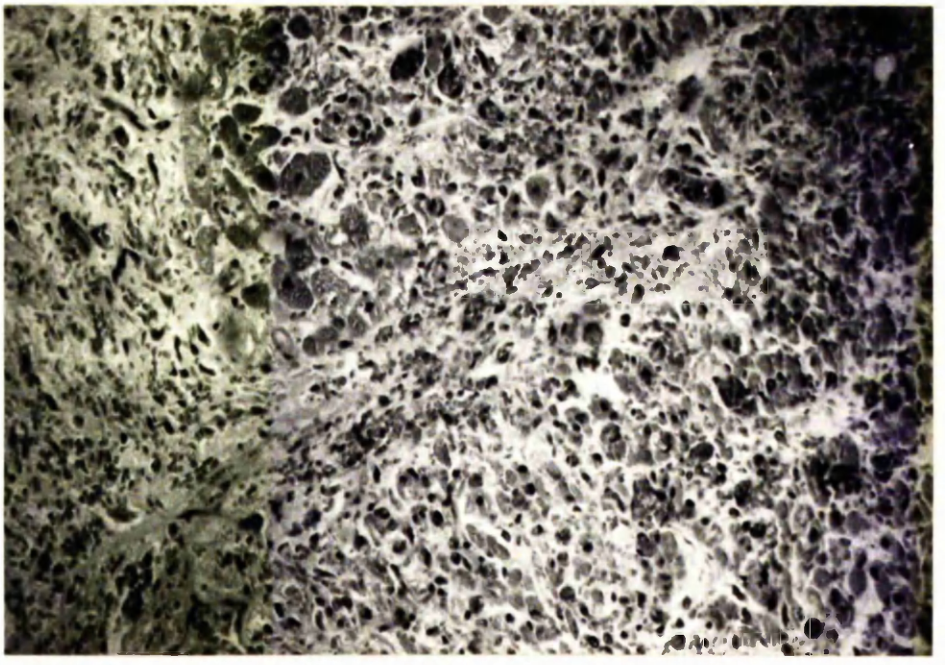


FIG. 25
Case No. F23, control, 8th. day.
(H. & E. x 150)

Phagocytosis is advanced in both hearts, irrespective of treatment.

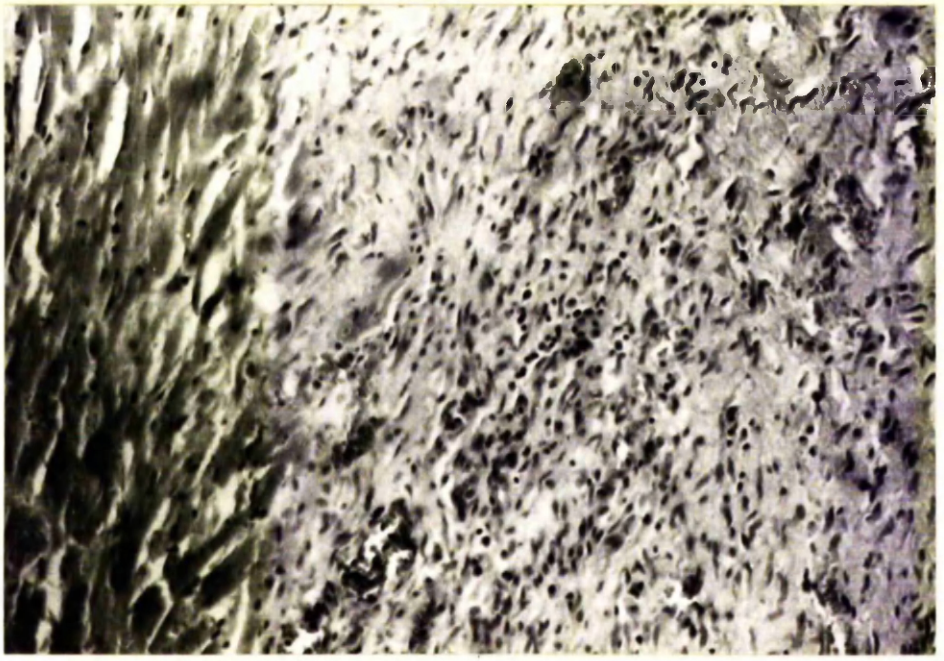


FIG. 26

Case No. A21, steroid treated, 11th. day.

(H. & E. x 150)

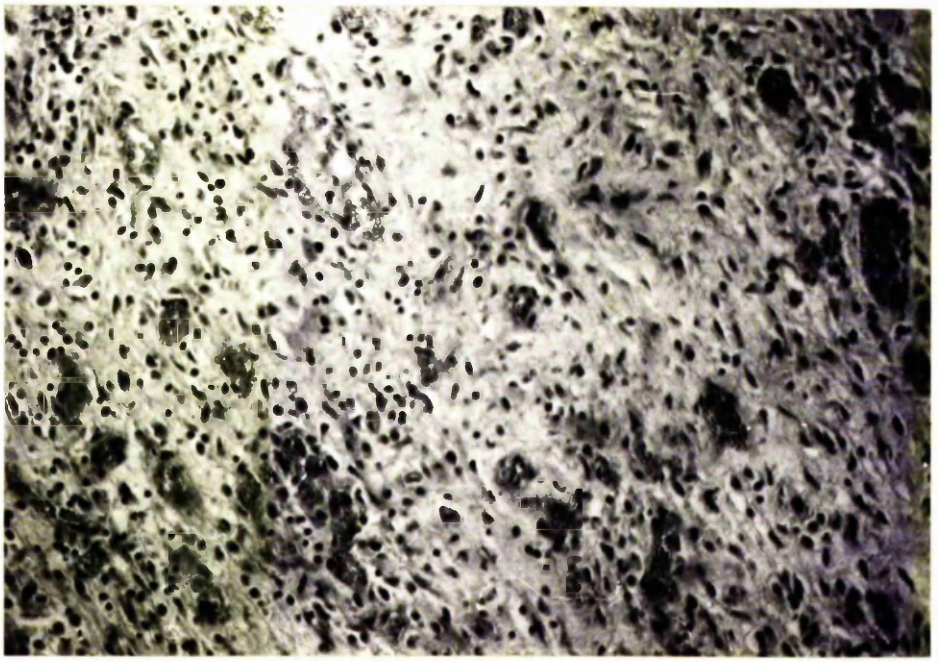


FIG. 27

Case No. H13, supplementary control group, 10th. day. (H. & E. x 150)

In each case the infarct has been replaced by vascular fibrous tissue; commencing hyalinisation of this tissue is less apparent in the steroid treated heart compared with the control.

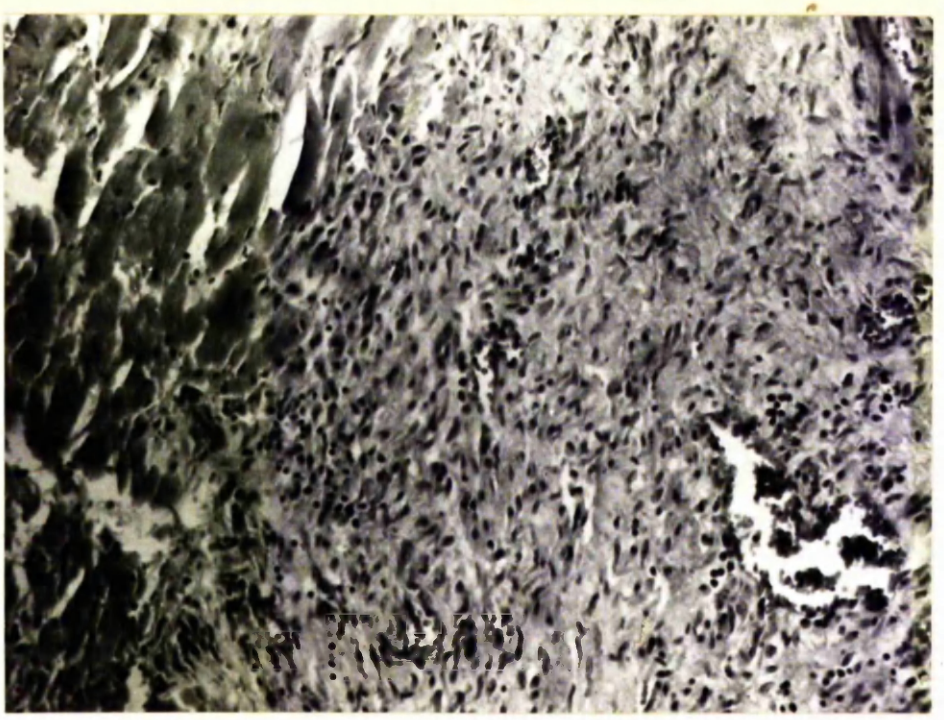


FIG. 28

Case No. C10, steroid treated, 13th. day.

(H. & E. x 150)

FIG. 29

Case No. F35, control, 12th. day.

(H. & E. x 150)

Hyalinisation is more marked in the control heart compared with that treated with steroids.

FIG. 30

Case No. A31, steroid treated, 16th. day.
(H. & E. x 180)

FIG. 31

Case No. F10, control, 14th. day.
(H. & E. x 150)

The reduction in the number of fibroblasts and the hyalinisation of the healing infarct is much more apparent in the control case than that given steroids.

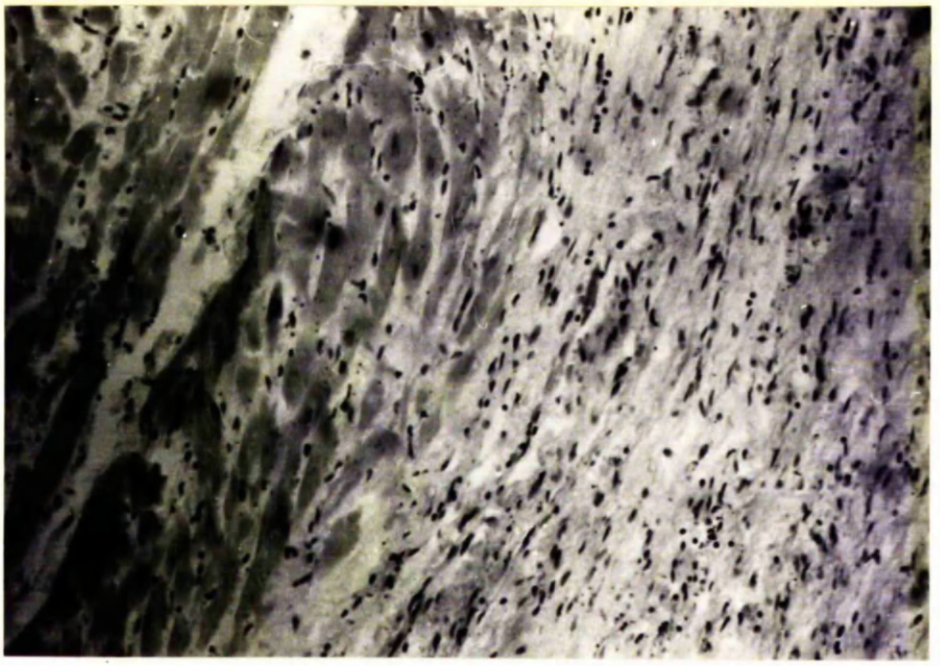


FIG. 32

Case No. C2, steroid treated, 22nd. day.
(H. & E. x 150)

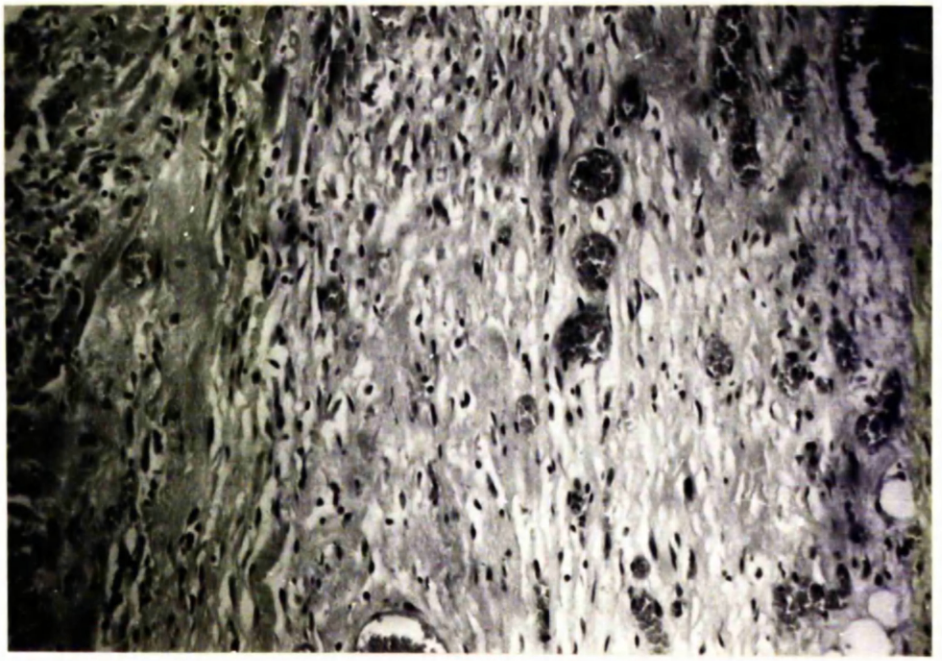


FIG. 33

Case No. F13, control, 22nd. day.
(H. & E. x 150)

The steroid treated infarct is less hyalinised than the control.

FIG. 34

Case No. D4, steroid treated, 3 months old.
(H. & E. x 220)

FIG. 35

Case No. 165/62, Series J, 3 months old.
(H. & E. x 220)

The steroid treated infarct is more vascular and less hyalinised than the control.

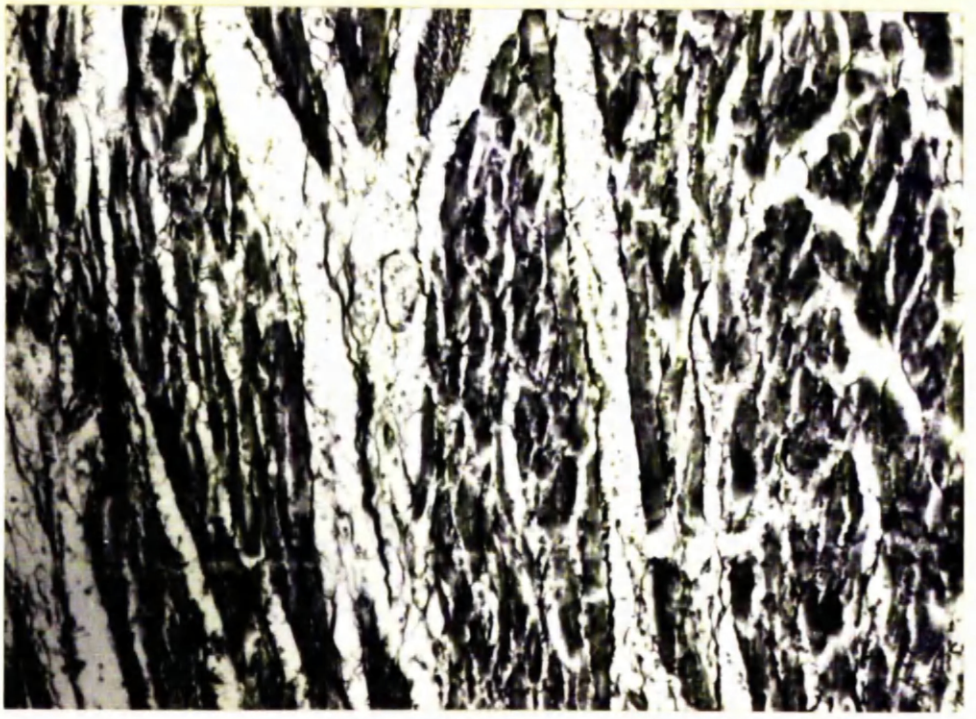


FIG. 36

Case No. A36, steroid treated, 3rd. day.
(Retic. x 150)

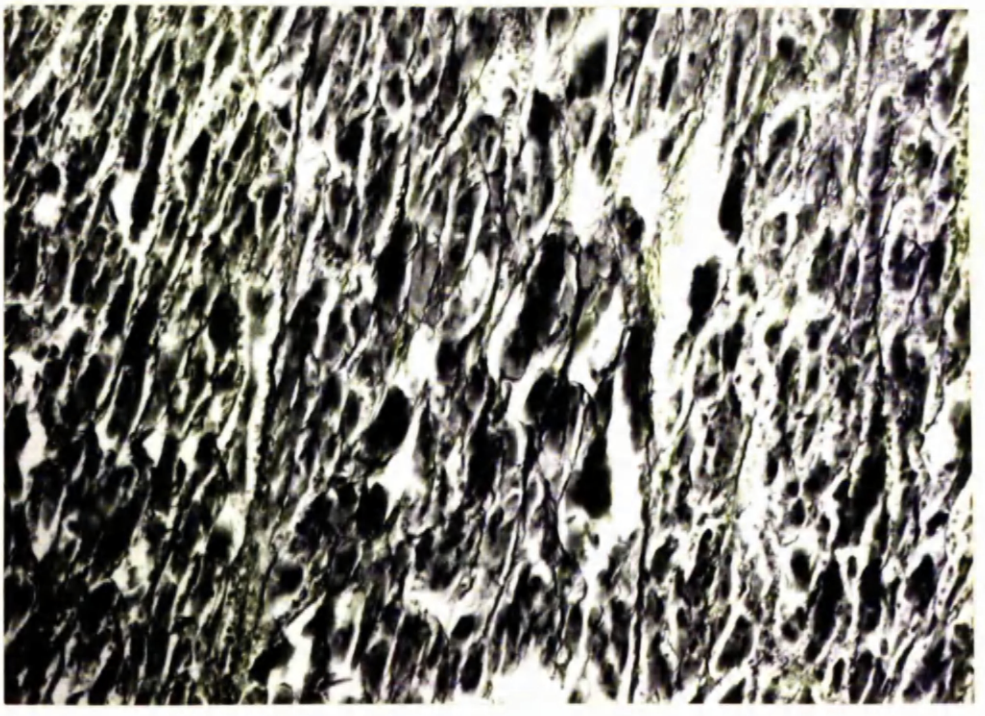


FIG. 37

Case No. F49, control, 3rd. day.
(Retic. x 150)

The reticulin pattern is only slightly disrupted and no difference can be seen between the two hearts.

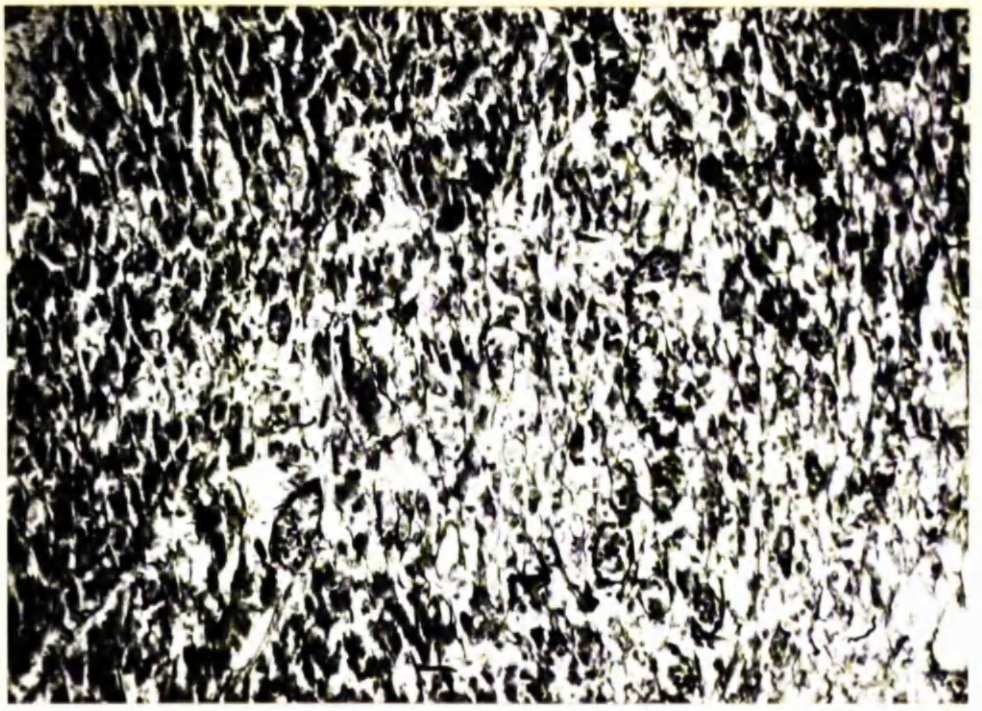


FIG. 38

Case No. A22, steroid treated, 6th. day.

(Retic. x 150)

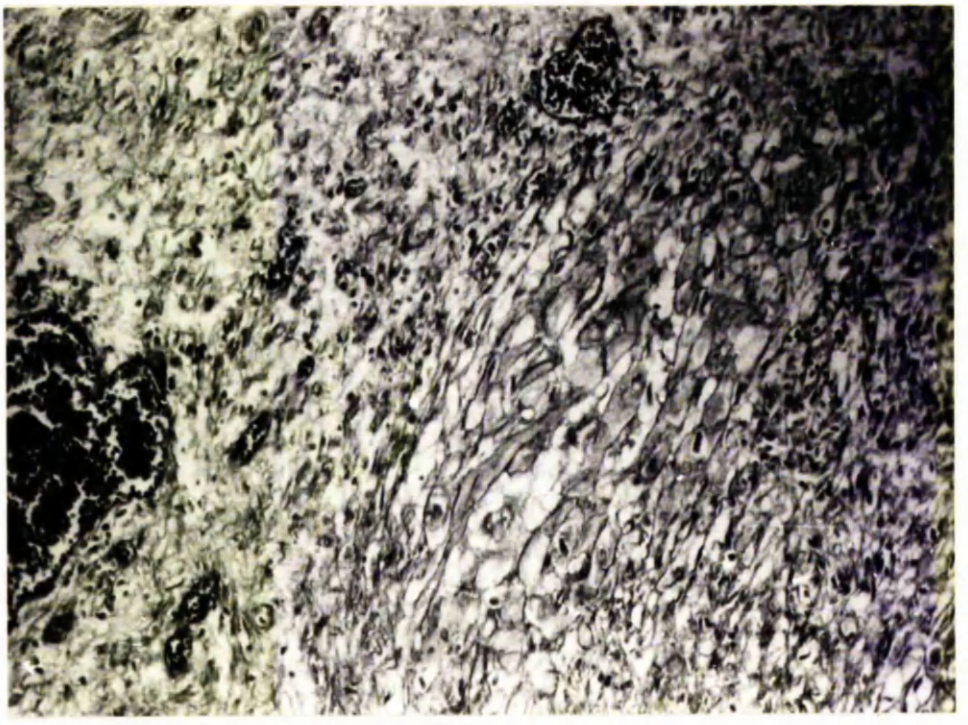


FIG. 39

Case No. F52, control, 6th. day.

(Retic. x 150)

The original reticulin network has been replaced by a fine meshwork of new fibres; no difference is seen between the two cases.

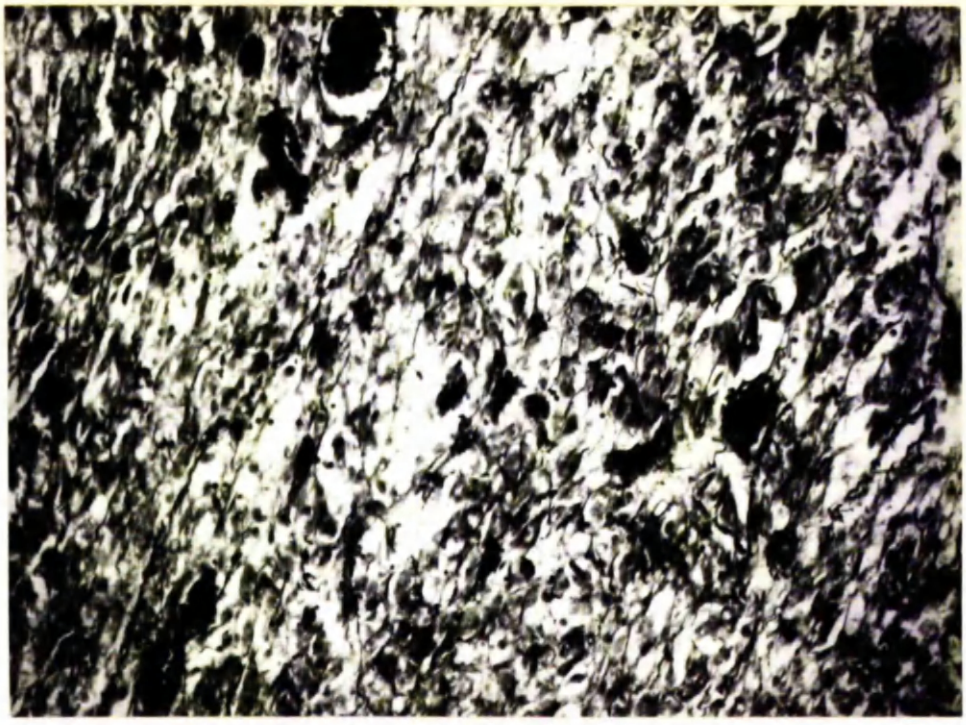


FIG. 40

Case No. C7, steroid treated, 11th. day.

(Retic. x 150)

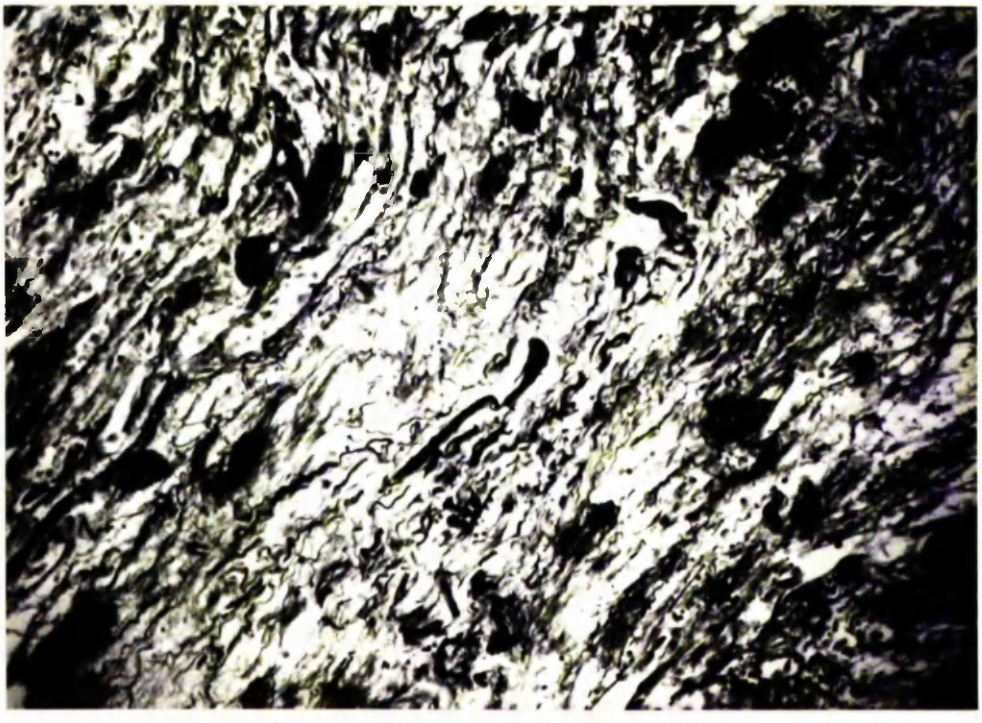


FIG. 41

Case No. F35, control, 12th. day.

(Retic. x 150)

In the control infarct thick reticulin fibres lie parallel to each other; in contrast a much finer meshwork persists in the steroid treated infarct.

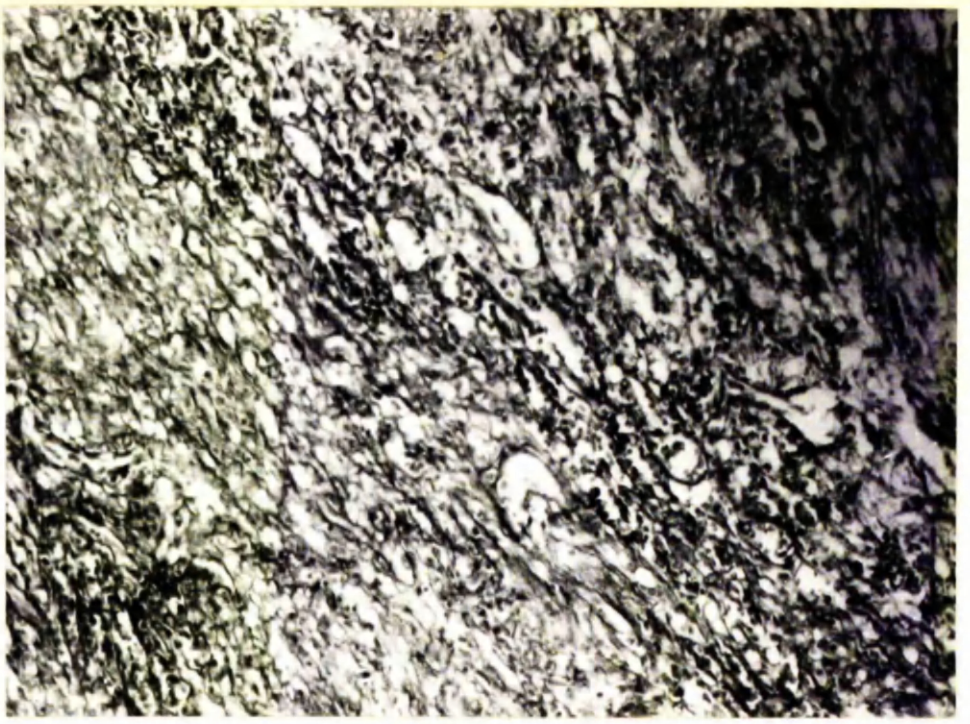


FIG. 42

Case No. A31, steroid treated, 16th. day.

(Retic. x 150)

FIG. 43

Case No. F10, control, 14th. day.

(Retic. x 150)

A dense meshwork of thick reticulin fibres persists in the steroid treated infarct; in contrast the meshwork is much less dense in the control infarct.

FIG. 44

Case No. D4, steroid treated, 3 months old.

(Retic. x 150)

FIG. 45

Case No. 165/62, Series J, 3 months old.

(Retic. x 150)

A number of coarse reticulin fibres persist in the steroid treated infarct but the control infarct contains virtually no fibres.

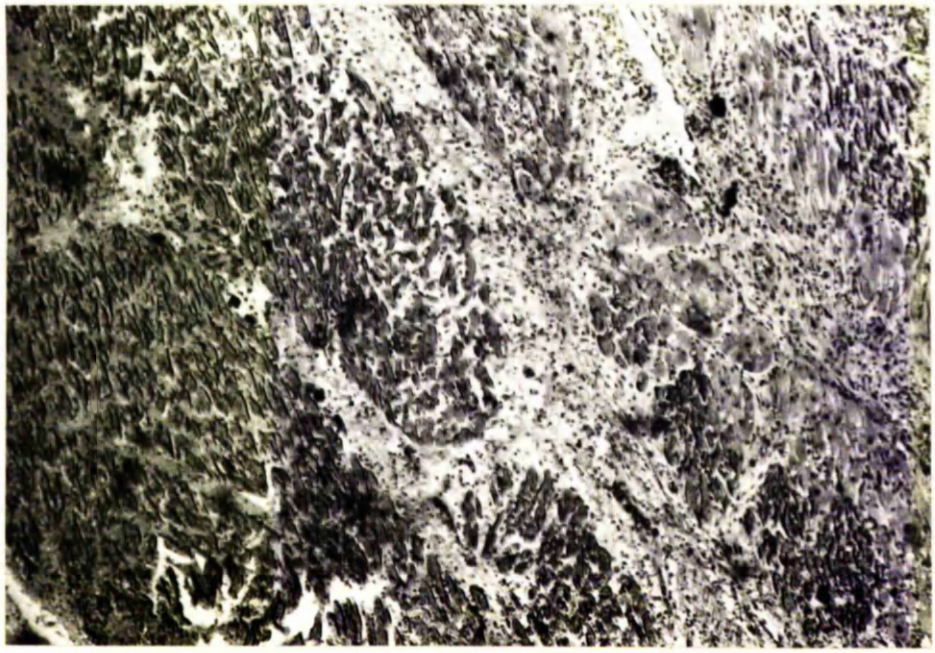


FIG. 46

Case No. F39, control, 6th. day.

(H. & E. x 50)

Recent infarct on right. Between the surviving myocardial fibres on the left is abundant granular basophilic material.

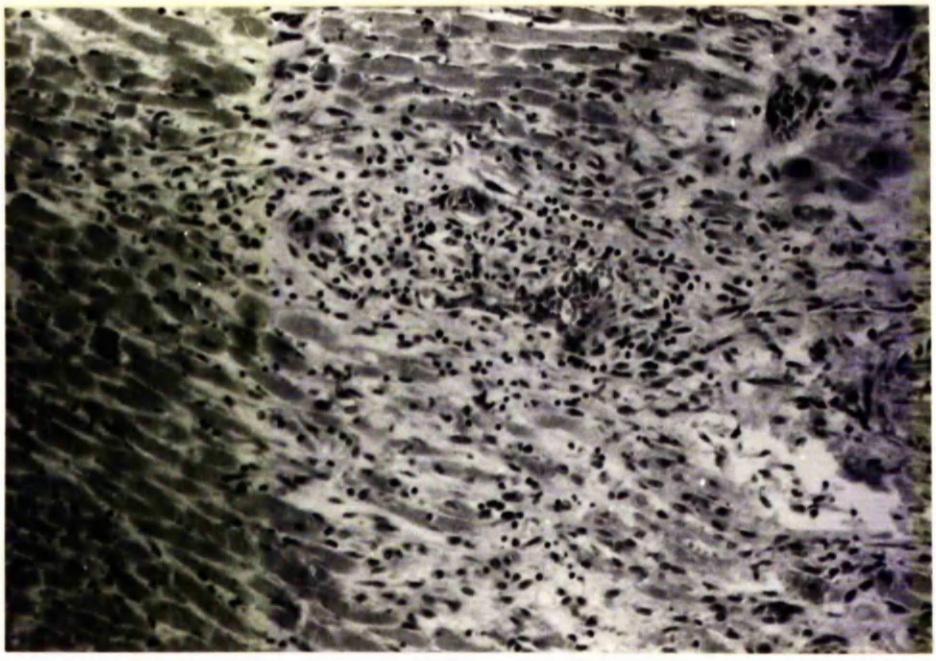


FIG. 47

Case No. A12, steroid treated, 12th. day.
(H. & E. x 150)

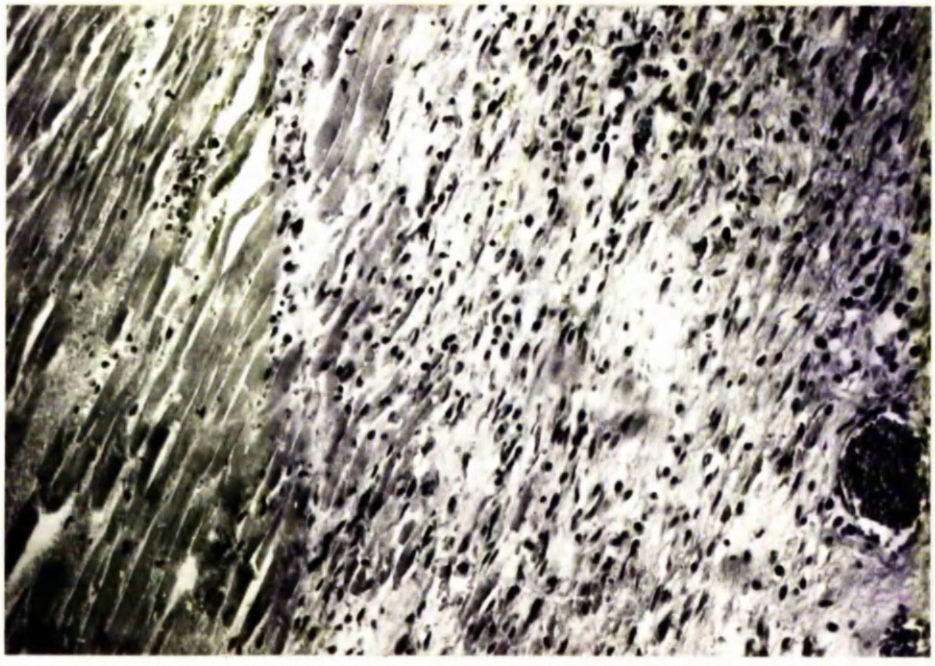


FIG. 48

Case No. F35, control, 12th. day.
(H. & E. x 150)

Granular material occupies the space between the surviving fibres (left) in the control infarct but none is seen in the steroid treated case (left).

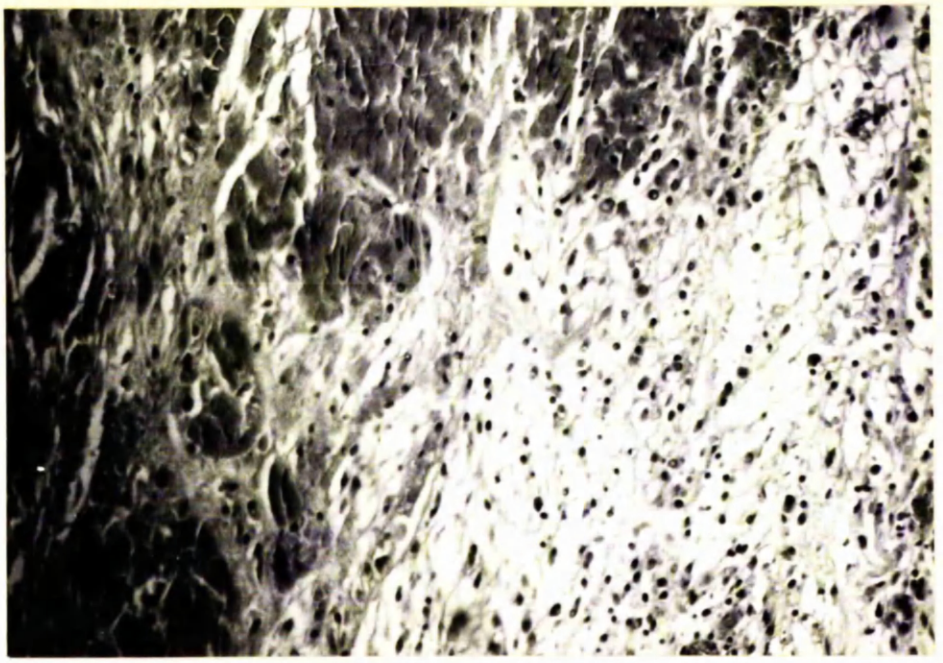


FIG. 49
Case No. F13, control, 22nd. day.
(H. & E. x 150)

The basophilic material between the myocardial fibres (left) contains occasional macrophages, leucocytes, and fibroblasts.

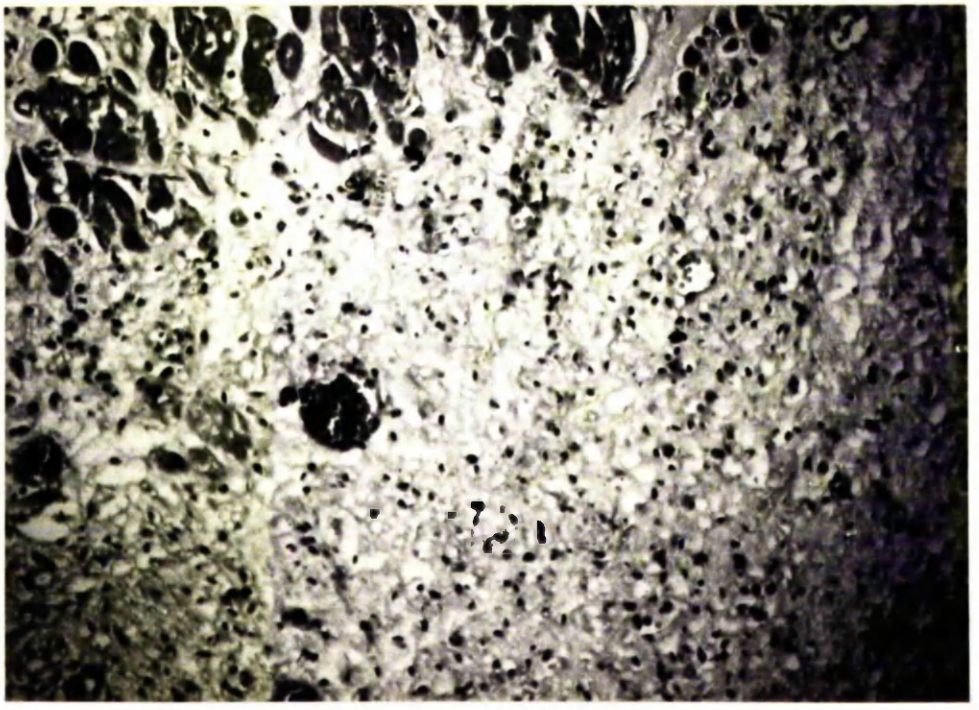


FIG. 50
Case No. F27, control, 29th. day.
(H. & E. x 150)

Fibrous tissue has replaced the basophilic granular material between the surviving myocardial cells (top left).

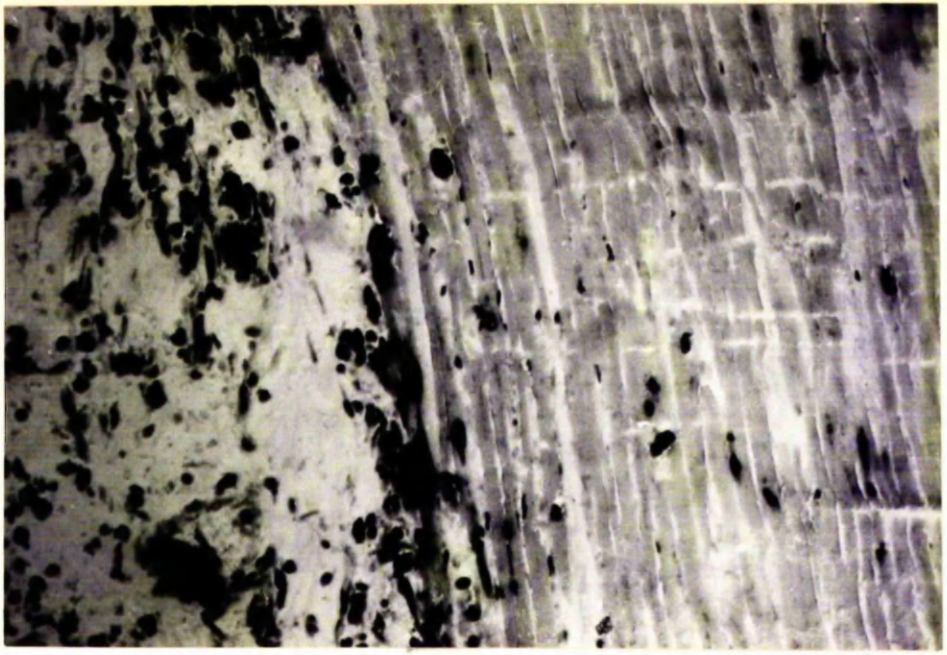


FIG. 51

Case No. A22, steroid treated, 6th. day.

(Gomori - no nuclear stain, x 250)

Moderate amounts of acid phosphatase are present in the infarct (left) with virtually none in the adjacent myocardium (right).

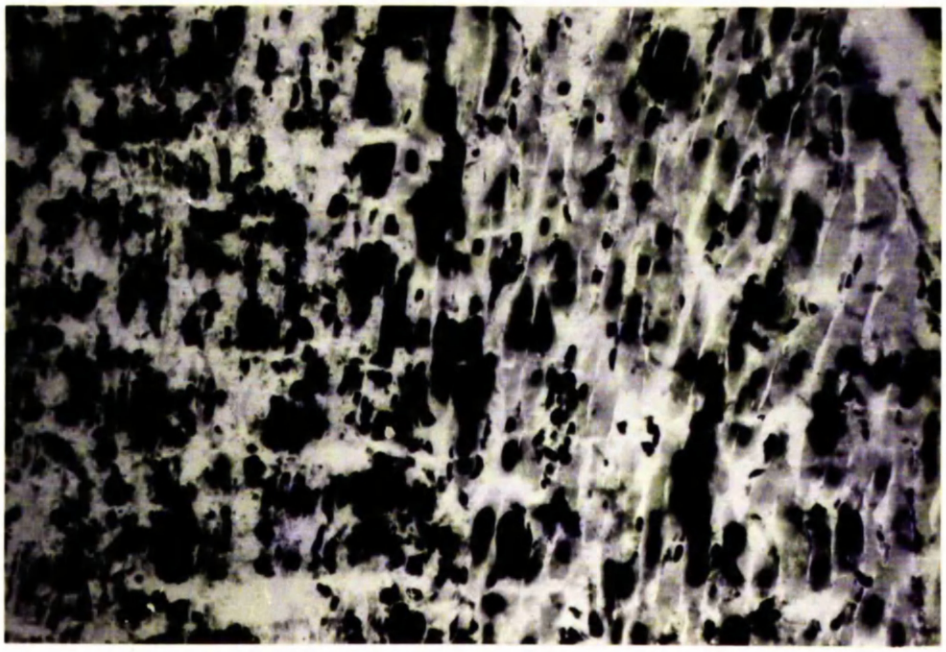


FIG. 52

Case No. F39, control, 6th. day.

(Gomori - no nuclear stain, x 250)

Large amounts of acid phosphatase are present in the infarct (left) and adjacent myocardium (right).

S U P P L E M E N T S T O C H A P T E R T W O

TISSUE PROCESSING AND CUTTING METHODS.

(1) PREPARATION OF CRYOSTAT SECTIONS.

Fresh tissue in blocks 10 x 10 x 3 mm. approximately was quenched in liquid nitrogen as soon as possible after removal from the body, and was then frozen to a metal stud already cooled by embedding in CO_2 snow. The blocks were now ready for cutting, and the cryostat used was a Slee instrument incorporating a Cambridge rock microtome. At least six sections at 6μ thickness were cut from each block, which was then removed from the stud and transferred to formal acetate fixative prior to undergoing paraffin processing.

(2) PARAFFIN PROCESSING AND CUTTING.

Portions of tissue measuring up to 15 x 15 x 4 mm. were fixed in an 8% solution of formalin in 4% aqueous sodium acetate for 16-18 hours and then in saturated aqueous mercuric chloride with 10% formalin for a further 24 hours. Thereafter the blocks were transferred to an automatic tissue processor in which they underwent this regime:-

(a) 3 changes of 8% phenol in absolute industrial methylated spirit, 74 o.p., (99% ethyl alcohol), (3, 2 and 2 hours respectively).

(b) 3 changes of absolute industrial methylated spirit (1 hour in each).

(c) 2 changes of methcol benzene (1 and 2 hours respectively).

(d) 2 changes of benzene (2 hours in each).

(e) 2 changes of paraffin at 54°C (2 and 3 hours respectively).

The tissues were then blocked out in paraffin at 56°C and subsequently were cut on a rotary microtome at 6μ.

CLINICAL SUMMARIES and AUTOPSY

Descriptions of patients included in

Series A - H inclusive.

A pink sheet separates each series.

The results shown from serum transaminases are in Cabaud Units, and those for serum electrolytes are in M. eq./litre.

CASE NO. A. 1. A. M. Female, 46 years. Registration No. 176560

Admitted 29.12.61, Discharged 30.1.62 (32 Days)

	A.S.	Ph.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX.	0	3	5	1	4	4	17

Admitted shocked, B.P. 60/?, with basal crepitations but no peripheral oedema. Pulse 90 - 105/minute irregularly irregular due to ventricular extrasystoles. Past history of effort dyspnoea for a few months.

E.C.G. Anteroseptal infarct, Q S waves in leads I, Avl, V1 - V5. P - R interval on admission 0.24 seconds, 8th. day 0.16 seconds, 28th. day 0.18 seconds.

TRANSAMINASES	Day	1	2	3	5
	S.G.O.T.	73	40	38	35
	S.G.P.T.	54	57	72	50

HAEMOGLOBIN 96% (cyan method)

PROGRESS Improved rapidly with bed rest and sedation, and after 6 hours B. P. was 130/70, pulse 85/minute regular. On 3rd. day B. P. was 110/40 and thereafter around 120-130/75. Subsequent progress uneventful and remained well for next 2½ years having only slight exertional dyspnoea (Category II).

CASE NO. A. 2. J.W. Male, 50 years. Registration No. 186911

Admitted 13.5.62, Discharged 17.6.62. (36 Days)

	A.S.	P.H.	S.	F.	R.	E.C.G.	TOTAL.
PROGNOSTIC INDEX	0	0	5	4	4	4	17

Admitted shocked, B. P. 100/65, with peripheral oedema and basal crepitations. Pulse 50/minute regular with bursts of auricular fibrillation producing a heart rate of 160/minute. No relevant previous history.

E.C.G. Posterior infarct with Q S. waves in leads II, III, Avf, P-R interval on admission 0.20 seconds, thereafter steady at 0.16 seconds.

TRANSAMINASES	Day	1	2	3	5
	S.G.O.T.	103	135	56	31
	S.G.P.T.	62	23	47	36

HAEMOGLOBIN 92% (cyan method)

PROGRESS Rapid improvement following the administration of procaine amide and digitalis, B.P. soon 130/78, and pulse 75/minute almost regular. Fibrillation not seen after 3rd. day. Subsequent progress uneventful; remained well for next 2 years, with only slight effort dyspnoea (Category II) except at 6 months when he had a brief attack of ventricular extrasystoles.

CASE NO. A. 3. M. McM. Female, 62 years. Registration No. 197064

Admitted 30.9.62, Discharged 9.11.62. (39 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	6	1	-	4	4	17

Slight shock on admission, B.P. 100/70, Pulse 80-110/minute, irregular due to bursts of auricular fibrillation. Failure not present. Myocardial infarct four months before.

E.C.G. Between bursts of auricular fibrillation there was an anteroseptal infarct pattern with Q S waves in leads I, Avl, Vl - V3, and P-R interval was at all times 0.16 seconds.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	20	23	20
	S.G.P.T.	12	10	40

HAEMOGLOBIN 84% (cyan method).

PROGRESS Recovery from shock was speedy, B.P. soon 120/80 and stabilised later around 110/80. Fibrillation persisted intermittently despite administration of digoxin and quinidine, and was present continuously from 13.10.62. After discharge irreversible congestive failure developed and death occurred on 27.12.62. There was no autopsy.

CASE NO. A. 4. M. B. Female, 55 years. Registration No. 182897

Admitted 28.3.62, Died 30.3.62, (2 Days)

	A.S.	P.H.	S.	F.	R.	E.C.G.	TOTAL.
PROGNOSTIC INDEX	3	1	5	4	-	4	17

Admitted with moderate shock, B. P. 100/60, Pulse 100/minute and slight ankle oedema. Past history of angina for 3 years.

E.C.G. Posterior infarct, Q.S. waves affected in leads III Avf. P-R interval 0.19 seconds.

TRANSAMINASES	Day	1
	S.G.O.T.	57
	S.G.P.T.	38

PROGRESS Despite treatment with hydrocortisone, digoxin and mersalyl, the blood pressure fell slowly, the pulse rose to 120/minute and congestive failure developed. Death occurred on the 2nd. day.

POSTMORTEM NO. 76/62 The two layers of the pericardium were adherent over the posterior aspect of heart, and when separated gave a "bread and butter" appearance, the posterior wall being injected and dark in colour. Heart weighed 280g., the chambers were normal in size and the valves showed no abnormality. Section revealed a recent haemorrhagic infarct involving two-thirds of posterior wall and the posterior one-third of interventricular septum. A small old fibrotic area was seen in the lateral wall. The coronary arteries showed gross atheroma, were greatly narrowed but not occluded; no antemortem thrombus was present.

L. V. WALL THICKNESS Normal wall 15mm., Infarct 13mm.

CASE NO. A. 5. M. W. Female, 67 years. Registration No. 204707

Admitted 27.12.62, Discharged 16.1.63 (20 Days)

	A.S.	P.H.	S.	F.	R.	E.C.G.	TOTAL
PROGNOSTIC INDEX	3	1	5	1	4	4	18

Moderately shocked, with sacral oedema and slight neck vein distension on admission. B. P. 114/70, Pulse 80/minute irregular due to ventricular extrasystoles. Past history of angina on effort for some years.

E.C.G. Posterior infarct, Q. S. waves in leads III, and Avf. P-R interval on admission 0.17 seconds, on 3rd. day 0.18 seconds, on 20th. day 0.18 seconds.

TRANSAMINASES	Day	1	2	4
	S.G.O.T.	370	380	130
	S.G.P.T.	286	295	295

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	7th	114	4.5	106	25.5

HAEMOGLOBIN 81% (cyan method).

PROGRESS Settled well on pethidine, saluric and hydrocortisone, B.P. rose to 140/86; further progress uneventful. Remained reasonably well for 13 months (Category II) but died on 22.2.64 $_2$ days after sustaining a further myocardial infarct.

CASE NO. A. 6 A. McC. Male, 62 years. Registration No. 206412

Admitted 20.1.63, Discharged 19.2.63. (30 Days)

	A.S.	P.H.	S.	F.	R.	E.C.G.	TOTAL.
PROGNOSTIC INDEX	2	6	1	1	4	4	18

Admitted with slight shock, basal crepitations, but no neck vein distension or peripheral oedema. B. P. 175/115 on admission falling rapidly to 125/95. 125/95. Pulse 120/minute irregular, due to ventricular extrasystoles. Previous history of myocardial infarct 5 years before.

E.C.G. Posterior infarct, Q. S. waves in leads III and Avf. P-R interval 0.15 seconds on all occasions.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	78	152	85
	S.G.P.T.	19	34	47

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	6th	144	4.1	102	29.0
	15th	143	4.7	103	28.6

HAEMOGLOBIN 99% (cyan method).

PROGRESS Extrasystoles suppressed by procaine amide, pulse settled at 80/minute after 6 hours, and B. P. soon stabilised at 140/80. Further progress uneventful and remained well (Category II) for one year. Thereafter became more disabled (Category III) remaining thus at 27 months.

CASE NO. A. 7. D. B. Male, 50 years. Registration No. 206358

Admitted 19.1.63, Discharged 19.2.63 (31 days).

	A. S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX.	0	1	5	4	4	4	18

Admitted shocked, B. P. 105/72, pulse 125/minute regular. Past history of angina for one year.

E.C.G. Anteroseptal infarct, Q S waves in leads I, Avl, V1 - V5 P - R interval on admission 0.17 seconds, on 8th. day 0.16 seconds on 28th. day 0.17 seconds.

TRANSAMINASES	Day	2	3
	S.G.O.T.	40	48
	S.G.P.T.	10	16

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	6th	140	3.8	101	24.0

HAEMOGLOBIN 95% (Sahli).

PROGRESS Sedated with pethidine, and settled well on steroids and chlorothiazide; Further progress uneventful. Alive and symptom free after 24 months (Category I)

CASE NO. A. 8 J. D. Male, 62 years. Registration No. 202911.

Admitted 1.12.62, Discharged 23.1.63, (53 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	3	5	1	4	3	18

Admitted moderately shocked, B.P. 200/125 falling quickly to 150/100, pulse 92/minute irregular, due to auricular fibrillation. Past history of hypertension and effort dyspnoea (4 years).

E.C.G. Posterior infarct, Q S waves affected in leads II, III, Avf. P-R interval on admission 0.18 seconds, on 8th. day 0.18 seconds, on 28th. day 0.16 seconds. Auricular fibrillation present intermittently.

TRANSAMINASES	Day	2	3	4
	S.G.O.T.	170	170	40
	S.G.P.T.	47	56	47

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	5th.	145	4.3	103	21.6

HAEMOGLOBIN 100% (Sahli)

PROGRESS Sedated with pethidine, incipient failure controlled with chloro-:thiazide and auricular fibrillation, partially suppressed with digoxin. B.P. stabilised around 120-130/95, pulse 85/minute irregular at times due to bursts of fibrillation. Never well after discharge from hospital, being in congestive failure much of the time despite repeated administration of digoxin and Chlorothiazide. (Category IV) but was still alive at 22 months after the infarct.

CASE NO. A. 9. M. S. Female, 50 years. Registration No. 166940

Admitted 12.9.61. Discharged 15.10.61. (34 Days)

	A. S.	P. H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	3	5	4	0	4	18

Moderately shocked on admission, B. P. 105/68, pulse 100/minute, regular. Ankle oedema and neck vein over filling present. Past history of mild cerebral thrombosis in March, 1961; dyspnoeic on effort thereafter.

E.C.G. Acute posterior infarct affecting Q S waves in leads II, III, Avf, P-R interval on admission 0.17 seconds, 3rd. day 0.16 seconds, 28th. day 0.17 seconds.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	58	18	18
	S.G.P.T.	18	25	25

HAEMOGLOBIN 88% (cyan method)

PROGRESS Sedated with pethidine, B. P. soon rose to 145/88, pulse soon 80-90/minute regular. Digoxin and Chlorothiazide given for 2 days only and thereafter congestive failure was absent. Considerable dyspnoea after discharge (Category III) uhtil 22.9.62 when re-admitted with a further infarct (prognostic score 10). Thereafter felt better, dyspnoea absent except on considerable effort, no evidence of congestive failure, (Category II). Still alive and quite well 36 months after original myocardial infarct.

CASE NO. A. 10 A. B. Male, 59 years. Registration No. 183593

Admitted 31.12.63, Died 6.1.64 (7 Days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	3	5	1	4	3	18

Admitted shocked, B. P. 45/3, with basal crepitations but no peripheral oedema, pulse 64-130/minute, irregular due to sinus tachycardia alternating with episodes of 2 : 1 heart block. Past history of intermittent claudication for 3 years, cerebral thrombosis 1959.

E.C.G. Posterior infarct involving Q R waves in leads III, Avf. On admission 2 : 1 heart block alternated with sinus rhythm, P. R. interval 0.26 seconds. Periods of block continued on days 2 - 6 but on 2nd. day P-R interval fell to 0.16 seconds rising again to 0.26 seconds on the 4th. day. On the 7th. day 2 : 1 blocks became continuous.

TRANSAMINASES	Day	1	2
	S. G. O. T.	124	115
	S. G. P. T.	32	100

HAEMOGLOBIN 104% (Sahli).

PROGRESS Recovered slowly from shock, B. P. 125/90 at end of 1st. day. Partial heart block showed no real improvement despite steroid therapy and progressive congestive failure supervened despite digoxin, chlorothiazide and mersalyl. On 7th. day 2 : 1 block became continuous and the patient died suddenly. External cardiac massage was of no avail.

POST /

POSTMORTEM NO. 19/64.

The pericardial sac was distended and contained 420 c.c. of blood clot. The posterior wall of left ventricle showed aneurysmal bulging over an area $\frac{3}{4}$" in diameter, and in adjacent part of right ventricle was a small rupture. Heart weighed 435g., showed some left ventricular hypertrophy but no abnormality of valves. Section showed a recent yellow/red infarct involving the entire posterior wall of left ventricle, the posterior half of interventricular septum, and a small part of adjacent right ventricle through which was a zig zag rupture (fig. 16). The affected parts of left ventricle were thinned and showed aneurysmal bulging.

The coronary arteries showed gross atheroma, many plaques being ulcerated; over one such plaque at the origin of the right artery was an antemortem thrombus occluding the lumen.

L. V. WALL THICKNESS Normal wall 17mm. Infarct 2mm.

CASE NO. A. 11 J. S. Female, 72 years. Registration No. 211308.

Admitted 19.3.63, Died 20.3.63. (1 Day)

	A. S.	P. H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	0	3	18

Admitted in shock, B. P. 110/78, pulse 110/minute thready in character, and in congestive failure. Past history of effort dyspnoea in recent years.

E. C. G. Posterior infarct. Q S waves affected in leads II, III, Avf. P-R interval on admission 0.16 seconds.

TRANSAMINASES	Day	1
	S. G. O. T.	45
	S. G. P. T.	47

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	1st.	137	5.0	102	24

PROGRESS Did not respond to steroids, pethidine, digoxin or chlorothiazide, B. P. fell gradually to 50/? , pulse became weaker, and failure more severe. Death occurred 20 hours after admission, external cardiac massage being attempted without success.

POSTMORTEM NO. 92/63.

Pericardium injected over posterior wall; fluid normal in nature and volume. Heart weighed 350g. and left ventricle appeared slightly hypertrophied. Mitral and aortic valves slightly stretched but not incompetent; other valves normal. Recent infarct involved entire posterior wall and small part of inter-ventricular septum. Other parts of myocardium normal. Coronary arteries greatly narrowed by grossly ulcerated atheroma but no complete occlusion or

antemortem thrombus seen.

WALL THICKNESS Normal wall 15mm. Infarot 15mm.

CASE NO. A. 12. W. H. Male, 70 years. Registration No. 177743

Admitted 29.12.62. Died 9. 1.63 (12 Days).

	A. S.	P. H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	6	5	0	0	4	18

Admitted shocked, B. P. 100/60, pulse 100/minute regular, no evidence of congestive failure. Past history of myocardial infarct on 27. 1. 62.

E.C.G. Old anteroseptal infarct and new posterior infarct affecting Q S waves in leads II, III, Avf. P-R interval steady at 0.15 seconds.

TRANSAMINASES	Day	1	2	3	4
	S.G.O.T.	63	32	27	35
	S.G.P.T.	12	14	12	34

HAEMOGLOBIN 91% (Sahli).

PROGRESS Improved at first, B. P. stabilised around 120/70 but then slipped into congestive failure despite administration of chlorothiazide and mersalyl.

POSTMORTEM NO.

Pericardium normal and pericardial sac contained normal volume of straw coloured fluid. Heart weighed 580f. and left ventricle was greatly hypertrophied. Aortic valve ring slightly stretched but valve remained competent. Section showed recent transmural infarct measuring 45 x 30mm. in posterior wall, and small old fibrosed infarct at apex; interventricular septum normal. Coronary arteries greatly narrowed by ulcerated atheromatous plaques and no antemortem thrombus was seen.

L.V. WALL THICKNESS Normal wall 25mm. Infarct 17mm.

CASE NO. A. 13. R. M. Male, 46 years. Registration No. 162218

Admitted 28.7.63, Died 31.7.63. (3 Days)

	A. S.	P. H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	0	3	5	4	4	4	18

Admitted shocked, B.P. 65/40, pulse 125/minute regular, with slight ankle oedema and neck vein over filling. Past history of angina for many years, and dyspnoea on effort for a few months.

E. C. G. Posterior infarct, with left bundle branch block; Q S waves affected in leads II, III, Avf. P-R interval 0.16 seconds on admission, on 3rd. day 0.15 seconds. Sinus tachycardia present initially.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	72	131	121
	S.G.P.T.	140	225	214

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	2	139	4.0	99	23.4

HAEMOGLOBIN 102% (cyan method).

PROGRESS Improved a little initially, B. P. 100/40 at end of first day, but then deteriorated gradually, B.P. falling to 60/20 on 3rd. day. Congestive failure became more severe despite digoxin and chlorothiazide. Death occurred later on 3rd. day; permission for autopsy was refused.

CASE NO. A. 14. J. H. Male, 63 years. Registration No. 229720

Admitted 10.11.63, Died 11.11.63. (1 Day)

	A. S.	P. H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	3	1	4	4	4	18

Admitted with transient shock, B. P. 80/40 rising soon to 125/75, pulse 100/minute irregular due to auricular fibrillation. Moderate ankle oedema and basal crepitations present. Past history of angina, effort dyspnoea and intermittent claudication for one year.

E.C.G. Anterlateral infarct affecting Q S waves in leads I, Avl, V2 - V6 P-R interval 0.14 seconds.

TRANSAMINASES	Day	1
	S.G.O.T.	12
	S.G.P.T.	11

ELECTROLYTES	Day	Sodium.	Potassium.	Chloride	CO_2
	1	138	3.6	103	199

PROGRESS Deteriorated very rapidly soon after admission. Auricular fibrillation was persistent and congestive failure developed rapidly. The administration of chlorothiazide and digoxin had no obvious effect and a defibrillator was used without success. After death internal cardiac massage was attempted without success.

CASE NO. A. 15. E. R. Female, 59 years. Registration No.193346

Admitted 11.8.62, Discharged 10.9.62 (30 Days)

	A. S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	0	5	4	4	4	19

Shocked on admission, B.P. 90/70, pulse 88/minute irregular due to intermittent auricular fibrillation. Mild ankle oedema and neck vein over filling present. No relevant past history.

E.C.G. Anteroseptal infarct; Q S waves affected in leads I, Avl, V1 - V5. Auricular fibrillation present most of the time; when absent P-R interval 0.14 seconds.

TRANSAMINASES	Day	2	3	4
	S.G.O.T.	175	118	58
	S.G.P.T.	63	49	38

HAEMOGLOBIN 94% (cyan method)

PROGRESS B. P. rose over 24 hours to 120/80. Following the administra-:tion of digoxin and chlorothiazide outbursts of fibrillation became less frequent but did recur occasionally. Evidence of congestive failure regressed, disappearing on the 6th. day. Subsequent progress uneventful until discharged.

Re-admitted 21.11.62 with attack of ventricular extrasystoles but thereafter remained well (Category II) for 2 years. At 27 months dyspnoea on effort became troublesome (Category III) though there was no evidence of further infarction.

CASE NO. A. 16. I. S. Female, 55 years. Registration No. 176289

Admitted 12.1.62, Discharged 20.2.62 (39 Days)

	A. S.	P. H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	0	5	4	4	4	19

Very shocked, B. P. 105/70, pulse 130/minute regular, with pulmonary oedema, neck veins over filling and slight ankle oedema on admission. No relevant previous history.

E.C.G. Anteroseptal infarct involving Q S waves in leads I, Avl, V1 - V5. P-R interval 0.15 seconds throughout first week, 0.16 seconds thereafter. Sinus rhythm always present.

TRANSAMINASES	Day	2	3
	S.G.O.T.	71	64
	S.G.P.T.	40	50

HAEMOGLOBIN 87% (cyan method)

PROGRESS Speedy improvement after administration of pethidine, aminophyline, digoxin, chlorothiazide and hydrocortisone. B. P. 115/85 after 5 hours and 125/80 on 3rd. day. Pulse 90/minute, regular after 3 hours. Subsequent progress uneventful and remained well (Category II) until 7.3.64 when she sustained a further myocardial infarct and died later that day.

CASE NO. A. 17 W. H. Male, 78 years. Registration No. 195463

Admitted 10.9.62, Discharged 16.10.62 (36 Days)

	A. S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	1	1	4	4	19

Transient shock on admission, B.P. 125/65, pulse 44/minute mostly regular. Basal crepitations present but no peripheral oedema or neck vein over filling. History of myocardial infarct several years earlier.

E.C.G. Complete heart block, ventricular complexes 1.36 seconds apart, auricular impulses 0.48 - 0.53 seconds apart. 9 hours later (after steroids) sinus rhythm restored, P-R interval 0.16 seconds, with changes of posterior infarct involving Q S waves in leads II, III, Avf. On 3rd. and 28th. days P.R. interval 0.17 seconds.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	36	46	44
	S.G.P.T.	10	82	40

HAEMOGLOBIN 98% (Sahli)

PROGRESS Speedy abolition of block following administration of hydrocortisone; completely normal conduction after 9 hours. B. P. soon rose and on 2nd. day was 148/90, pulse 76/minute regular. Subsequent progress uneventful. After discharge moderate dyspnoea on effort present (Category III). In congestive failure at 7 and 18 months following onset of ventricular extrasystoles. Re-admitted for treatment of failure at 7 months, but relatively well at 2 years.

CASE NO. A. 18. M. G. Female, 81 years. Registration No. 184445

Admitted 13.10.62, Died 18.1.63 (63 Days)

	A. S.	P. H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	3	1	4	4	4	19

Admitted with slight shock, and marked oedema of legs, B.P. 140/85, pulse 48/minute regular. History of dyspnoea on effort for 5 years.

E.C.G. Complete heart block initially, but 2 hours after starting steroids changed to 2 : 1 block; one hour later sinus rhythm restored, P-R interval 0.22 seconds with changes of a posterolateral infarct; Q S wave changes in leads II, III, Avf, V5 and V6. P-R interval 0.20 seconds on 8th. day, 0.17 seconds on 28th. day. Ventricular extrasystoles on 2nd. and 10th. days.

TRANSAMINASES

Day	2	3
S.G.O.T.	105	53
S.G.P.T.	190	33

ELECTROLYTES

Day	Sodium	Potassium	Chloride	CO_2
2	143	4.3	104	26.1
8	138	3.1	97	24.7
15	140	3.1	100	27.1
23	141	4.0	101	28.6

HAEMOGLOBIN 81% (cyan method)

PROGRESS Never well. Despite administration of digoxin, chlorothiazide and mersalyl, congestive failure became worse and eventually a hypostatic pneumonia developed 3 days before death.

CASE NO. A. 19. W. N. Male, 52 years. Registration No. 167396.

Admitted 17.9.61 Discharged 20.10.51 (34 Days)

	A. S.	P. H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	0	6	5	4	0	4	19

Admitted shocked, B. P. 76/?, pulse 105/minute regular, with pulmonary oedema and slight ankle oedema. Posterior myocardial infarct in 1960.

E.C.G. Acute anteroseptal infarct involving Q S waves in leads I, V1 - V4. P-R interval constant at 0.20 seconds; left bundle branch block present.

TRANSAMINASES	Day	2
	S.G.O.T.	110
	S.G.P.T.	45

HAEMOGLOBIN 93% (cyan method)

PROGRESS Steady improvement after receiving chlorothiazide and steroids, B. P. 130/75 after 15 hours. Subsequent progress uneventful. After discharge troubled by moderate exercise intolerance (Category III) but still alive and able to perform light work 39 months after infarction.

CASE NO. A. 20. S. H. Female, 62 years. Registration No. 210308.

Admitted 9.3.63 Died 10.3.63 (2 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	0	4	19

Admitted shocked, B.P. 100/70, pulse 95/minute regular, with marked ankle oedema, neck vein distension and orthopnoea.

E.C.G. Anterior infarct with Q S wave changes in leads I, Avl, V3 - V6. P-R interval 0.17 seconds.

TRANSAMINASES	Day	1
	S.G.O.T.	56
	S.G.P.T.	52

PROGRESS Improved initially, B.P. rising to 135/92, but after 36 hours auricular fibrillation appeared, and death followed soon after.

CASE NO. A. 21 M. S. Female, 63 years. Registration No. 178685

Admitted 8.2.62, Died 19.2.62 (11 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	3	5	4	4	1	19

Shocked on admission, B.P. 70/30, pulse 105/minute irregularly irregular, with oedema of legs and neck vein over filling. Cerebral upset one year before (? embolus); rheumatic heart disease as child with mild mitral stenosis, and intermittent auricular fibrillation in recent years.

E. C. G. Auricular fibrillation marked appearances most of the time but changes of ? A/S infarct seen late on 1st. day. P-R internal uncertain.

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	2	159	2.96	116	16.8
	3	156	4.05	107	33.5
	6	159	4.02	109	33.0

HAEMOGLOBIN 88% (Sahli).

PROGRESS After 12 hours B. P. rose to 115/60 and on 3rd. day was 120/72. Digoxin, chlorothiazide and mersalyl were given and peripheral oedema slowly improved but auricular fibrillation persisted most of the time. Oral potassium given on 2nd. day. Death occurred suddenly and unexpectedly on the 11th. day.

POSTMORTEM NO. 42/62.

Pericardium and pericardial sac appeared normal. Heart weighed

420g. /

420g., left ventricle was very prominent and there was slight hypertrophy of right ventricle. Section showed a relatively recent infarct involving the anterior ⅓ of interventricular septum, and an area of anterior wall 35mm. in diameter. The mitral valve cusps were thickened and the valve showed minimal stenosis and incompetence; other valves were normal. No antemortem thrombus was seen within the auricular appendages. The coronary arteries showed gross atheroma, were very narrowed, but nowhere occluded completely; no antemortem thrombus was seen.

L. V. WALL THICKNESS Normal wall 16mm. Infarct 11mm.

BRAIN A recent softening was present in right cerebral hemisphere, affecting internal capsule and basal ganglia.

CASE NO. A. 22. M. W. Female, 74 years. Registration No. 194456

Admitted 28.8.62, Died 3.9.62 (6 Days)

	A. S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	3	1	4	4	4	19

Admitted to surgical department with acute abdominal pain but transferred to medical ward within one hour. B. P. 130/90 falling swiftly to 96/60, pulse 128/minute regular. Slight ankle oedema present but no neck vein over filling. Dyspnoea on effort for 5 years.

E.C.G. Anteroseptal infarct affecting Q S waves in leads I, Avr, V1-V4. P-R interval on admission 0.14 seconds, on 3rd. day 0.17 seconds, on 5th. day 0.16 seconds.

HAEMOGLOBIN 88% (cyan method)

PROGRESS Shock was transient, B.P. soon 110/80, pulse 90/minute. Despite digoxin and mersalyl, congestive failure worsened gradually until death.

POSTMORTEM NO. 622672.

Pericardial sac contained a normal amount of straw coloured fluid but pericardium over anterior wall and apex was injected. Heart weighed 490g. Left ventricle was a little prominent but valves were normal. Section showed a recent infarct involving anterior $\frac{1}{3}$ of interventricular septum, half of anterior wall and apex. A small fibrotic area was seen in posterior wall. The coronary vessels showed gross atheroma and the

left /

left was occluded by an antemortem thrombus attached apparently to an ulcerated plaque.

L.V. WALL THICKNESS Normal Wall 20mm., Infarct 17mm.

CASE NO. A. 23 H. S. Male, 59 years. Registration No. 194509

Admitted 23.10.62 Died 25.10.62 (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	1	3	7	0	4	4	19

Admitted grossly shocked, B.P. 100/70 falling steadily, pulse 150/minute and no evidence of failure. History of severe angina and effort dyspnoea for some months.

E.C.G. Anterior infarct affecting Q S waves in leads I, Avl, V4 - V6. P-R interval 0.14 seconds. Sinus tachycardia present at all times.

TRANSAMINASES	Day	1
	S.G.O.T.	59
	S.G.P.T.	27

PROGRESS Never recovered from initial shock, and throughout most of admission B. P. was about 70/42, and sinus tachycardia persisted. Congestive failure supervened on the 2nd. day and did not improve with mersalyl or digoxin.

CASE NO. A. 24. J. T., Male, 58 years. Registration No. 206706

Admitted 24.1.63, Discharged 23.2.63, (31 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	1	0	7	4	4	4	20

Admitted gravely shocked, B.P. 85/60, pulse 46/minute regular, with moderate neck vein over filling and slight ankle oedema. No relevant previous history.

E.C.G. Anterolateral infarct affecting Q S waves in leads I, Avl., V3-V6 P-R interval constant at 0.18 seconds. Sinus Bradycardia present on first day; no evidence of conduction defect.

TRANSAMINASES	Day	4	5
	S.G.O.T.	26	16
	S.G.P.T.	123	76

HAEMOGLOBIN 93% (cyan method)

PROGRESS After the administration of steroids and chlorothiazide there was a gradual improvement, B.P. 110/70 at end of 1st. day, 130/82 on 3rd. day. Pulse rate 70/minute on 2nd. day. Subsequent progress uneventful. After discharge very dyspnoeic at first, with congestive failure at 3 months (Category III) requiring hospitilisation but thereafter was much less disabled (Category II) and at 21 months was alive and active.

CASE NO. A. 25. A. C. Male, 60 years. Registration No. 204216

Admitted 19.12.62, Died 20.12.62 (2 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	1	5	4	4	4	20

Admitted with moderate shock, B.P. 100/62, pulse 95/minute, irregular; marked ankle oedema and neck vein over filling present. History of angina for 2 years.

E.C.G. Posterior infarct; Q S waves affected in leads III, Avf. P-R interval 0.16 seconds. Ventricular extrasystoles occurred frequently.

TRANSAMINASES	Day	1	2
	S.G.O.T.	28	109
	S.G.P.T.	13	122

PROGRESS Did not improve despite administration of hydrocortisone, digoxin and chlorothiazide. B.P. rose to 125/70 on 1st. day then fell away gradually: Congestive failure worsened until death on the 2nd. day.

CASE NO. A. 26. E.S. Male, 65 years. Registration No. 216290

Admitted 4.5.63, Died 5.5.63 (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	4	1	20

Severely shocked, B.P. 50/40, pulse 84/minute, irregular. Slight ankle oedema and many basal crepitations present. History of angina and slight ankle swelling recently.

E.C.G. Recent posterolateral infarct. T wave changes only seen in leads II, III, Avf, V5-V6. P-R interval 0.16 seconds.

TRANSAMINASES	Day	1	2
	S.G.O.T.	42	52
	S.G.P.T.	100	110

PROGRESS Improved a little at first, then condition deteriorated rapidly despite digoxin and mersalyl.

CASE NO. A. 27 T.R. Male, 77 years Registration No. 187575

Admitted 22.5.62, Died 27.5.62 (5 Days)

	A. S.	P. G.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	7	4	0	3	20

Admitted grossly shocked, B. P. unrecordable, pulse 110-120/minute, regular; Triple rhythm at times. Moderate ankle oedema present. Past history of dyspnoea on effort for 5 years.

E.C.G. Posterior infarct. Q R wave changes in leads II, III, Avf. P-R interval 0.18 seconds.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	67	90	85
	S.G.P.T.	29	43	71

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	2	142	6.15	105	18.1
	3	133	5.76	97	17.2
	4.	130	4.02	92	19.4
	5	136	3.84	90	27.8

PROGRESS Never really came round from initial shock and systolic pressure never rose above 50mm. Hg. Despite steroids, digoxin and chlorothiazide congestive failure became more severe and death occurred on 5th. day.

POSTMORTEM NO. 101/62.

There was a fibrinoid pericarditis, particularly over the posterior wall /

wall which was injected. Heart weighed 465g., left ventricle was moderately prominent, and the valves were normal. Section showed a recent infarct involving most of the posterior wall and a small part of adjacent interventricular septum. Both coronary arteries showed moderate atheroma and an antemortem thrombus occluded the right artery 5mm. from its origin where the vessel was narrowed by an atheromatous plaque.

L V WALL THICKNESS Normal wall 21mm., Infarct 18mm.

CASE NO. A. 28 A. S. Male, 56 years. Registration No. 173403

Admitted 26.11.61, Died 28.11.61 (2 days)

	A. S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	1	0	7	4	4	4	20

Admitted grossly shocked, B.P. 80/60 falling to 60/? within one hour, pulse 105/minute irregular. Slight ankle oedema present. No relevant previous history.

E.C.G. Posterior infarct, Q S waves involved in leads II, III, Avf. P-R interval 0.22 seconds. Frequent ventricular extrasystoles present during 1st. day.

TRANSAMINASES	Day	1
	S.G.O.T.	200
	S.G.P.T.	105

HAEMOGLOBIN 97% (Sahli).

PROGRESS Never really recovered from initial shock, systolic pressure never above 80 mm. Hg. Congestive failure gradually worsened and death occurred at end of 2nd. day.

POSTMORTEM (File No. 640500).

Pericardial surface over posterior wall injected but pleural fluid normal in volume and character. Heart weighed 405g., left ventricle was slightly hypertrophied, and the valves were normal. A recent infarct involved ⅓ of posterior wall and the adjacent ½ of lateral wall. An old fibrosed infarct /

infarct involved the remainder of posterior wall and posterior ⅓ of inter-ventricular septum. The coronary arteries showed gross atheroma, were greatly narrowed, but no antemortem thrombus was seen.

L.V. WALL THICKNESS Normal wall 15mm., Recent Infarct 14mm.

CASE NO. A. 29. J. H. Male, 63 years. Registration No. 229848.

Admitted 10.11.63, Died 11.11.63, (2 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	6	7	4	0	1	20

Gravely shocked with peripheral oedema, B.P. 105/60, pulse 95/minute, regular. Myocardial infarct in 1961.

E.C.G. A/S infarct affecting T waves only in leads I, V1 - V4. P-R interval 0.18 seconds.

TRANSAMINASES	Day	1
	S.G.O.T.	42
	S.G.P.T.	19

PROGRESS Did not recover properly from initial shock despite hydrocortisone, digoxin and chlorothiazide.

POSTMORTEM NO. 219/63.

The pericardial sac contained a normal volume of straw coloured fluid and anterior surface was injected. Heart weighed 600g., left ventricle was greatly hypertrophied, but the other chambers and the valves were within normal limits A rather poorly defined recent infarct was seen in anterior half of interventricular septum, apex, and most of anterior wall. The coronary arteries were narrowed by gross atheroma and an antemortem thrombus occluded the left ventricle at a point 7mm. from its origin.

L.V. WALL THICKNESS Normal wall 22mm. Infarct 19mm.

CASE NO. A. 30. E. B. Female, 73 years. Registration No. 189167

Admitted 9.6.62, Discharged 27.7.62, (48 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	0	7	4	4	3	21

Admitted severely shocked, B.P. 75/50, pulse 100/minute irregular, with gross ankle oedema and neck vein distension. No relevant previous history.

E.C.G. Posterior infarct with changes in leads II, III, Avf confined to P-R wave until 5th. day when Q S waves become affected. P-R interval 0.24 seconds on admission, 0.18 seconds on 3rd. day and 0.20 seconds on 28th. day.

TRANSAMINASES

Day	1	2	3	4	5
S.G.O.T.	62	47	68	23	22
S.G.P.T.	85	33	82	55	33

ELECTROLYTES

Day	Sodium	Potassium	Chloride	CO_2
7	139.7	3.90	100	25.1
30	136.8	4.42	99	25.3

HAEMOGLOBIN 86% (Sahli)

PROGRESS Improved extremely slowly despite administration of steroids, digoxin, aminophyline and chlorothiazide. B.P. on 2nd. day 130/80. Ventricular extrasystoles controlled after 4-5 days but returned briefly during the 4th. week when quinidine was given. Congestive failure was slow to improve, was absent at time of discharge but recurred from time to time later (Category III). Death occurred eventually on 12.2.63 following a further myocardial infarct.

CASE NO. A. 31 E.B. Male, 73 years. Registration No. 201250

Admitted 23.11.62, Died 8.12.62 (16 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	1	5	4	4	4	21

Was being investigated for complaint of progressive angina and collapsed with myocardial infarct in medical department. Shock was severe, B.P. 85/30 congestive failure developed quickly and persistant tachycardia was troublesome, pulse 125/minute regular.

E.C.G. Posterior infarct; Q S waves effect in leads II, III, Avf. P-R interval 0.15 seconds initially, 0.17 seconds on 8th. day. Sinus tachycardia present throughout first day.

TRANSAMINASES	Day	1	3
	S.G.O.T.	240	170
	S.G.P.T.	52	175

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	7	138	3.4	96	25.7

PROGRESS Never well, B. P. rose slowly to 125/80, pulse settled after first day, but congestive failure gradually supervened despite administration of digoxin and mersalyl.

POSTMORTEM NO. 268/62.

A localised fibrin exudate was present over the posterior wall, which was dusky in colcut, and moderately injected. Heart weighed 350g., the chambers were of normal size and the valves were within normal limits. A yellowish /

yellowish infarct 55 x 28mm. affected the full thickness of posterior wall. An antemortem thrombus fixed to an ulcerated atheromatous plaque occluded the right coronary artery 15mm. from its origin, although most parts of the coronary arteries were widely patent.

L.V. WALL THICKNESS Normal wall 15mm. Infarct 10mm.

CASE NO. A. 32. S.M. Male, 43 years. Registration No. 189761

Admitted 16.6.62, Died 16.6.62. (1Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	0	6	7	4	0	4	21

Admitted grossly shocked and in congestive failure, B.P. 190/130, pulse 110/minutes regular. Mild myocardial infarct one month before.

PROGRESS Condition rapidly deteriorated, B.P. fell quickly to 60/?, pulse became imperceptible and death occurred later that day despite administration of mersalyl, digoxin, morphia and hydrocortisone.

POSTMORTEM No. 123/62.

The pericardium and pericardial sac appeared normal. Heart weighed 350g., the chambers were of normal size and the valves all seemed normal. A rather poorly defined haemorrhagic infarct, 30mm. in diameter, was seen in posterior wall. An older fibrosed infarct was present in anterior ⅓ of interventricular septum, apex and a small area of anterior wall. The coronary arteries showed gross atheroma, were greatly narrowed, but showed no antemortem thrombosis and no area of complete occlusion.

L.V. WALL THICKNESS. Normal wall 14mm. New infarct 14mm.

CASE NO. A. 33. E.M. Female, 54 years. Registration No. 181105

Admitted 3.3.63, Died 3.3.63 (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	0	7	4	4	4	21

Admitted shocked, B. P. unrecordable, pulse 40/minute regular, in gross oedema and with gross pulmonary oedema. No relevant previous history.

E.C.G. Not available. Note in Case Sheet indicates that records showed sinus bradycardia.

TRANSAMINASES	Day	1
	S.G.O.T.	45
	S.G.P.T.	12

PROGRESS Did not respond to digoxin, mersalyl or aminophyline, and died within a few hours of admission. External cardiac massage was unsuccessful.

CASE NO. A. 34. J. S. Male, 66 years. Registration No. 203432

Admitted 8.12.62, Died 9.12.62, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	1	5	4	4	4	21

Admitted shocked, B.P. 110/90, pulse 108/minute irregular, with slight ankle oedema and neck vein over filling. History of angina for 3 years.

E.C.G. A/S infarct affecting Q S waves in leads I, Avl, V1 - V5. Multiple ventricular extrasystoles present. P-R interval 0.20 seconds; left bundle branch block present.

TRANSAMINASES	Day	2
	S.G.O.T.	35
	S.G.P.T.	10

PROGRESS Improved a little at first but B.P. then slowly declined, pulse rate fell to 64/minute regular and congestive failure became progressively more severe despite the administration of steroids and chlorothiazide. Death occurred 20 hours after admission.

CASE NO. A. 35. A.C. Female, 53 years. Registration No. 170396.

Admitted 15.10.62, Died 17.10.62, (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	0	7	4	4	4	21

Grossly shocked on admission. B.P. ?/?, semicomatose, pulse 40/minute regular, with marked leg and sacral oedema. No relevant previous history.

E.C.G. Complete heart block with anteroseptal infarct. Late on second day sinus rhythm suddenly restored, P-R interval 0.24 seconds, ventricular rate 68/minute. Q S wave changes now more clearly apparent in leads V1 - V3.

TRANSAMINASES	Day	1
	S.G.O.T.	184
	S.G.P.T.	320

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	2	144	4.7	98	18.8

HAEMOGLOBIN 93% (sahli).

PROGRESS Never fully recovered from shock, B.P. on 2nd. and 3rd. days 70/50 and 90/60 respectively despite sedation with morphia. Digoxin and mersalyl did not improve the congestive failure and oath occurred late on the 3rd. day.

POSTMORTEM/

POSTMORTEM NO. 222/62.

Anterior surface of heart injected but pericardial sac normal. Heart weighed 360g. and left ventricle was slightly prominent; valves were all normal. Section showed a yellow/red transmural infarct involving anterior $\frac{1}{3}$ of interventricular septum, apex and adjacent $\frac{1}{3}$ of anterior wall. The coronary arteries showed gross atheroma and an antemortem thrombus overlying one plaque near the origin of the left artery occluded the lumen.

L.V. WALL THICKNESS Normal wall 16mm., Infarct 14mm.

CASE NO. A. 36. I.M. Female, 58 years. Registration No. 178230

Admitted 1.2.62, Discharged 11.4.62 (100 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	3	5	4	4	4	22

Admitted shocked, in marked congestive failure. B.P. 100/68, pulse 100-120/minute irregular due to bouts of auricular fibrillation. History of hypertension and recent dyspnoea on effort.

E.C.G. Anteroseptal infarct affecting Q S waves in leads I, Avl, V1-V5. Auricular fibrillation present much of the time but when absent P-R interval 0.17 seconds.

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	3	146	4.2	103	22.6
	8	140	3.9	102	23.0
	28	138	4.4	96	24.1

HAEMOGLOBIN 97% (Sahli)

PROGRESS Recovered slowly from shock, B.P. 80/50 at end of 1st. day, 120/70 on 2nd. day. Auricular fibrillation persisted throughout the first month despite use of digoxin and mersalyl. Congestive failure remained for over 2 months but eventually the patient made a good recovery. After discharge effort dyspnoea was troublesome for about 6 months (Category III) but thereafter she improved and had little trouble (Category II). Congestive failure recurred in June, 1964 and subsequently dyspnoea on effort was more evident (Category III) although the patient was still alive 30 months after the infarct.

CASE NO. A. 37. H. M. Male, 71 years. Registration No. 156099.

Admitted 4.12.62, Discharged 22.12.62 (19 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	5	4	0	4	22

Moderately shocked on admission, B.P. 132/90, pulse 105/minute regular, with slight ankle oedema and marked pulmonary oedema. History of myocardial infarcts in 1952 and 1957, cerebral thrombosis 1952 and angina in recent months.

E.C.G. Anterolateral infarct involving Q S waves in leads I, II, Avl. V1 - V6. P-R interval 0.18 seconds on admission, 0.16 seconds on 8th. day, 0.17 seconds on 19th. day.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	12	15	19
	S.G.P.T.	23	23	20

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	7	135	3.6	100	22.7

HAEMOGLOBIN 87% (Sahli)

PROGRESS Responded well to aminophyline, chlorothiazide and hydrocortisone. B.P. stabilised around 130/85 and congestive failure improved speedily. Discharged home and remained well at first having only slight effort dyspnoea (Category II) but on 12.6.63 was admitted to Hawkhead Hospital because of psychotic manifestations considered to be due to cerebral ischaemia. He died there on 11.9.63 following a further cerebral thrombosis. There was no autopsy.

CASE NO. A. 38. T.P. Female, 73 years. Registration No. 152587

Admitted 22.9.62, Died 25.9.62, (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	5	4	0	4	22

Severely shocked, B.P. 110/20, pulse 90/minute regular, with slight ankle oedema. Two previous myocardial infarcts, in 1954 and 1960.

E.C.G. Posterolateral infarct, with changes in Q S waves in leads II, III, Avf, V4 - V6. P-R interval constant at 0.16 seconds.

TRANSAMINASES	Day	1	2
	S.G.O.T.	81	72
	S.G.P.T.	17	38

HAEMOGLOBIN 90% (Sahli)

PROGRESS B.P. rose initially to 130/60 but then despite aminophyline, chloro-thiazide and hydrocortisone, the B.P. fell slowly, congestive failure became progressively more severe until death.

POSTMORTEM NO. 203/62.

The pericardial sac was distended, containing 265 c.c. of straw coloured fluid and a fibrin exudate was present over posterior wall. Heart weighed 520g. and left ventricle was markedly prominent; the aortic valve ring was a little stretched but the valve was competent. There was a massive infarct involving the entire posterior wall, the adjacent ½ of lateral wall and a small part of interventricular septum. The coronary arteries were markedly narrowed, though not /

not occluded by calcified, ulcerated plaques. No antemortem thrombus present.

L.V. WALL THICKNESS Normal wall 20mm. Infarct 16mm.

CASE NO. A. 39 G. S. Male, 74 years. Registration No. 1668818

Admitted 4.10.61, Died 6.10.61 (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	6	1	4	4	4	22

Admitted with slight shock, B.P. 130/90, pulse 45/minute, regular; slight ankle oedema present. Previous myocardial infarct 1959.

E.C.G. Recent anterolateral infarct affecting Q S waves in leads I, II, Avl, V1 -- V6. Old posterior infarct present. Complete heart block present.

TRANSAMINASES	Day	2
	S.G.O.T.	210
	S.G.P.T.	240

PROGRESS B. P. rose to 145/90 on 2nd. day but heart block did not improve. Congestive failure became more severe despite mersalyl and hydrocortisone, and death occurred on the 3rd. day. Stoke-Adams attacks occurred repeatedly throughout the admission.

CASE NO. A. 40. A. R. Male, 68 years. Registration No. 153381

Admitted 25.4.61, Died 25.4.61 (1 Day)

	A.S.	P.H.	S.	F.	R.	B.	TOTAL
PROGNOSTIC INDEX	3	0	7	4	4	4	22

Admitted in gross shock with marked leg and sacral oedema. B.P. 40/?, pulse barely perceptible. No relevant past history.

TRANSAMINASES	Day	1
	S.G.O.T.	51
	S.G.P.T.	47

PROGRESS Did not recover despite steroids, digoxin and mersalyl.

POSTMORTEM NO. 611095B

Pericardium and pericardial sac appeared normal. Heart weighed 360g. all chambers were normal in size and the valves were normal. A haemorrhagic infarct was found in anterior ⅓ of interventricular septum, apex and a small area of anterior wall. The coronary arteries were narrowed by gross atheroma but there was no antemortem thrombus.

L.V. WALL THICKNESS Normal wall 16mm. Infarct 16mm.

CASE NO. A. 41. J.C. Male, 72 years. Registration No. 193323

Admitted 9.8.62, Discharged 11.9.62, (32 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	1	4	4	4	22

Admitted with transient shock, B.P. 120/75 rising to 138/78 within one hour, pulse 130/minute regular. There was oedema of the legs and lungs. Previous history of myocardial infarct one year before.

E.C.G. Anterlateral infarct, Q S waves affected in leads I, II, V1 - V6. Supraventricular tachycardia present during first 2 hours. P-R interval 0.18 seconds on admission, 0.16 seconds on 8th. day, 0.18 seconds on 28th. Day.

TRANSAMINASES	Day	1	2
	S.G.O.T.	49	130
	S.G.P.T.	14	26

HAEMOGLOBIN 93% (cyan method).

PROGRESS Quick improvement following administration of aminophyline, digoxin and chlorothiazide. Pulse 85/minute after 2 hours. Penicillin given for a chest infection from 21.8.62 until 24.8.62. Progress otherwise uneventful. After discharge was quite fit having only slight dyspnoea (Category II) but died in March, 1964, following a further myocardial infarct.

CASE NO. A. 42. A.K. Male, 65 years. Registration No. 174994

Admitted 20.12.61, Discharged 21.1.62, (32 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	4	4	23

Admitted moderately shocked, B.P. 95/70, pulse 100/minute regular, with leg and sacral oedema. Previous history of dyspnoea on effort for 6 months.

E.C.G. Anteroseptal infarct involving Q S waves in leads I, Avl, Vl - V5. P-R interval on admission 0.22 seconds, soon 0.32 seconds, 6 hours later 0.26 seconds. On 2nd. day 0.17 seconds, 3rd. and subsequent days 0.18 seconds. Ventricular extrasystoles on 1st. day.

TRANSAMINASES	Day	2
	S.G.O.T.	72
	S.G.P.T.	51

PROGRESS Condition much improved after 12 hours, B.P. 145/90, pulse 80/minute regular. Congestive failure improved slowly with digoxin and chlorothiazide. After discharge unwell due to dyspnoea on moderate effort and to intermittent claudication, (Category III). Dyspnoea became so severe that the patient became a cardiac cripple early in 1963. Re-admitted in April, 1963, with congestive failure and incipient gangrene of both feet, and died in hospital on 28.7.63.

CASE NO. A. 43. A. M. female, 66 years. Registration No.188534

Admitted 3.6.62, Died 3.6.62 (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	7	4	0	3	23

Admitted grossly shocked, B.P. 60/?, pulse barely perceptible, gross oedema of legs and sacrum present. Myocardial infarct 2 years before.

E.C.G. Posterior infarct Q R waves affected in leads III Avf. An old anteroseptal infarct was also present. P-R interval 0.19 seconds.

PROGRESS Despite the administration of morphia, aminophyline and hydrocor-:tisone, there was no improvement. Ventricular fibrillation immediately preceded death.

CASE NO. A. 44. T.B. Male, 72 years. Registration No. 196418

Admitted 20.9.62, Died 20. 9.62, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	7	4	0	3	23

Admitted severely shocked, B.P. 70/20, pulse 72/minute regular; neck veins over filled, lungs oedematous, but peripheral oedema absent. Previous myocardial infarct in 1961; angina for 6 years.

E.C.G. Lateral infarct, Q. R. waves in leads I, II, Avf, V5 - V6.
P-R interval 0.17 seconds.

PROGRESS Showed no improvement despite administration of digoxin prior to death late on the 1st. day.

CASE NO. A. 45. J.S. Male, 72 years. Registration No. 162371

Admitted 20. 7. 61., Died 22. 7. 61. (2 Days).

PROGNOSTIC INDEX	A.S.	P.M.	S.	F.	R.	E.	TOTAL
	3	3	5	4	4	4	23

Admitted shocked, B.P. 100/60, Pulse 78/minute, moderate ankle oedema present. History of effort dyspnoea for 3 months.

E. C. G. Anteoseptal infarct involving QS waves in leads I, V1 - V4. P.R. interval at first 0.26 seconds, then on 2nd day complete block.

PROGRESS Improved after pethidine, digoxin, chlorothiazide and hydrocortisone but on second day complete block occurred, pulse rate 50/minute, and death followed soon after.

POSTMORTEM No. 127/61.

Pericardial sac normal but anterior wall of heart injected. Heart weighed 575 G. and left ventricle was extremely prominent; valves appeared normal. There was a recent haemorrhagic infarct involving anterior half of interventricular septum, apex, and most of anterior wall. Both coronary arteries showed gross atherome and the left was occluded at its origin by an antemortem thrombus, overlying an ulcerated plaque.

L.V. wall thickness. Normal 24 mm. Infarct 16 mm.

CASE NO. A. 46. N. S. Male, 58 years. Registration No. 183907

Admitted 23. 1. 63. Discharged 7. 2. 63. (15 Days).

PROGNOSTIC INDEX	A.S.	P.M.	S.	F.	R.	E.	TOTAL
	1	6	5	4	4	4	24.

Moderately shocked on admission, B.P. 128/64 falling to 105/60, Pulse 100/min irregular; moderate ankle oedema present. Previous myocardial infarct 1 year before (included in survey as No. E 4), ? cerebral in 1945. E.C.G. Anterior infarct with QS wave changes in leads I, Avl, QS V_1 - V5. P.R. interval 0.17 seconds in all tracings. Runs of centricular extrasystoles on first day.

TRANSAMINASES	day	1	2	3
	S.G.O.T.	9	25	5
	S.G.P.T.	17	21	14

HAEMOGLOBIN 96% (cyan method).

PROGRESS. Quinidine administered during first 4 days and this controlled the extrasystoles. Shock improved slowly, B.P. 115/60 on 3rd day. Chlorothiazide helped to counter congestive failure during first week; subsequent progress uneventful. After discharge dyspnoea on effort was troublesome (Category III) but this then improved gradually and after 24 months the patient was relatively well and active (category II).

CASE No. A. 47. A.S. Female, 74 years. Registration No. 193091

Admitted 7. 8. 62. Died 8. 8. 62. (2 Days).

PROGNOSTIC INDEX	A.S.	P.M.	S.	F.	R.	E.	TOTAL
	3	3	7	4	4	4	25

Admitted grossly shocked and in marked congestive failure, B.P. 70/50, Pulse 98-105/minute irregular. Past history of angina and dyspnoea on effort for 3 years.

E.C.G. Posterior infarct, QS waves in leads II, III, Avf. P.R. interval 0.15 seconds. Ventricular extrasystoles present much of the time.

TRANSAMINASES	day	1	2
	S.G.O.T.	28	34
	S.G.P.T.	26	23

HAEMOGLOBIN 89% (Sahli)

PROGRESS Failed to respond to chlorothiazide, hydrocortisone, or aminophyline. B.P. fell speedily, congestive failure worsened and death occurred early on the second day.

CASE NO. A. 48. S. McB. female, 60 years. Registration No. 153021

Admitted 7.4.63, Died 10.4.63 (3 Days)

	A. S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	7	4	4	4	27

Admitted grossly shocked, B.P. 40/?, pulse 40/minute: considerable leg oedema present. Past history of myocardial infarction on 8.5.61.

E.C.G. Anterior infarct. Complete heart block present at all times.

TRANSAMINASES	Day	1	2
	S.G.O.T.	40	20
	S.G.P.T.	24	56

PROGRESS Did not respond to hydrocortisone, mersalyl or digoxin. Heart block did not improve and B. P. was never above 60/35. Death occurred on the 3rd. day and external cardiac massage failed.

CASE NO. A.49 K. McL. Male, 68 years. Registration No. 186906

Admitted 12.5.62, Died 12.5.62 (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	7	4	4	4	28

Admitted moribund, B.P. 90/40 but falling rapidly, pulse barely perceptible 105/minute; Marked peripheral and pulmonary oedema present. History of myocardial infarct in 1960.

ELECTROLYTES	Day.	Sodium.	Potassium.	Chloride.	CO_2	Protein
	1st	135.6	3.89	97	23.5	17

HAEMOGLOBIN 105% (cyan method)

PROGRESS Treatment with digoxin, chlorothiazide and hydrocortisone had no effect and death occurred 2 hours after admission.

CASE NO. B. 1. J. C. Male, 59 years. Registration No. 182523

Admitted 22.3.62, Discharged 26.4.62 (35 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	1	3	1	4	4	4	17

Admitted with slight shock, B.P. 195/110, falling soon to 105/45, pulse 92/minute irregular alternating with a rate of 57/minute irregular. Moderate ankle oedema and neck vein over filling present. History of angina and dyspnoea on effort for 10 years.

E.C.G. Initially 3 : 1 heart block present, alternating with short periods of sinus rhythm, P.R. interval 0.20 seconds. 4 hours after receiving steroids block was completely abolished and P-R interval was 0.16 seconds; On 8th and 28th. days interval was 0.20 seconds. In addition there were the changes of a posterior infarct affecting Q S waves in leads II, III, Avf.

TRANSAMINASES	Day	2	3
	S.G.O.T.	30	25
	S.G.P.T.	29	51

HAEMOGLOBIN 98% (Sahli)

PROGRESS Responded well to chlorothiazide and steroids, B.P. 140/90 at end of 1st day. Recovert was uneventful and after discharge was quite well (Category II) at first. After 27 months dyspnoea on effort became more troublesome and bursts of ventricular extrasystoles were noted on E.C.G. tracings At this time was seen at Eye Department because of failing vision and the fundi showed moderately severe hypertensive changes, B.P. 165/98. Still alive at /

at 33 months though dyspnoea on effort restricted his activities (Catogory III).

CASE NO. B. 2. S. K. Male, 64 years. Registration No. 174452

Admitted 12.12.61, Died 19.1.62 (39 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	1	5	4	4	4	20

Admitted moderately shocked, B.P. 130/80, pulse 150/minute, neck veins distended and with slight ankle oedema. Past history of angina for years.

E.C.G. Posterolateral infarct affecting Q S waves in leads II, III, Avf, V5 - V6. At first supraventricular tachycardia present, alternating with auricular flutter. These arrythmias continued intermittently until 15.12.61 when sinus rhythm was restored, P-R interval 0.16 seconds.

TRANSAMINASES	Day	1	2
	S.G.O.T.	12	26
	S.G.P.T.	12	25

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	7	138	4.62	105	21.9

PROGRESS Improved slowly at first, B.P. stabilising at 120/70. Procaine amide had little effect initially on the arrythmias but sinus rhythm restored after digoxin and mersalyl had also been given. Increasing severe congestive failure developed, however, and death occurred on the 39th. day.

POSTMORTEM NO. 620351.

Pericardium and pericardial sac normal. Heart weighed 510g., left ventricle was hypertrophied and the aortic valve ring seemed a little stretched although the valve was competent. A transmural whitish infarct

40/

40 x 17mm. lay partly in posterior and partly in lateral wall. The coronary arteries were narrowed by gross ulcerated atheroma but no antemortem thrombus was present.

L.V.WALL THICKNESS Normal wall 24mm. Infarot 10mm.

CASE NO. B. 3. J.R. Female, 76 years. Registration No. 175193

Admitted 20.12.61, Died 22.12.61, (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	0	5	4	4	4	20

Admitted shocked, B.P. 100/?, pulse 48-58/minute regular. Neck veins distended, basal crepitations present, but no peripheral oedema. No relevant past history.

E.C.G. Anteroseptal infarct involving Q S waves in leads I, Avl. V1 - V4. P-R interval 0.18 seconds on 1st. and 3rd. days. Sinus bradycardia present initially; no conduction defect seen at any time.

TRANSAMINASES	Day	2
	S.G.O.T.	51
	S.G.P.T.	50

HAEMOGLOBIN 86% (Sahli)

PROGRESS Responded initially to hydrocortisone and chlorothiazide, B. P. 110/45 at end of 1st. day, but congestive failure became rapidly more severe and death occurred at the end of the 3rd. day.

CASE NO. B. 4. D. McD. Male, 61 years. Registration No. 187092

Admitted 15.5.62, Discharged 5.6.62 (21 days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	0	7	4	4	4	21

Admitted gravely shocked, B.P. 95/40, pulse 95/minute irregularly irregular. Moderate ankle oedema and neck vein over filling was present. No relevant past history.

E.C.G. Posterolateral infarct involving Q S waves in leads II, III, V4 - V6. Auricular fibrillation present most of the time but when absent P-R interval was 0.17 seconds (8th. and 21st. days).

TRANSAMINASES	Day	1	2	3	4	5
	S.G.O.T.	38	29	103	58	34
	S.G.P.T.	38	25	70	80	77

HAEMOGLOBIN 94% (Sahli)

PROGRESS Did not recover fully from initial shock for 2 days but eventually B.P. rose to 135/75. Auricular fibrillation partially controlled by digoxin, and the congestive failure regressed slowly with mersalyl and later chloro-thiazide therapy. Subsequent progress uneventful. After discharge able to do light work only due to persistent dyspnoea (Category III) remained alive at 30 months after infarction.

CASE NO. C.1. C.T. Female, 59 years. Registration No. 176404

Admitted 12.1.62, Died 27.1.62, (16 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	0	7	4	0	4	17

Admitted grossly shocked, B.P. 120/90, pulse 100/minute regular, with marked oedema of legs and sacrum, and marked neck vein over filling. No relevant past history.

E.C.G. Anteroseptal infarct affecting Q S waves in leads I, Avl Vl - V3. P-R interval on admission 0.20 seconds, 0.16 seconds on 8th. day.

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2	Protein
	7	137	4.3	97	25.6	15

HAEMOGLOBIN 104% (Sahli).

PROGRESS Remained shocked for 3 days despite administration with steroids, aminophyline, digoxin and oxygen. B. P. on 2nd. day 90/60, on 4th. day 130/78. During last week of life irreversible congestive failure developed.

POSTMORTEM NO. 162/62.

The pericardial sac contained 100ml. of straw-coloured fluid, the anterior aspect of heart was slightly injected and the posterior wall showed slight aneurysmal bulging. Heart weighed 340g., all chambers were of normal size and all valves seem normal. Section revealed a recent whitish infarct in anterior ⅓ of interventricular septum, apex and adjacent ⅓ of anterior wall. The infarcted area was thin but there was no actual aneurysm. Posterior wall contained /

contained an extensive old fibrosed infarct showing slight aneurysmal bulging. The coronary arteries were greatly narrowed by atheroma; No antemortem thrombus was seen.

L.V.WALL THICKNESS Normal wall 16mm. New infarct 5mm. Old infarct 3mm.

CASE NO. C.2. E.D. Female, 67 years. Registration No. 156737.

Admitted 18.12.62, Died 9.1.63 (22 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	1	1	4	4	4	17

Admitted with slight shock, B.P. 105/62, pulse 130/minute, with gross leg and sacral oedema. Past history of angina for years.

E.C.G. Anteroseptal infarct with Q S wave changes in leads I, V1 - V4. P-R interval constant at 0.16 seconds. Persistent sinus tachycardia on 1st. day.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	25	58	32
	S.G.P.T.	16	42	48

HAEMOGLOBIN 98% (Sahli).

PROGRESS Improved initially after administration of digoxin, chlorothiazide, B. P. 125/82 at end of 1st. day, 130/85, 2nd. day. Congestive failure returned after 10 days and became progressively more severe.

POSTMORTEM, No. 11/63.

The pericardium and pericardial sac were normal but aneurysmal bulging of anterior wall was present. Heart weighed 340g. and the chambers were of normal size. A recent partially fibrosed infarct was present in anterior $\frac{1}{3}$ of interventricular septum, apex and adjacent $\frac{1}{3}$ of anterior wall and the anterior wall was thin and showed aneurysmal change. In posterior wall /

wall was an old densely fibrosed wall. The coronary arteries showed gross atheroma with great narrowing of the lumina. No antemortem thrombus was present.

L.V. WALL THICKNESS Normal wall 16mm. Infarct 5 mm.

CASE NO. C.3. J. S. Male, 58 years. Registration No. 139248

Admitted 31.1.61, Discharged 2.3.61 (33 Days)

	A.S.	F.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	1	0	5	4	4	4	18

Admitted virtually moribund, B. P. ?/?, pulse 14/minute. Neck veins distended but no peripheral oedema present. No relevant past history.

E.C.G. Anteroseptal infarct. Complete heart block on admission with atrioventricular dissociation. After 2 hours sinus rhythm restored, P-R interval 0.15 seconds. On 8th. and 28th. days P-R interval 0.17 seconds.

TRANSAMINASES	Day	2
	S.G.O.T.	49
	S.G.P.T.	38

HAEMOGLOBIN 104% (Sahli)

PROGRESS Two hours after receiving hydrocortisone, sinus rhythm restored and thereafter rapid recovery from shock, B.P. 142/90 at end of 1st. day. Mersalyl given during first week and thereafter there was no evidence of failure. After patient was discharged he was symptom free (Category I) for one year but thereafter there was slight dyspnoea on effort (Category II); patient still alive and quite well 4 years after the infarct.

CASE NO. C. 4. R. M. Male, 50 years. Registration No. 159986

Admitted 6.5.61, Discharged 8.6.61 (34 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	3	1	4	4	4	18

Admitted with slight shock, B.P. 100/65, pulse 35-40/minute, with slight ankle oedema. History of dyspnoea on effort for one year.

E.C.G. Anteroseptal infarct and complete heart block with atrioventricular dissociation on admission. On next day sinus rhythm restored, P-R interval 0.15 seconds; 0.18 seconds on 28th. day.

TRANSAMINASES	Day	1
	S.G.O.T.	53
	S.G.P.T.	32

HAEMOGLOBIN 97% (Sahli).

PROGRESS Shock was transient and within a few hours B. P. rose to 135/72. 24 hours after starting treatment with hydrocortisone heart block was abolished. Within 3 days congestive failure had disappeared, chlorothiazide being given throughout the first week. Subsequent progress uneventful. After discharge patient was symptom free, remaining so three years later.

CASE NO. C. 5. H. Y. Male, 63 years. Registration No. 159917

Admitted 22.6.61, Discharged 27.7.61 (36 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	3	5	4	0	4	18

Initial shock, B.P. 105/62, pulse 90/minute irregular, with mild ankle oedema. History of angina and dyspnoea on effort for 2 years.

E.C.G. Posterolateral infarct affecting Q S waves in leads II, III, Avf, V5 - V6. P-R interval 0.19 seconds steady.

TRANSAMINASES	Day	2
	S.G.O.T.	16
	S.G.P.T.	18

PROGRESS After treatment with pethidine, chlorothiazide and steroids, B.P. soon rose being 145/78 on the second day. Congestive failure soon reversed, and absent after 4th. day. Subsequent progress uneventful. Symptoms free for 18 months after discharge (Category I) but thereafter had slight dyspnoea on effort (Category II). When seen at the hospital dental department 3 years after infarction he was considered fit for an anaesthetic and several teeth were extracted under a general anaesthetic without incident.

CASE NO. C. 6. M. F. Female, 60 years. Registration No. 157019.

Admitted 14.4.61, Discharged 13.5.61, (30 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	0	5	4	4	4	19

Admitted moderately shocked, B.P. 140/90, pulse 60/minute regular, with oedema of legs and sacral area. No relevant previous history.

E.C.G. Posterior infarct affecting Q S waves in leads II, III, Avf. P-R interval constant at 0.22 seconds.

PROGRESS B. P. fell initially to 100/50 but after treatment with morphia, digoxin, chlorothiazide and hydrocortisone, showed a swift improvement, B. P. 145/90 at end of first day. Oedema cleared after 5 days and subsequent progress uneventful. After discharge was well at first, having only slight dyspnoea on effort (Category II) but she sustained a cerebral haemorrhage on 11.1.62 and died later that day, 8 months after the infarct.

POSTMORTEM NO. 11/62.

Pericardium and pericardial sac normal. Heart weighed 340g., the chambers were of normal size but the posterior wall showed slight aneurysmal bulging; the valves showed no abnormality. Section showed a fibroid infarct occupying most of posterior wall but not involving interventricular septum. Parts of posterior wall were thin and showed aneurysmal bulging. The coronary arteries were greatly narrowed by ulcerated atheromatous plaques but there was no antemortem thrombus.

L. /

L.V. WALL THICKNESS Normal wall 16mm. Infarct 3mm.

Brain showed massive right cerebral haemorrhage.

CASE NO. C. 7. J. McF. Male, 63 years. Registration No. 168238.

Admitted 28.9.61 Died 8.10.61 (11 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	0	5	4	4	4	19

Admitted shocked, B. P. 116/80, pulse 130/minute regular alternating with 68/minute irregular. Marked neck vein over filling and slight ankle oedema present. No relevant past history.

E.C.G. Posterior infarct affecting Q S waves in leads III, Avf. Sinus tachycardia alternating with episodes of 2 : 1 heart block which swiftly became constant. Ventricular extrasystoles from 3.10.61.

TRANSAMINASES	Day	1
	S.G.O.T.	38
	S.G.P.T.	46

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	6	139	3.9	104	13.7
	7	135.6	4.1	101	14.2

HAEMOGLOBIN 92% (Sahli)

PROGRESS Recovered slowly from shock after administration of morphia and hydrocortisone. B. P. 120/72 on 2nd. day. After treatment with mersalyl and digoxin congestive failure improved a little though the heart block did not improve. On 3.10.61 ventricular extrasystoles appeared and were only partially controlled by quinidine. Congestive failure became more severe and the patient died on 8.10.61.

POSTMORTEM NO. 164/61.

The pericardium and pericardial sac appeared normal. Heart weighed 400g. and left ventricle was prominent. The valves all appeared normal. Section showed a recent yellowish infarct involving an area of posterior wall measuring 46 x 30mm, together with posterior half of interventricular septum. Small fibrotic areas were also noted within the affected area of septum. The coronary arteries were narrowed by ulcerated atheromatous plaques and no antemortem thrombus was seen.

L V WALL THICKNESS Normal wall 17mm. Infarct 16mm.

CASE NO. C. 8. D. A. Male, 61 years. Registration No. 153376

Admitted 25.4.61, Discharged 30.5.61 (36 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	1	5	4	4	4	20

Admitted with slight shock, B.P. 105/30 rising quickly to 125/80, pulse 40/minute regular. Slight ankle oedema present. Angina in recent months.

E.C.G. Posterior infarct and complete block with atrioventricular dissociation. Sinus rhythm restored in 14 hours, P-R interval at first 0.32 seconds; 0.19 seconds on 8th. and 28th. days. Q S waves affected by infarct pattern on first day in leads II, III, Avf.

TRANSAMINASES	Day	2	3	4	5
	S.G.O.T.	135	66	50	27
	S.G.P.T.	150	102	66	55

HAEMOGLOBIN 102% (cyan method)

PROGRESS Heart block abolished 15 hours after receiving steroids. Congestive failure became more marked initially but improved steadily thereafter under the influences of chlorothiazide. Subsequent progress uneventful. After discharge slight effort dyspnoea only (Category II) and patient was alive and active 3 years after the infarct.

CASE NO. C. 9. H. S. Female, 76 years. Registration No. 168588.

Admitted 4.10.61, Discharged 11.11.61, (39 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	0	5	4	4	4	20

Admitted in rapidly deteriorating condition, shocked. B. P. 125/75, pulse 40/minute regular, and with marked oedema of lungs, legs and sacral area. No relevant past history.

E.C.G. Anteroseptal infarct with Q S wave changes in leads I, Avl. V1 - V4. Conduction normal, P-R interval 0.20 seconds on admission, 0.18 seconds on 8th. day and 0.20 seconds on 28th. day. Sinus bradycardia present on admission.

PROGRESS Responded well to aminophyline, pethidine, digoxin and mersalyl and on 2nd. day B. P. was 135/85, pulse 69/minute regular. Congestive failure cleared by end of first week and subsequent progress uneventful. After discharge, fairly well but troubled by effort on moderate exertion, palpitations, and slight angina, (Category III). Re-admitted with severe congestive failure and dyspnoea at rest (Category IV) in March, 1964, and discharged after 3 weeks much improved. Patient alive but rather dyspnoeic 3 years after infarction.

CASE NO. C.10. L. D. Male, 66 years. Registration No. 167031

Admitted 12.9.61, Died 25.9.61, (13 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	1	4	4	20

Moderately shocked on admission, B. P. 170/115 falling within one hour to 120/70, pulse 68/minute regular. Moderate congestive failure present. History of hypertension for a few years.

E.C.G. Posterior infarct with Q S wave changes in leads III, Avf. P-R interval on admission 0.26 seconds, subsequently 0.22 seconds.

TRANSAMINASES	Day	1	3
	S.G.O.T.	18	90
	S.G.P.T.	17	31

HAEMOGLOBIN 100% (cyan method)

PROGRESS Improved a little at first but on 2nd. day B.P. fell to 115/70, pulse 92/minute regular. Steroids had no effect on potential heart block and congestive failure became worse despite therapy with digoxin, chlorothiazide and later mersalyl.

POSTMORTEM NO. 162/61.

Pericardial fluid was normal but there was a fibrin exudate over posterior wall of heart. Heart weighed 390g., right ventricle was a little hypertrophied and valves were normal. There was a recent, transmural, yellow /

yellow/red infarct involving posterior ⅓ of interventricular septum, and adjacent 2/3rds of posterior wall. There was gross coronary atheroma and the right vessel was occluded by an antemortem thrombus 3mm. from its origin.

L V WALL THICKNESS Normal wall 17mm. Infarct 16mm.

CASE NO. C.11. H. C. Female, 71 years. Registration No. 174548.

Admitted 12.12.61, Died 15.12.61, (3 Days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	0	5	4	4	4	20

Admitted shocked, B.P. 160/70 falling within 3 hours to 130/70, pulse 128/minute regular. Moderate oedema of legs and sacral area present. No relevant previous history.

E.C.G. Anteroseptal infarct, Q S wave changes in leads I, V1 - V4. Sinus tachycardia on 1st. day; P-R interval constant 0.18.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	22	22	43
	S.G.P.T.	16	16	28

HAEMOGLOBIN 83% (Sahli).

PROGRESS Shock improved after receiving pethodine, B.P. 140/76 on 3rd. day, but despite steroids, digoxin and chlorothiazide, congestive failure became steadily worse and death occurred late on 3rd. day.

CASE NO. C.12. M. T. Female, 61 years. Registration No. 180092

Admitted 14.10.63, Discharged 15.11.63, (32 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	0	4	4	21

Admitted shocked, B.P. 110/60, pulse 95/minute irregular. No evidence of failure. Previous myocardial infarct in 1960; angina and dyspnoea on effort since then.

E.C.G. Posterolateral infarct; Q S wave changes in leads II, III, Avf, V5 - V6. Auricular fibrillation during first day and occasionally thereafter; P-R interval 0.17 seconds.

TRANSAMINASES

Day	2	3	4	5
S.G.O.T.	35	18	13	46
S.G.P.T.	23	31	43	12

ELECTROLYTES

Day	Sodium	Potassium	Chloride	CO_2
15	145	4.3	104	25.7
17	143	5.1	107	25.5
19	141	5.0	106	22.7
24	139	4.7	107	24.0
30	140	4.0	100	25.5

HAEMOGLOBIN 102% (Cyan method)

PROGRESS /

PROGRESS Improved a little at first after receiving morphia and hydrocortisone but then collapsed with persistent auricular fibrillation. Defibrillator used four times on 1st. day before rhythm restored to near normal; fibrillation did occur occasionally thereafter but was not troublesome. Went into congestive failure and despite digoxin and chlorothiazide remained in failure for 2 weeks. After discharge remained extremely dyspnoeic on slight effort, with severe angina and slight congestive failure at times (Category IV). Patient still in this condition one year after the infarct.

CASE NO. D.1. N. M. Male, 67 years. Registration No. 229494

Admitted 5.11.63, Discharged 11.12.63, (36 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	1	1	4	4	19

Admitted with mild shock, B.P. 120/85, pulse 132/minute irregular, with basal crepitations but no neck vein over filling or peripheral oedema.

History of attacks of chest pain thought to be myocardial, in July, 1955, October, 1955, April, 1956 and May, 1960. Frequent attacks of angina in preceding 6 months.

E.C.G. Acute anteroseptal infarct with Q S wave changes in leads I, II, Avl, V1 - V6. P-R interval 0.16 seconds on admission, 0.15 seconds on 8th. day, 0.16 seconds on 28th. day. Sinus tachycardia on admission and frequent ventricular extrasystoles on 1st. day and during 2nd. and 3rd. weeks.

TRANSAMINASES	Day	1	2	3	4
	S.G.O.T.	148	133	70	16
	S.G.P.T.	46	76	32	11

HAEMOGLOBIN 111% (cyan method)

PROGRESS Improved initially but at the end of first week ventricular extrasystoles became troublesome and oedema of legs and sacrum developed. Extrasystoles not controlled initially by quinidine but abolished after 12 days and soon thereafter peripheral oedema regressed. Subsequent progress uneventful /

uneventful though an occasional extrasystole was seen up to the time of discharge. After discharge was well and almost symptom free (Category II) but within a few months dyspnoea again restricted activities (Category III); still alive at one year after the infarct.

CASE NO. D.2. J. A. Male, 63 years. Registration No. 136752

Admitted 17.1.61, Died 20.1.61, (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	0	7	4	4	4	21

Very shocked on admission, B.P. 80/30, pulse 72/minute irregular, with gross peripheral oedema. No relevant past history.

E.C.G. Anteroseptal infarct, Q. S. wave changes in leads I, V1 - V6. P-R interval 0.20 seconds. On 2nd. day complete heart block.

TRANSAMINASES	Day	1
	S.G.O.T.	110
	S.G.P.T.	27

HAEMOGLOBIN 90% (Sahli)

PROGRESS Never recovered from initial shock, B.P. never above 80/30, and often 40/?. Extrasystoles frequent and congestive failure slowly became worse. At end of 2nd. day complete heart block supervened, pulse rate 45/minute and congestive failure rapidly worsened. Despite administration of digoxin, chlorothiazide, hydrocortisone, Betamethasone and auinidine, there was no improvement and death occurred late on the 3rd. day.

POSTMORTEM NO. 14/61.

Pericardial sac distended with straw coloured fluid while anterior and lateral aspects of heart were injected and covered by a fine fibrin exudate. /

exudate. Heart weighed 460g., left ventricle was prominent and aortic valve ring was stretched, the valve being minimally incompetent. There was a massive transmural infarct involving virtually the entire interventricular septum, all of anterior wall with apex, and a small adjacent area of lateral wall. Both coronary arteries showed gross atheroma for the first 20mm. of their length and the left was occluded by an antemortem thrombus 15mm. from its origin.

L.V. WALL THICKNESS Normal wall 18mm. Infarct 15mm.

CASE NO. D. 3. E. C. Male, 45 years. Registration No. 168151

Admitted 20.10.61, Discharged 19.11.61 (30 Days)

	A. S.	P. H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	0	6	5	4	4	4	23

Severely shocked, B. P. 40/?, pulse 95/minute irregular, marked oedema of legs and sacrum. Was just recovering from an infarct sustained on 26.9.61

E.C.G. Anteroseptal infarct with Q S wave changes in leads I, Avl, V1 - V5. P-R interval constant at 0.18 seconds. Late on 1st day ventricular fibrillation noted for a brief period.

TRANSAMINASES	Day	1
	S.G.O.T.	118
	S.G.P.T.	23

HAEMOGLOBIN 105% (cyan method)

PROGRESS Improved by morphia, digoxin, chlorothiazide and steroids. B. P. late on 1st. day 125/70. Quinidine immediately checked the ventricular extrasystoles but at the end of the 1st. day the patient collapsed and E.C.G. confirmed the presence of ventricular fibrillation. This did not respond to external cardiac massage or to the use of a defibrillater. Normal rhythm was restored almost immediately after the administration of procaine amide /

amide. Ventricular extrasystoles recurred briefly on 9.11.61 but otherwise subsequent recovery was uneventful.

After discharge was well at first but on 3.2.62 sustained a further severe infarct, was re-admitted to the survey as Case No. D.4, and died on 5.2.62.

CASE NO. D. 4. E. C. Male, 45 years. Registration No. 168151

Admitted 3.2.62, Died 5.2.62 (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	0	6	5	4	4	4	23

Admitted gravely shocked, B. P. 110/80 falling to 35/10, with marked neck vein distension, slight pulmonary oedema but no peripheral oedema. Two previous infarcts in 1961, the second being severe and included in this survey (see Case No. D. 3).

E.C.G. Fresh changes in chest leads V1 - V4 suggesting extension of anterior infarct. P-R interval 0.16 seconds on admission, 0.18 on 3rd. day. Ventricular extrasystoles present throughout admission.

TRANSAMINASES

Day	1	2	3
S.G.O.T.	118	95	46
S.G.P.T.	55	37	16

PROGRESS Improved slowly at first but ventricular extrasystoles persisted and were not checked by procaine amide or steroids. Congestive failure persisted despite digoxin and mersalyl. On the 3rd. day the B.P. fell to 35/10 and death occurred suddenly.

POSTMORTEM NO. 35/62.

The pericardial sac was obliterated and fine adhesions were present between the two layers of the pericardium, especially over the anterior wall. The heart weighed 720g. and both ventricles were massively hypertrophied.

The /

The aortic valve ring was slightly stretched and the valve was minimally incompetent. A very recent haemorrhagic infarct involved apex and small parts of the anterior wall. Slightly older areas of infarction involved anterior 2/3rds of interventricular septum, the remainder of anterior wall and the adjacent half of lateral wall. The most recent area of infarction in anterior wall was thinned and shows slight aneurysmal dilatation. The right coronary artery was narrowed by atheroma particularly at a point 25mm. from its origin. The left artery showed similar gross atheroma and a broken down plaque caused complete occlusion just above the junction of the inter-:ventricular branch.

L.V.WALL THICKNESS Normal wall 24mm. New Infarct 4mm. Old massive infarct 17mm.

CASE NO. D. 5. L. McL. Female, 76 years. Registration No. 215555

Admitted 23.4.63, Died 30.4.63, (7 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	7	4	0	4	24

Admitted in gross shock. B.P. 110/85, pulse 100/minute steady, with moderate ankle oedema. Cardiac invalid due to angina; myocardial infarct in 1950.

E.C.G. Acute anteroseptal infarct with old posterior infarct. Q S wave changes in leads I, Avl, V1 - V3. P-R interval steady at 0.16 seconds.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	36	22	15
	S.G.P.T.	15	29	10

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	6	137	3.9	97	25.4

HAEMOGLOBIN 90% (Sahli)

PROGRESS Did not recover from shock for two days despite therapy, with steroids and morphia but B. P. stabilised at 110/60 on 3rd. day. Despite digoxin and mersalyl therapy congestive failure worsened gradually till death late on the 7th. day.

CASE NO. E. 1. A.F. Female, 66 years. Registration No. 215783

Admitted 25.4.63, Discharged 29.5.63, (34 Days)

PROGNOSTIC INDEX	A.S.	P.H.	S.	F.	R.	E.	TOTAL
	3	3	1	4	4	3	18

Admitted slightly shocked, B.P. 140/80, Pulse 56/minute regular, with marked distention of neck veins but no peripheral oedema. History of angina for 12 years with dyspnoea on effort recently.

E.C.G. Anteroseptal infarct involving QR waves in leads I, Avl, V1-V5. P.R. interval 0.19 seconds. Sinus bradycardia present on admission, and QS waves became affected on 5th day.

TRANSAMINASES	Day	1	2	4
	S.G.O.T.	62	106	46
	S.G.P.T.	45	72	68

HAEMOGLOBIN 92% (Sahli)

PROGRESS Improved speedily after receiving steroids, and mersalyl. B.P. remained around 14/80, and pulse rate rose to 80/minute at end of first day. Subsequent progress uneventful. After discharge quite well apart from slight angina on effort at 18 months.

CASE NO. E.2. E.W. Female, 76 years Registration No. 214126.

Admitted 2.5.63, Died 4.5.63. (3 Days)

PROGNOSTIC INDEX	A.S.	P.H.	S.	F.	R.	E.	TOTAL
	3	3	5	4	0	4	19

Admitted shocked 105/70, pulse 92/minute regular, with peripheral oedema, and distended neck veins. History of angina for years, and dyspnoea on effort for 8 months.

E.C.G. Anterior infarct with QS wave changes in leads I, Avl, V3-V6. P.R. interval 0.18 seconds.

TRANSAMINASES Day 2

S.G.O.T. 110

S.G.P.T. 25

ELECTROLYTES

Day	Sodium	Potassium	Chloride	Co_2
2	140	43	102	27.4

HAEMOGLOBIN 102% (cyan method).

PROGRESS Responded quickly to sedation with omnopon, B.P. 128/72 at end of 1st day. Mersalyl and digoxin were given in addition to Betamethasone and congestive failure began to improve but death occurred unexpectedly on the 3rd day. Permission for autopsy examination was refused.

CASE NO. E.3. E.M. Female, 59 years. Registration No.

Admitted 2.4.63, Discharged 10.6.63, (68 Days)

PROGNOSTIC INDEX	A.S.	P.H.	S.	F.	R.	E.	TOTAL
	2	1	5	4	4	4	20

Moderately shocked on admission, B.P. 90/60, pulse 68/minute irregular, with marked oedema of legs and sacrum. History of angina for 2 years.

E.C.G. Acute posterior infarct with 2:1 heart block. 2 hours after receiving steroids, sinus rythum was restored, P.R. interval 0.2 - 0.22 seconds on first day, 0.20 seconds on 8th day, and 0.16 seconds on 28th day. In later tracings there were appearances suggestive of an old anterior infarct.

TRANSAMINASES	Day	1	2
	S.G.O.T.	26	200
	S.G.P.T.	18	49

ELECTROLYTES

Day	Sodium	Potassium	Chloride	Co_2
11	142	5.1	102	28.0
18	143	4.7	105	26.8

HAEMOGLOBIN 88% (Sahli).

PROGRESS 2:1 heart block abolished only 2 hours after receiving Betamethasone. Despite treatment with digoxin and mersalyl congestive failure persisted at first, but eventually improved slowly. After discharge moderate dyspnoea persisted with occasional episodes of congestive failure (category III) but patient /

contd./

remained alive at 18 months.

CASE NO. E. 4. N.S. Male, 57 years Registration No. 183907.

Admitted 30.4.62, Discharged 2.6.62, (33 Days).

PROGNOSTIC INDEX	A.S.	P.H.	S.	F.	R.	E.	TOTAL
	1	3	5	4	4	4	21

Admitted shocked, B.P. 105/60, pulse 95/minute irregular, with distended neck veins and slight ankle oedema. Past history of ? cerebral in 1945.

E.C.G. Anterolateral infarct, QS wave changes in leads I, II, V4 - V6. P.R. interval 0.16 seconds at all times.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	22	55	5
	S.G.P.T.	25	22	14

HAEMOGLOBIN 100% (Sahli).

PROGRESS Improved after sedation with omnopon, and congestive failure improved after receiving digoxin and mersalyl. The ventricular extrasystoles were resistant to procaine amide therapy but were partially controlled with quinidine, although even up to the time of discharge occasional extrasystoles occurred. After discharge was troubled by dyspnoea on effort, and sustained a further severe myocardial infarct on 24.1.63 being readmitted to the survey as Case No. A. 24. After recovery from this infarct he was again troubled by dyspnoea and angina on effort (category III) but was alive at 33 months after the initial infarct.

CASE NO. F. 1. S. N. Male, 65 years. Registration No. 156272

Admitted 28.11.61, Discharged 2.1.62. (35 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	1	4	0	3	17

Admitted with slight shock, B.P. 120/80, pulse 85/minute regular, with slight ankle oedema, neck vein over filling and pulmonary oedema. Previous history of myocardial infarct in May, 1961, and of dyspnoea on effort for many years.

E.C.G. Lateral infarct; Q R wave changes in leads II, Avl., V4 - V6. P-R interval 0.18 seconds on admission, 0.16 seconds thereafter.

TRANSAMINASES	Day	1	2	4
	S.G.O.T.	141	284	51
	S.G.P.T.	210	380	216

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	21	137	47	101	23.2

HAEMOGLOBIN 95% (cyan method)

PROGRESS Soon recovered from shock after sedation with pethidine. Chlorothiazide given for a few days and obvious congestive failure was absent after the 5th. day. Subsequent progress uneventful. After discharge symptom fre for one year (Category I), slight dyspnoea on effort for 2nd. year (Category II) and more severe dyspnoea thereafter (Category III) though the patient was still alive at 3 years after the infarct.

CASE NO. F. 2. W. M. Male, 72 years. Registration No. 183289

Admitted 3.4.62, Discharged 3.5.62 (30 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	0	4	4	3	17

Not shocked on admission, B. P. 140/80, pulse 72/minute irregular, with slight ankle oedema and neck vein distension. History of dyspnoea on effort for a few years.

E.C.G. Posterior infarct, involving Q S waves in leads II, III, Avf. P-R interval 0.16 seconds, but auricular fibrillation present intermittently during first 8 days.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	20	33	172
	S.G.P.T.	25	32	47

HAEMOGLOBIN 102% (Sahli)

PROGRESS Improved speedily with digoxin and chlorothiazide. The auricular fibrillation present on admission was speedily partially controlled and was abolished completely after 8th. day. Subsequent progress uneventful and after discharge was relatively well (Category II) having a slight exertional dyspnoea at 30 months after the infarct.

CASE NO. F. 3. T. K. Male, 50 years. Registration No. 220064

Admitted 22.6.63, Discharged 20.7.63, (28 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	0	3	5	1	4	4	17

Admitted moderately shocked, B. P. 84/50, pulse 120/minute regular alternating with periods at 60/minute mostly regular. Basal crepitations present but no peripheral oedema was present. History of dyspnoea on effort for one year.

E.C.G. Anterolateral infarct. Q S waves affected in leads I, II, V3 - V4. 2 : 1 heart block present at times with periods of auricular flutter. At times sinus rhythm was present, P-R interval 0.28 seconds.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	100	140	52
	S.G.P.T.	182	230	130

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	2	142	4.7	102	26.8
	8	141	4.9	102	25.2

HAEMOGLOBIN 105% (cyan method)

PROGRESS Shock quickly controlled after sedation with morphia. Digoxin and chlorothiazide were given during first week and evidence of failure was absent after three days. The atrioventricular conduction did not improve and when seen 6 months after discharge the P-R interval remained 0.28 seconds with short periods of 2 : 1 block. After discharge, remained quite well for

6 months (Category II) but throughout the next 2 years dyspnoea on effort was troublesome and slight ankle oedema was present at times (Category III).

CASE NO. F. 4. G. R. Male, 56 years. Registration No. 191105

Admitted 4.7.62, Discharged 3.8.62. (30 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	1	3	1	4	4	4	17

Admitted slightly shocked, B.P. 140/85, pulse 50-90/minute, irregular, with slight ankle oedema and neck vein over-filling. History of effort dyspnoea for one year.

E.C.G. Bizarre picture initially with periods of complete heart block alternating with periods of sinus rhythm: runs of ventricular extrasystoles every few minutes. Evidence of acute posterior infarct affecting Q S waves in leads II, III, Avf. P-R interval variable on admission, 0.16 seconds on 2nd. day and subsequent days.

TRANSAMINASES	Day	2	3	4	5
	S.G.O.T.	15	12	15	16
	S.G.P.T.	15	10	40	9

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	8	138	4.8	100	26.7
	14	141	4.7	102	25.1

PROGRESS Sedated with morphia, and treated with quinidine, digoxin and chlorothiazide. On 2nd. day B.P. 130/75, pulse 92/minute irregular, and evidence of congestive failure almost absent, Ventricular extrasystoles did not disappear until the 6th. day, though their occurrence was infrequent after /

after the 2nd. day. Subsequent progress uneventful. After discharge, well for nearly 2 years, having only slight dyspnoea on effort, (Category II) thereafter dyspnoea was more troublesome (Category III) though the patient was still alive 30 months after the infarct.

CASE NO. F. 5. E. B. Female, 73 years. Registration No. 154252

Admitted 23.3.61 Discharged 27.5.61 (65 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	0	4	0	4	17

Admitted in severe congestive failure, but not shocked, B.P. 145/90, pulse 80/minute regular. History of hypertension for years, and a myocardial infarct in October, 1960.

E.C.G. Lateral infarct affecting Q S waves in leads II, V4 - V6. P-R interval 0.18 seconds on admission, 0.16 seconds on 8th. day, 0.16 seconds on 28th. day.

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	7	144	4.6	106	23.9
	14	140	4.2	102	24.6
	21	138	4.5	100	25.8
	26	143	4.4	101	25.1

HAEMOGLOBIN 90% (Sahli)

PROGRESS Congestive failure responded slowly to digoxin, chlorothiazide and later mersalyl, and on discharge failure was absent. Was quite well after discharge having only slight dyspnoea on effort (Category II), but died on 20th. July, 1962 a few hours after sustaining a cerebral thrombosis.

CASE NO. F. 6. G. S. Male, 64 years. Registration No. 156437

Admitted 30.5.61, Died 30.6.61 (30 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	3	1	4	4	3	17

Slightly shocked on admission with persistant tachycardia, B. P. 128/72, pulse 120 - 130 / minute during first 5 days. Moderate oedema of legs present. History of intermittent claudication recently; a known diabetic.

E.C.G. Posterior infarct involving Q S waves in leads II, III, Avf. P-R interval steady at 0.15 seconds. Sinus tachycardia present during first 5 days.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.		155	35
	S.G.P.T.		68	130

HAEMOGLOBIN 89% (Sahli)

PROGRESS Improved only slightly during first week despite treatment with digoxin, chlorothiazide and later mersalyl. Sinus tachycardia subsided after 5th. day, but thereafter congestive failure slowly became worse and death occurred on the 30th. day.

POSTMORTEM /

POSTMORTEM (File No. 640723).

Pericardium and pericardial sac normal. Heart weighed 420g. and left ventricle was a little prominent. The valves appeared normal. In posterior wall there was a relatively recent though fibrosed infarct up to 30mm. in diameter affecting a small adjacent area of interventricular septum. The coronary arteries showed gross atheroma, and a few areas of ulceration but there was no antemortem thrombus.

L. V. WALL THICKNESS Normal wall 17mm. Infarct 14mm.

CASE NO. F. 7. W. McG. Male, 60 years. Registration No. 152133

Admitted 13.5.62, Died 23.6.62 (40 days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	1	4	0	4	17

Admitted slightly shocked and in moderate congestive failure. B.P. 110/70, pulse 90/minute, regular. History of dyspnoea on effort and angina for 2 years Anteroseptal infarct 2 years ago.

E.C.G. Posterior infarct, Q S wave changes in leads II, III, Avf. Old anterior infarct. P-R interval steady at 0.15 seconds.

TRANSAMINASES	Day	3	4	5
	S.G.O.T.	170	75	158
	S.G.P.T.	325	290	340

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2	Protein
	14	137.2	4.26	98	20.6	17

HAEMOGLOBIN 99% (Sahli)

PROGRESS Improved a little after administration of omnopon, digoxin and chlorothiazide, B. P. 125/80, on 3rd. day, but then slowly went into irreversible congestive failure and death occurred on the 40th. day.

POSTMORTEM NO. 127/62.

The pericardial sac and heart surfaces were normal; heart weighed 625g. and left ventricle was markedly hypertrophied. The aortic valve cusps were slightly thickened, and the valve was slightly incompetent. There was

a /

a relatively recent infarct occupying almost all of posterior wall and a small adjacent area of interventricular septum. An old well healed infarct 15mm. in diameter was present in anterior wall. The coronary arteries showed slight atheroma but the ostia were greatly narrowed by gross atheroma of the ascending aorta.

L V WALL THICKNESS Normal wall 24mm. Infarct 15mm.

CASE NO. F. 8. J. G. Female, 72 years. Registration No. 163690

Admitted 5.8.61, Discharged 5.8.61, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	1	5	0	4	4	17

Admitted shocked but not in failure, B.P. 100/7, pulse 88/minute irregular. Past history of angina for one year.

E.C.G. Anteroseptal infarct, Q S wave changes in leads I, Avl V1 - V5. P-R interval 0.18 seconds. Multiple ventricular extrasystoles present.

TRANSAMINASES	Day	1
	S.G.O.T.	162
	S.G.P.T.	21

PROGRESS After administration of morphis, there was some improvement, B.P. rising to 120/80, but at the end of the first day the petient collapsed again and died within minutes.

CASE NO. F. 9. H. C. Female, 79 years. Registration No. 192380

Admitted 26.7.62. Died 27.7.62. (2 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	7	0	0	4	17

Admitted grossly shocked, B. P. 80/?, pulse 100/minute regular but often barely perceptible. No failure present initially. History of cerebral thrombosis in 1958.

E.C.G. Anteroseptal infarct affecting Q S waves in leads I, Avl. V1 - V4. P-R interval 0.20 on admission, falling to 0.17 seconds on second day.

TRANSAMINASES	Day	2
	S.G.O.T.	175
	S.G.P.T.	71

PROGRESS Never recovered from shock though sedated with morphia, and in spite of treatment with digoxin and mersalyl, severe congestive failure developed. Death occurred on the 2nd. day.

POSTMORTEM NO. 148/62.

The pericardial sac was slightly distended with straw coloured fluid and the anterior aspect of the heart was markedly injected. The heart weighed 360g., the left ventricle was slightly prominent and the aortic valve ring was slightly stretched. A recent yellowish/haemorrhagic infarct was seen involving most of the posterior wall together with the posterior $\frac{1}{3}$ of interventricular septum. The coronary arteries showed gross atheroma and the right vessel was occluded by an antemortem thrombus 7mm. from its origin, where /

where the lumen was greatly narrowed by an atheromatous plaque.

L V WALL THICKNESS Normal wall 14mm. Infarct 14mm.

CASE NO. F.10. J. P. Female, 69 years. Registration No. 181619

Admitted 13.3.62, Died 27.3.62 (14 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	6	1	4	0	3	17

Admitted slightly shocked, B. P. 160/100, Pulse 84/minute regular, with marked oedema of legs and sacrum. Previous myocardial infarct in 1957.

E.C.G. Posterior infarct with Q R wave changes in leads II, III, Avf, V6. P-R interval steady at 0.20 seconds.

TRANSAMINASES	Day	2
	S.G.O.T.	140
	S.G.P.T.	69

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	7	142	4.6	103	24.7

PROGRESS Oedema worsened at first but following intensive therapy with digoxin, chlorothiazide and mersalyl, there was a gradual improvement. Death on the 14th. day was sudden and unexpected, and internal cardiac massage was of no avail.

POSTMORTEM No. 622672.

The pericardial sac contained a normal volume of straw-coloured fluid and the posterior surface of the heart was covered by a thin layer of fibrin, and appeared injected. Heart weighed 360g., the left ventricle was slightly /

slightly prominent and the valves were within normal limits.

An enormous yellowish/white infarct was found involving the entire posterior wall, small portions of lateral wall, and the posterior ⅓ of interventricular septum. Small areas at the junction of septum and posterior wall were densely fibrosed and were considered to represent an older infarct. The coronary arteries showed moderate atheroma and the right was occluded by an antemortem thrombus close to its origin.

L V WALL THICKNESS Normal wall 16mm. Infarct 14mm.

CASE NO. F. 11. M. F. Female, 70 years. Registration No. 161330

Admitted 21.5.63, Died 27.5.63, (7 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	0	4	0	4	17

Not shocked on admission but in marked congestive failure. B.P. 115/70, pulse 85/minute, regular. Previous myocardial infarct in December, 1962.

E.C.G. Acute posterolateral infarct and old posterior infarct. Q S wave changes in leads II, III, Avf. V4 - V6. P-R interval steady at 0.16 seconds.

TRANSAMINASES	Day	1	2	3	4
	S.G.O.T.	37	155	194	98
	S.G.P.T.	40	49	60	68

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	2	146	4.2	109	18.4
	6	142	4.6	104	23.5

PROGRESS Despite administration of digoxin and mersalyl, went into irreversible congestive failure and died on the 7th. day.

CASE NO. F. 12. J. R. Female, 69 years. Registration No. 175608

Admitted 30.12.61, Died 1.1.62 (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	1	5	4	0	4	17

Admitted shocked, B. P. 100/65, pulse 90/minute regular, with marked oedema of legs and sacrum. History of angina for 14 years.

E.C.G. Posterior infarct, Q S waves in leads II, III, Avf. P-R interval 0.18 seconds on admission, 0.16 seconds on 3rd. day.

TRANSAMINASES	Day	2
	S.G.O.T.	156
	S.G.P.T.	45

PROGRESS Favourable initial response to morphia, digoxin and chlorothiazide but on 2nd. day congestive failure became much more marked and death occurred early on the 3rd. day.

CASE NO. F. 13. A. R. Male, 68 years. Registration No. 183958

Admitted 6.4.62, Died 28.4.62, (22 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	0	4	4	3	17

Not shocked but in congestive failure on admission. B.P. 130/75, pulse 125/minute regular throughout first two days. History of dyspnoea on effort for 18 months.

E.C.G. Posterior infarct affecting Q R waves in leads II, III, Avf. Supraventricular tachycardia throughout first 2 days, and intermittently for next 4 days, P-R interval 0.16 seconds. After 6th. day auricular fibrillation.

TRANSAMINASES.	Day	1	2	3	4
	S.G.O.T.	85	125	263	41
	S.G.P.T.	43	36	36	28

HAEMOGLOBIN 89% (cyan method)

PROGRESS Digitalised and given procaine amide on admission but the supra-:ventricular tachycardia did not subside completely for 6 days when it was replaced by auricular fibrillation. Congestive failure improved gradually, but became more severe during the last few days of life despite the adminis-:tration of mersalyl. A hypostatic pneumonia was noted on the day of death.

POSTMORTEM /

POSTMORTEM NO. 622683.

The pericardium and pericardial sac appeared normal. Heart weighed 410g. and left ventricle was a little prominent. There was a recent whitish infarct involving 2/3rds of posterior wall and a little of interventricular septum. The coronary arteries were greatly narrowed by atheroma and the right was occluded by an antemortem thrombus 5mm. from its origin.

L V WALL THICKNESS Normal wall 15mm. Infarct 11mm.

CASE NO. F. 14 L. G. Male, 64 years. Registration No. 150829

Admitted 19.11.61, Discharged 26.12.61, (37 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	4	0	1	18

Admitted shocked, B.P. 110/68, pulse 85/minute regular, with slight ankle oedema and neck vein over filling. History of myocardial infarct in 1960.

E.C.G. Recent anterior infarct. T wave changes only seen, on leads I, Avl. V3 - V6. P-R interval steady at 0.16 seconds. Old posterior infarct present.

TRANSAMINASES	Day	1	2
	S.G.O.T.	125	28
	S.G.P.T.	260	73

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	3	138	4.6	105	24.6
	10	139	4.4	102	25.0
	22	134	5.6	99	24.0

HAEMOGLOBIN 93% (Sahli)

PROGRESS Made steady uneventful recovery. Chlorothiazied used during first week. Died on 1.4.62 following a further myocardial infarct (as Case F69).

CASE NO. F. 15. W. B. Male, 68 years. Registration No. 207743

Admitted 1.2.63 Discharged 11.3.63, (39 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	0	4	4	4	18

Admitted with moderate oedema of legs and marked neck vein over filling, B.P. 140/80, pulse 75/minute irregular. History of dyspnoea and angina on effort for one year.

E.C.G. Anteroseptal infarct, Q S waves affected in leads I, V1 - V4. P-R interval 0.15 seconds on admission, 0.17 seconds on 8th. day and 0.16 seconds on 28th. day. Many auricular extrasystoles on admission.

TRANSAMINASES	Day	1	2
	S.G.O.T.	110	80
	S.G.P.T.	30	27

PROGRESS Responded well to sedation with morphia and to digoxin and chlorothiazide. A chest infection was present from 5.2.63 until 12.2.63, but responded to tetracycline. After discharge was well at first having only slight effort dyspnoea, (Category II), but died on 17.5.63, two days after sustaining a further myocardial infarct.

CASE NO. F.16. E. C. Female, 65 years. Registration No. 229498

Admitted 5.11.63, Discharged 11.12.63, (36 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	0	4	4	4	18

Admitted with slight ankle oedema, marked neck vein over filling, but no shock, B. P. 150/110, pulse 80/minute irregular. Past history of hypertension and dyspnoea on effort.

E.C.G. Anteroseptal infarct with 2 : 1 heart block. On 15.11.63 block became complete but within a few hours sinus rhythm was reduced spontaneously with a P-R interval of 0.24 seconds.

TRANSAMINASES	Day	2	3	4
	S.G.O.T.	34	51	12
	S.G.P.T.	12	14	17

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2	Protein
	8	142	4.3	105	21.6	

PROGRESS Remained in failure throughout first week despite digoxin and mersalyl therapy. On 18.11.63 complete heart block ensued, pulse 38/minute, but within a few hours sinus rhythm returned spontaneously and thereafter recovery was uneventful. One year after the infarct the patient was alive, though troubled by dyspnoea on effort and slight ankle oedema (Category III).

CASE NO. F. 17 G. B. Male, 62 years. Registration No. 166688

Admitted 8.7.63, Discharged 7.8.63, (30 Days)

	A. S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	3	1	4	4	4	18

Admitted with slight shock, B.P. 100/60, pulse 120/minute irregular, with slight ankle oedema. History of gangrene of left leg, amputated above knee in 1957.

E.C.G. Posterolateral infarct, Q S waves leads II, III, Avf, V5 - V6. P-R interval indeterminate on admission due to continuous auricular fibrillation, on 28th. day 0.18 seconds.

TRANSAMINASES	Day	2
	S.G.O.T.	140
	S.G.P.T.	34

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	2	143	4.9	102	26.1
	8	142	4.6	103	24.7
	15	140	4.5	101	25.2

PROGRESS Reseponded well to digoxin and chlorothiazide, auricular fibrillation became largely controlled and congestive failure was absent by 5th. day. After discharge continued to have slight congestive failure at times (Category III) but was alive one year after the infarct.

CASE NO. F.18 E. L. Male, 45 years. Registration No. 198231

Admitted 11.10.62, Discharged 13.11.62, (33 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	0	1	5	4	4	4	18

Admitted shocked, B. P. 90/40, pulse 45/minute regular, with neck vein over filling and peripheral oedema. History of slight angina for one year.

E.C.G. Anterolateral infarct with Q S waves in leads I, II, V2 - V5. P-R interval 0.16 seconds at all times. Sinus bradycardia on admission.

TRANSAMINASES	Day	1	2
	S.G.O.T.	122	250
	S.G.P.T.	250	44

HAEMOGLOBIN 106% (Sahli)

PROGRESS Responded well to morphia, digoxin and chlorothiazide; at end of first day B. P. 120/65, pulse 82/minute regular. Subsequent progress uneventful. After discharge remained symptom free (Category I) for 15 months, but thereafter experienced slight angina on effort (Category II) though remaining remarkably well at 2 years after the infarct.

CASE NO. F. 19. G. C. Male, 82 years. Registration No. 181604.

Admitted 13.3.62, Discharged 22.5.62, (69 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	1	4	0	4	18

Admitted only slightly shocked, B.P. 110/70, pulse 90/minute regular, but in marked congestive failure and with a chest infection. History of myocardial infarct years before.

E.C.G. Posterior infarct; Q S waves involved in leads III, Avf. P-R interval 0.19 seconds on admission, 0.20 seconds on 3rd. day, 0.17 seconds on 28th. day.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	62	128	74
	S.G.P.T.	29	38	32

HAEMOGLOBIN 83% (Sahli)

PROGRESS Slow improvement after administration of morphia, digoxin and mersalyl, though B. P. was 150/95 on 2nd. day and chest infection was swiftly controlled by tetracycline. Congestive failure was very slow to clear but was absent at the time of discharge. After discharge the patient was never well being crippled by dyspnoea, congestive failure and leg pain, (Category IV) for which he was re-admitted on 30.5.62. Despite treatment with digoxin and mersalyl periphoral oedema worsened, early gangrenous changes appeared in left foot, and death occurred on 6.7.62.

CASE NO. F. 20. J. McI. Female, 73 years. Registration No. 163689.

Admitted 5.8.61, Died 17.10.61, (93 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	0	4	4	4	18

In marked congestive failure on admission, but not shocked, B.P. 100/70, pulse 95/minute irregular. Past history of intermittent congestive failure in recent years;

E.C.G. Posterior infarct, Q S waves in leads II, III, Avf. Ventricular extrasystoles present on admission with auricular fibrillation much of the time after the 3rd. day. P-R interval when recordable 0.18 seconds during the first 3 days, 0.16 seconds thereafter.

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	29	137.8	4.8	101	23.1
	53	135.4	4.3	98	22.9

HAEMOGLOBIN 89% (Sahli)

PROGRESS Despite treatment with digoxin and mersalyl remained in congestive failure. Death occurred soon after an attack of ventricular extrasystoles.

CASE NO. F. 21 J. R. Female, 76 years. Registration No. 10520

Admitted, 4.2.61, Died 7.2.61, (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	0	7	4	0	4	18

Admitted grossly shocked, B. P. 40/?, pulse 100/minute barely perceptible, with marked congestive failure. No relevant past history.

E.C.G. Posterior infarct. Q S waves affected in leads III, Avf. P-R interval 0.14 seconds on admission, 0.15 seconds on 3rd. day.

PROGRESS Never recovered from shock, B. P. never above 80/?;despite administration of digoxin and mersalyl congestive failure never improved.

CASE NO. F. 22. J. B. Male, 66 years. Registration No. 165118.

Admitted 23.8.61, Died 2.9.61, (10 Days)

	A.S.	F.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	0	3	18

Admitted moderately shocked, B. P. 130/80, pulse 120/minute but soon falling to 90/minute regular. Slight peripheral oedema was present. Past history of hypertension for years; angina and intermittent claudication recently.

E.C.G. Posterior infarct; Q S wave changes in leads II, III, Avf. P-R interval 0.15 seconds at all times.

TRANSAMINASES	Day	1	2	3	5
	S.G.O.T.	56	98	134	42
	S.G.P.T.	21	35	45	51

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	7	146	4.9	106	29.0

PROGRESS Despite administration of digoxin and chlorothiazide the congestive failure became progressively more severe and death occurred on the 10th. day.

CASE NO. F. 23. E. G. Female, 60 years. Registration No. 169484.

Admitted 9.5.63, Died 17.5.63, (8Days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	3	1	4	4	4	18

Admitted slightly shocked, B.P. 180/100, pulse 88/minute regular alternating with periods at 115/minute irregular. Slight ankle oedema present. History of hypertension and effort dyspnoea for one year.

E.C.G. Anteroseptal infarct, Q S waves affected in leads I, V1 - V5. P-R interval steady at 0.21 seconds. Supraventricular tachycardia on first day.

TRANSAMINASES	Day	1	2	3
	S.G.O.T	88	250	141
	S.G.P.T.	34	63	67

HAEMOGLOBIN 109% (cyan method)

PROGRESS Despite the administration of digoxin and mersalyl, congestive failure became progressively more severe. After death internal cardiac massage was attempted without success.

POSTMORTEM NO. 630874.

The pericardial sac contained 270 ml. of straw-coloured fluid and the anterior aspect of the heart was dusky red in colour and covered by a slight fibrin exudate. Heart weighed 345g., the chambers were of normal size, and the valves were normal. A recent yellowish infarct occupied the anterior $\frac{1}{3}$ of interventricular septum, apex and $\frac{1}{4}$ of adjacent anterior wall. The coronary /

coronary arteries showed moderate atheroma and the left was occluded by an antemortem thrombus 12mm. from its origin.

L V WALL THICKNESS Normal wall 18mm. Infarct 16mm.

CASE NO. F. 24. J. S. Male, 68 years. Registration No. 205198

Admitted 6.1.63, Died 7.1.63, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	0	4	4	4	18

Not shocked but in marked congestive failure. B.P. 90/50, pulse 130/minute. Past history of dyspnoea on effort for several years.

E.C.G. Posterior infarct with Q S wave changes in leads II, III, Avf. P-R interval 0.14 seconds. Sinus tachycardia present on admission.

TRANSAMINASES	Day	1
	S.G.O.T.	138
	S.G.P.T.	55

PROGRESS Failed to respond to treatment with digoxin and chlorothiazide, and died within 6 hours.

POSTMORTEM No. 8/63.

The pericardial sac contained a normal volume of straw coloured fluid but the posterior wall of the heart was covered by a fibrin exudate and was extremely injected. Heart weighed 420g. and left ventricle was slightly hypertrophied. There was a rather ill-defined haemorrhagic infarct occupying the posterior $\frac{1}{3}$ of the interventricular septum and about $\frac{1}{2}$ of the adjacent posterior wall. The coronary arteries showed gross atheroma and were markedly narrowed; an antemortem thrombus completely occluded the right artery 10mm. from its origin.

L V WALL THICKNESS Normal wall 20mm. Infarct 20mm.

CASE NO. F. 25. D. L. Male, 67 years. Registration No. 139914.

Admitted 10.3.61, Died 11.3.61, (2 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	1	0	4	4	18

Admitted slightly shocked but not in failure, B.P. 110/68, pulse 92/minute irregular. Past history of myocardial infarct several years before.

E.C.G. Anteroseptal infarct; Q S wave changes in leads I, Avl, V1 - V5. Auricular fibrillation present.

PROGRESS Did not improve, B.P. soon fell to 80/? and congestive failure developed. In spite of digoxin and chlorothiazide therapy death occurred early on the 2nd. day.

POSTMORTEM (File No. 641070).

The pericardial sac contained virtually no fluid and the pericardial surfaces showed a fibrin exudate especially over the anterior wall. Heart weighed 385g., the chambers appeared of normal size and the valves were normal. A recent haemorrhagic infarct involved the anterior $\frac{1}{3}$ of the interventricular septum, apex and most of the anterior wall. A small old fibrosed infarct was present in the posterior wall. The coronary arteries showed gross atheroma with marked narrowing of the lumina though nowhere was any complete occlusion seen.

L V WALL THICKNESS Normal wall 20mm. Infarct 18mm.

CASE NO. F. 26. M. H. Female, 60 years. Registration No. 161897.

Admitted 4.7.63, Discharged 10.7.63, (6 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	1	1	4	4	18

Admitted with slight shock, B. P. 125/80, pulse 90/minute irregular, with basal crepitations but no peripheral oedema. Previous myocardial infarct in 1961.

E.C.G. Anteroseptal infarct, with Q S wave changes in leads I, Avl. V1 - V5. P-R interval 0.28 seconds with runs of 2 : 1 heart block on admission; third day 0.24 seconds.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	28	52	72
	S.G.P.T.	10	14	41

HAEMOGLOBIN 76% (cyan method). Film slightly hypochromic.

PROGRESS Responded well to rest alone and on 6th. day was transferred home to be cared for by a private nurse. Never well afterwards; in congestive failure in November and December, 1963, January, April and October, 1964, and severely restricted by dyspnoea on even slight effort (Category IV). Still alive at 15 months.

CASE NO. F. 27. M. B. Female, 75 years. Registration No. 192292

Admitted 26.7.62, Died 23.8.62. (29 Days)

PROGNOSTIC INDEX	A. S.	P.H.	S.	F.	R.	E.	TOTAL
	2	0	5	4	4	4	19

Admitted moderately shocked with moderate ankle oedema and slight neck vein over filling, B. P. 142/84, pulse 78/minute irregular. No relevant past history.

E.C.G. Anteroseptal infarct with Q S wave changes in leads I, V1 - V4. Auricular fibrillation on admission. P-R interval 0.16 seconds on 8th. day, 0.18 seconds on 28th. day.

TRANSAMINASES	Day	1	2
	S.G.O.T.	170	200
	S.G.P.T.	24	62

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	21	133	5.4	102	15.5
	23	144	4.7	103	24.2

HAEMOGLOBIN 76% (cyan method). Film slightly hypochromic.

PROGRESS Improved rapidly after administration of morphia, digoxin, and chlorothiazide, B.P. 140/80 at end of first day, pulse 68/minute. Auricular fibrillation troublesome initially but controlled largely after 30.7.62 following the administration of quinidine. Was quite well until 5.8.62 /

5.8.62 but thereafter congestive failure slowly became more severe. Digoxin, chlorothiazide and later mersalyl had no effect and death occurred on 23.8.62.

POSTMORTEM NO. MK/62.

Pericardial sac contained a normal volume of straw coloured fluid and the anterior wall of the heart was slightly injected. The heart weighed 370g., the chambers were all of normal size, and the valves were within normal limits. An extensive whitish infarct occupied the anterior ¼ of the interventricular septum, apex, and half of the anterior wall. The coronary arteries showed moderate atheroma and the left artery was occluded by an antemortem thrombus 7mm. from its origin.

L V WALL THICKNESS Normal wall 16mm. Infarct 12mm.

CASE NO. F. 28. J. J. Female, 76 years. Registration No. 213518

Admitted 14.4.63, Discharged 3.6.63, (51 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	3	1	4	4	4	19

Admitted slightly shocked with considerable oedema of legs and slight oedema of sacrum, B. P. 140/90, pulse 88/minute irregular.

E.C.G. Anteroseptal infarct affecting Q S waves in leads I, Avl V1 - V5. Auricular fibrillation present intermittently throughout first two weeks of admission but when absent P-R interval 0.18 seconds.

TRANSAMINASES	Day	2	3
	S.G.O.T.	84	45
	S.G.P.T.	20	17

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	3	144	4.64	106	26.3
	13	142	4.92	105	29.0

PROGRESS Responded slowly to digoxin and chlorothiazide. Auricular fibrillation not controlled fully for 2 weeks and congestive failure improved very slowly. After discharge was fairly well at first though troubled by dyspnoea on effort (Category III). Severe congestive failure developed again early in August, 1963; she was admitted again because of this on 2.8.63 and died that day.

CASE NO. F. 29. H. H. Female, 58 years. Registration No. 152601.

Admitted 29.12.62, Discharged 1.2.63, (34 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	3	5	1	4	4	19

Admitted moderately shocked with basal crepitations but no peripheral oedema or neck vein over filling. B. P. 130/75, pulse 130/minute, regular. History of dyspnoea on effort; a known diabetic.

E.C.G. Posterior infarct with Q S changes in leads II, III, Avf. Sinus tachycardia present on admission; P-R interval 0.18 seconds on admission, 0.15 seconds on 8th. day, 0.16 seconds on 28th. day.

TRANSAMINASES	Day	1	2
	S.G.O.T.	170	40
	S.G.P.T.	18	13

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	14	143	4.6	102	26.4

PROGRESS Improved rapidly after administration of morphia, digoxin and chlorothiazide. Recovery uneventful and after discharge was well (Category II) for a short time only. Congestive failure recurred and the patient was re-admitted from 22.5.63 until 26.6.63, and again from 24.8.63 until 29.8.63 because of this. Subsequently dyspnoea on effort was troublesome and bursts of ventricular extrasystoles were noted occasionally (Category III) though the patient was still alive at 18 months.

CASE NO. F. 30. S. T. Female, 77 years. Registration No. 158939

Admitted 23.7.62, Died 23.7.62 (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	1	7	4	0	4	19

Admitted shocked with marked oedema of legs and sacrum, B. P. 130/50, pulse 88/minute regular. History of angina for 3 years.

PROGRESS Sedated with morphia and given digoxin and mersalyl, but with no effect. B. P. fell to unrecordable levels within one hour and death occurred soon after.

CASE NO. F. 31. R. M. Female, 78 years. Registration No. 139343

Admitted 5.2.61, Died 9.2.61, (5 Days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	3	5	4	0	4	19

Admitted severely shocked, B. P. 100/60, pulse 80/minute regular, with marked neck vein over filling and slightoedema of ankles, legs and sacrum. Past history of dyspnoea on effort for several years.

E.C.G. Anterolateral infarct involving Q S waves in leads I, II, V1 - V6. P-R interval 0.16 seconds on admission, 0.15 seconds on 3rd. day.

PROGRESS Never well, B.P. never rose above 110/65 despite sedation with morphia. Congestive failure persisted, digoxin and mersalyl having no apparent effect. A hypostatic pneumonia was present on the 4th. day and death occurred early on the 5th. day.

CASE NO. F. 32. M. C. Female, 82 years. Registration No. 181444

Admitted 10.3.62, Died 13.3.62, (3 Days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	1	4	4	4	19

Slightly shocked with slight peripheral oedema on admission, B. P. 150/80, pulse 110/minute irregular. History of dyspnoea on effort for 5 years.

E.C.G. Anterior infarct with Q S wave changes in leads I, Avl, V4 - V6.

TRANSAMINASES	Day	2	3	4
	S.G.O.T.	88	107	41
	S.G.P.T.	60	64	44

HAEMOGLOBIN 88% (Sahli)

PROGRESS Treated with morphia, digoxin and chlorothiazide, but showed only a slight improvement. A right basal pneumonia developed and this did not respond to tetracycline. Death, late on the 3rd. day, was, however, sudden.

POSTMORTEM NO. 65/62.

The pericardial sac was obliterated by fine adhesions over the anterior wall, and the anterior aspect of heart was purple in colour and showed slight aneurysmal bulging. Heart weighed 310G. the chambers were of normal size and the valves seemed normal. There was a recent rather haemorrhagic infarct 30mm. in diameter confined to anterior wall above apex and not affecting interventricular septum. The coronary arteries showed little atheroma but an isolated calcified plaque almost occluded the left artery 5mm. from its origin.

L /

L V WALL THICKNESS Normal wall 15mm. Infarct 3mm.

CASE NO. F. 33. M. W. Male, 69 years. Registration No. 175869

Admitted 6.1.62, Died 9.1.62 (4 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	1	1	4	4	19

Only slightly shocked on admission, B.P. 105/65, but with basal crepitations but no ankle oedema, and pulse 110/minute irregular.

E.C.G. On admission, acute anterior infarct with involvement of Q S waves in leads I, V1 - V4; P-R interval 0.17 seconds on admission, 0.15 seconds on 3rd. day. Bursts of ventricular extrasystoles on first day. On 4th. day ventricular fibrillation just prior to death.

TRANSAMINASES	Day	2
	S.G.O.T.	142
	S.G.P.T.	38

HAEMOGLOBIN 85% (Sahli)

PROGRESS Transient shock, B.P. 130/70 6 hours after admission and ventricular extrasystoles were not seen after the first day. Appeared to do well but early on 4th. day collapsed, ventricular fibrillation was seen briefly on electrocardiograph tracings, and death occurred at once.

CASE NO. F. 34. J. B. Male, 46 years. Registration No. 140383

Admitted 6.4.61, Died 9.4.61, (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	0	6	5	4	0	4	19

Admitted shocked, B.P. 118/70, pulse 85/minute regular, with moderate oedema of legs. Previous myocardial infarct in 1956, angina in recent months.

E.C.G. Acute posterior infarct with Q S wave changes in leads II, III, Avf. Old anterior infarct. P-R interval 0.24 seconds at all times.

HAEMOGLOBIN 100% (Sahli)

PROGRESS Sedated with pethidine and B. P. rose to 130/74 after 12 hours. Despite administration of digoxin and mersalyl, congestive failure became progressively more severe and death occurred on the 3rd. day.

CASE NO. F. 35. W. F. Male, 72 years. Registration No. 156139.

Admitted 17.5.61, Died 29.5.61 (12 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	3	1	4	4	4	19

Admitted slightly shocked with distended neck veins and slight ankle oedema, B.P. 115/70, pulse 85/minute irregular at times. History of intermittent claudication for 3 years.

E.C.G. Anterior infarct. Q. S. waves affected in leads I, II, V1 - V3, Multiple ventricular extrasystoles on first day. P-R interval 0.22 seconds initially, 0.17 seconds on 8th. day.

TRANSAMINASES	Day	2
	S.G.O.T.	128
	S.G.P.T.	61

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	9	139	4.35	100	24.2

HAEMOGLOBIN 98% (Cyan method).

PROGRESS Condition never satisfactory. B. P. rate rose to 130/75 but then fell again to 80 - 120/60 - 70. Ventricular extrasystoles occurred at times and congestive failure never cleared completely despite administration of digoxin and chlorothiazide.

POSTMORTEM /

POSTMORTEM NO. M'k/61.

Pericardial sac contained a normal volume of straw coloured fluid and the anterior aspect of heart was markedly injected. Heart weighed 470g., left ventricle was moderately hypertrophied and the aortic valve ring seemed a little stretched though the valve was not incompetent. A yellowish infarct was present in lower part of anterior half of interventricular septum, apex and lower half of anterior wall. The coronary arteries showed gross atheroma and th left appeared occluded at several points by softened plaques.

L V WALL THICKNESS Not recorded accurately.

CASE NO. F. 36. A. C. Male, 86 years. Registration No. 220444.

Admitted 26.9.63, Died 30.9.63. (4 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	0	4	19

Admitted with moderate shock, B. P. 120/80, pulse 90/minute regular, and with moderate ankle oedema. Past history of dyspnoea on effort and angina, for 6 months.

E.C.G. Anteroseptal infarct; Q S wave changes in leads I, Avl. V1 - V3. P-R interval steady at 0.20 seconds.

TRANSAMINASES	Day	2
	S.G.O.T.	152
	S.G.P.T.	44

PROGRESS Rallied at first following sedation with morphia, B. P. 125/90 on 2nd. day, but subsequently congestive failure worsened despite the administration of digoxin and chlorothiazide, and death occurred on the 4th. day.

CASE NO. F. 37. H. H. Male, 60 years. Registration No. 215998

Admitted 28.4.63, Discharged, 14.5.63, (17 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	0	4	4	4	20

Admitted with marked oedema of legs and sacrum, but no shock, B.P. 100/60 rising within one hour to 130/75, pulse 93/minute irregular. Past history of myocardial infarct in 1949.

E.C.G. Anteroseptal infarct and left bundle branch block; Q S waves affected in leads I, Avl. V1 - V5. R-R interval constant at 0.24 seconds, once auricular fibrillation had settled.

TRANSAMINASES	Day	2	3
	S.G.O.T.	90	153
	S.G.P.T.	30	24

HAEMOGLOBIN 97% (Sahli).

PROGRESS Improved slowly at first due to persistent auricular fibrillation. The rhythm improved as soon as quinidine was administered but fibrillation returned immediately quinidine was withdrawn. With digoxin and mersalyl the congestive failure improved gradually and eventually mersalyl was replaced by chlorothiazide. Discharged from hospital on long term quinidine and chloro-thiazide and remained fairly well throughout next 18 months though dyspnoea on effort was at times troublesome and slight congestive failure recurred occasionally (Category III).

CASE NO. F. 38. S. H. Female, 66 years. Registration No. 201155

Admitted 11.11.62, Discharged 16.1.63, (66 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	0	5	4	4	4	20

Admitted shocked, with peripheral oedema and tachycardia, B.P. 110/70, pulse 130/minute regular. No relevant previous history.

E.C.G. New lateral infarct, old anteroseptal infarct, with Q S wave changes in lead II, V3 - V6. Sinus tachycardia present initially. P-R interval 0.18 seconds on admission, 0.16 seconds on 8th. day, 0.18 seconds on 28th. day.

TRANSAMINASES

Day	1	2	3
S.G.O.T.	25	162	100
S.G.P.T.	63	157	207

ELECTROLYTES

Day	Sodium	Potassium	Chloride	CO_2
1	141	4.5	105	27.8
20	143	4.4	104	26.5
33	141	4.7	102	27.1

HAEMOGLOBIN 94% (Sahli)

PROGRESS Very ill initially but responded by the end of the first day to morphia, aminophyline, oxygen, digoxin and chlorothiazide. On 2nd. day B.P. /

B. P. 130/75, pulse 80/minute regular. Congestive failure persisted, however, in spite of prolonged use of digoxin, chlorothiazide and later mersalyl. Eventually failure was abolished and after discharge the patient was quite well experiencing only slight dyspnoea on effort (Category II) at 2 years.

CASE NO. F. 39. N. S. Male, 55 years. Registration No. 167902.

Admitted 23.9.61, Died 29.9.61, (6 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	1	6	5	4	0	4	20

Admitted shocked with slight ankle oedema, B. P. 180/130 on admission falling within 3 hours to 140/80, pulse 130/minute falling to 100/minute within one hour. History of myocardial infarction in 1956; hypertension for years.

E.C.G. Posterior infarct; Q S wave changes in leads II, III, Avf. P-R interval 0.16 seconds.

TRANSAMINASES	Day	2	3
	S.G.O.T.	70	58
	S.G.P.T.	51	93

HAEMOGLOBIN 107% (Sahli)

PROGRESS Settled with morphia though blood pressure never rose above 145/80. Digoxin and chlorothiazide given and evidence of congestive failure absent by 3rd. day. Died suddenly on 6th. day after onset of ventricular fibrillation; cardiac massage and the administration of intracardiac caramine were of no avail.

POSTMORTEM (File No. 641071)

Pericardial sac contained 95 c.c. of straw coloured fluid and posterior aspect of heart was moderately injected. Heart weighed 520g. and left ventricle /

ventricle was markedly prominent, though the valves seemed normal. A yellowish transmural infarct 45 x 30mm. involved posterior wall. The coronary arteries were greatly narrowed by gross atheroma and no antemortem thrombus was seen.

L V WALL THICKNESS Normal wall 25mm. Infarct 22mm.

CASE NO. F. 40. M. S. Female, 55 years. Registration No. 152140

Admitted 9.1.62, Died 9.1.62, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	7	4	0	1	20

Admitted grossly shocked, B. P. 90/70, pulse 105/minute almost imperceptible. Slight ankle oedema and moderate neck vein over filling present. Previous myocardial infarcts in 1958 and 1959. A known diabetic.

E.C.G. Posterior infarct. T wave changes only seen in leads III, Avf. P-R interval 0.17 seconds on admission.

PROGRESS Morphia, digoxin, mersalyl given but congestive failure worsened rapidly and the B.P. fell to unrecordable levels. At the time of death adrenaline was given by intracardiac injection and external cardiac massage attempted without success.

CASE NO. F. 41. R. C. Female, 61 years. Registration No. 220162

Admitted 25.6.63, Died, 26.6.63, (2 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	3	7	0	4	4	20

Admitted grossly shocked, B. P. 160/100 falling to 60/? in 4 hours, pulse 120 - 130 / minute regular. No evidence of failure initially. History of angina and dyspnoea on effort for one year.

E.C.G. Anteroseptal infarct with Q S wave changes in leads I, Avr, V1 - V4. P-R interval 0.18 seconds on admission. 0.15 seconds later.

TRANSAMINASES	Day	1	2
	S.G.O.T.	14	175
	S.G.P.T.	15	80

PROGRESS Shock did not improve and congestive failure developed despite sedation with morphia and administration of digoxin and mersalyl, and death occurred early on the 2nd. day.

CASE NO. F. 42. M. O'N. Male, 68 years. Registration No. 140104

Admitted 20.3.61, Died 20.3.61, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	7	4	0	3	20

Admitted shocked, in coma, with moderate oedema of legs and sacrum, B.P. 80/?, pulse 100/ minute regular.

E.C.G. Posterior infarct with Q S wave changes in leads II, III, Avf. P-R interval 0.16 seconds.

PROGRESS Did not respond to the administration of digoxin, chlorothiazide or coramine, and died within 8 hours.

CASE NO. F. 43. J. D. Female, 62 years. Registration No. 171800.

Admitted 7.11.61, Discharged 12.12.61. (36 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	6	5	4	0	4	21

Admitted moderately shocked with slight ankle oedema, B.P. 180/90 falling soon to 110/70, pulse 125/minute regular. History of myocardial infarct in 1956 and hypertension recently.

E.C.G. Posterior infarct with Q S wave changes in leads II, III, Avf. P-R interval constant at 0.16 seconds. Sinus tachycardia present on admission with evidence of old anterior infarct.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	34	38	78
	S.G.P.T.	11	19	27

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	8	139	4.3	101	25.1

PROGRESS Sedated with morphia and on 2nd. day B. P. was 160/85. Sinus tachycardia settled during first day and congestive failure cleared rapidly with the administration of digoxin and mersalyl. After discharge was fairly well at first though dyspnoea on effort was troublesome at times (Category III). Sustained a further infarct on 31.3.62 and died on 2.4.62.

CASE NO. F. 44. J. F. Male, 64 years. Registration No. 180709

Admitted 17.11.63, Discharged 30.12.63, (43 Days)

	A. S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	4	0	4	21

Admitted moderately shocked, with slight ankle oedema and neck vein over filling, B.P. 85/70, pulse 95/minute regular. Past history of myocardial infarction in 1954, cerebral thrombosis in 1960.

E.C.G. Anteroseptal infarct; Q S wave changes in leads I, Avl, V1 - V4. P-R interval on admission 0.15 seconds, on 8th. day 0.17 seconds, on 28th. day 0.16 seconds.

TRANSAMINASES	Day	1	2	3	4	5
	S.G.O.T.	44	103	25	28	17
	S.G.P.T.	25	30	26	15	25

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	7	143	4.5	102	28.6
	21	141	4.7	99	27.1

HAEMOGLOBIN 84% (cyan method).

PROGRESS Sedated with morphia, B.P. 130/90 at end of first day. Despite administration of digoxin and mersalyl condition deteriorated during the next few days and peripheral oedema increased in degree. Thereafter there was a slow and gradual improvement and subsequent progress was uneventful. Thereafter was not well, and was re-admitted on 19.1.64 for 5 days following an anginal /

anginal attack. Was still alive in December, 1964, though severely disabled by dyspnoea on effort (Category IV).

CASE NO. F. 45. P. C. Male, 64 years. Registration No. 154017

Admitted 8.11.61, Discharged 8.12.61. (32 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	1	4	4	4	21

Admitted slightly shocked, and with basal crepitations and slight congestive failure, B. P. 130/90, pulse 130/minute regular. History of myocardial infarct in April, 1961.

E.C.G. Anteroseptal infarct and left bundle branch block, Q S wave changes in leads I, Avl. Vl - V4. P-R interval 0.16 seconds on admission, 0.20 seconds on 8th. and 28th. days. Sinus tachycardia present throughout first 3 days.

TRANSAMINASES	Day	1	2	5
	S.G.O.T.	110	195	53
	S.G.P.T.	25	70	10

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	8	142	4.3	103	24.1

HAEMOGLOBIN 100% (Sahli)

PROGRESS Sinus tachycardia persisted throughout the first 3 days and settled only after the administration of quinidine sulphate. Congestive failure improved following the therapy with chlorothiazide. Subsequent progress uneventful. After discharge was quite fit (Category II) for one year but thereafter dyspnoea on effort became more troublesome (Category III) though he was still alive 30 months after the infarct.

CASE NO. F.46. J. McR. Male, 63 years. Registration No. 193880

Admitted 17.8.62, Died 17. 8.62, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	1	4	4	4	21

Admitted with mild shock, gross neck vein over filling, and slight ankle oedema. B. P. 100/70, pulse 120-130/minute, regular.

E.C.G. Anteroseptal infarct, Q S wave changes in leads I, V1 - V3. P-R interval 0.22 seconds.

PROGRESS Did not respond to administration of morphia, chlorothiazide and digoxin, and died within 3 hours of admission.

CASE NO. F. 47. J. W. Male, 72 years. Registration No. 176514

Admitted 13.1.62, Died 16.1.62, (4 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	7	4	0	4	21

Admitted grossly shocked and in slight congestive failure, B.P. 95/50, pulse 95/minute regular. History of dyspnoea on effort for several years.

E.C.G. Posterior infarct, Q S wave changes in leads II, Avf. P-R interval 0.20 seconds on admission, 0.18 seconds on 4th. day.

TRANSAMINASES	Day	1	2
	S.G.O.T.	40	92
	S.G.P.T.	155	170

HAEMOGLOBIN 94% (cyan method).

PROGRESS Responded initially to morphia, aminophyline, chlorothiazide and digoxin but never really recovered from shock and then gradually slipped into congestive failure and died.

CASE NO. F. 48. G. M. Male, 63 years. Registration No. 189863

Admitted 19.6.62, Died 23.6.62 (4 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	4	0	4	21

Admitted shocked and with oedema of the legs and sacrum, B.P. 115/75, pulse 88/minute regular. History of myocardial infarct in 1960.

E.C.G. Anteroseptal infarct, Q S wave changes in leads I, Avl, Vl - V5. P-R interval steady at 0.15 seconds.

TRANSAMINASES	Day	1	2	4
	S.G.O.T.	38	82	90
	S.G.P.T.	23	54	75

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
	2	137.1	5.3	101	25.5
	6	139.2	4.9	99	26.6

HAEMOGLOBIN 92% (cyan method)

PROGRESS Recovered from shock after treatment with morphia, B. P. on second day 120/80, and general state improved at first with administration of digoxin, mersalyl and aminophyline. After the second day congestive failure became much more severe and death occurred late on the fourth day.

POSTMORTEM /

POSTMORTEM NO. 623035.

The pericardial sac contained a small volume of straw coloured fluid and was obliterated by fibrin adhesions over the markedly injected anterior wall. Heart weighed 400g. and left ventricle showed some hypertrophy though the valves were normal. There was a recent necrotic infarct involving anterior half of interventricular septum, apex and most of anterior wall. The coronaries showed gross atheroma, were very narrowed, but contained no antemortem thrombus.

L V WALL THICKNESS Normal wall 15mm. Infarct 14mm.

CASE NO. F. 49. J. C. Female, 73 years. Registration No. 175419

Admitted 30.12.61, Died 1.1.62, (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	4	0	4	21

Admitted with moderate shock, distended neck veins and slight ankle oedema. B. P. 80/64, pulse 100/minute regular. History of myocardial infarct in 1960.

E.C.G. Posterolateral infarct: Q S wave changes in leads II, III, Avf, V5. P-R interval 0.15 seconds.

TRANSAMINASES	Day	2	3
	S.G.O.T.	225	185
	S.G.P.T.	150	203

HAEMOGLOBIN 87% (Sahli)

PROGRESS Sedated with morphia and B. P. rose to 110/70 early on 2nd. day. Congestive failure improved following administration of digoxin and mercalyl and death on the 3rd. day was sudden and unexpected.

POSTMORTEM NO. 1/62.

Pericardial sac contained a normal volume of straw coloured fluid and the posterior aspect of heart was injected. Heart weighed 385g., the chambers were of normal size and the valves were all normal. A haemorrhagic necrotic infarct occupied much of posterior wall and small parts of lateral wall, but not interventricular /

interventricular septum. A small old densely fibrosed infarct was found in a more anterior part of lateral wall. The coronary arteries showed gross ulcerated atheroma and the lumen were greatly narrowed; no antemortem thrombus was seen.

L V WALL THICKNESS Normal wall 16mm. Infarct 14mm.

CASE NO. F. 50. W. S. Male, 60 years. Registration No. 182285

Admitted 12.4.63, Died 12.4.63, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	4	0	4	21

Admitted almost moribund, with marked congestive failure, B.P. 60/?, pulse 100/minute regular. History of myocardial infarct many years before.

PROGRESS Improved initially after the administration of morphia and digoxin but within 4 hours B. P. fell to 20/? and death occurred soon after.

POSTMORTEM NO. 104/63.

Pericardium and pericardial sac appeared normal. Heart weighed 385g., the chambers appeared of normal size and the valves appeared normal. There was an old densely fibrosed infarct in posterior wall with a recent haemorrhagic extension into interventricular septum. The coronary arteries showed gross atheroma and a large softened plaque occluded the right artery 11mm. from its origin.

L. V. WALL THICKNESS Not relevant; no recent infarct in L. V. wall.

CASE NO. F. 51. J. F. Male, 70 years. Registration No. 151778

Admitted 3.2.61, Discharged, 11.3.61, (36 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	5	4	0	4	22

Admitted severely shocked, with pulmonary oedema, marked leg oedema and slight sacral oedema, B.P. 80/50, pulse 100/minute regular. History of myocardial infarct several years before.

E.C.G. Anteroseptal infarct with Q S wave changes in leads I, Avl, V1-V5. P-R interval 0.17 seconds on all occasions. Old posterior infarct noted.

PROGRESS Responded well to morphia and aminophyline, B.P. 115/70 at end of first day, 130/80 on 4th. day. Peripheral oedema improved with chlorothiazide and absent from 6th. day. Subsequent progress uneventful. After discharge well at first (Category II) but after 15 months dyspnoea recurred and slight congestive failure was present at times (Category III) though the patient was still alive at 24 months.

CASE NO. F. 52. A. M. Male, 71 years. Registration No. 179452.

Admitted 24.6.62, Died 29.6.62. (6 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX:	3	6	1	4	4	4	22

Admitted with slight shock, neck vein over filling and minimal ankle oedema, B. P. 120/70, pulse 140/minute regular. History of myocardial infarction in 1949.

E.C.G. Anteroseptal infarct, Q S wave changes in leads I, Avl, Vl - V6. P-R interval 0.16 seconds throughout admission. Sinus tachycardia throughout first day.

TRANSAMINASES	Day	1	2
	S.G.O.T.	170	146
	S.G.P.T.	29	48

HAEMOGLOBIN 89% (cyan method).

PROGRESS Despite the administration of digoxin and mersalyl intractable congestive failure developed and death occurred on the 6th. day.

POSTMORTEM NO. 129/62.

The pericardial sac contained slightly increased amounts of straw-coloured fluid and a mild fibrin exudate was present over the anterior wall. Heart weighed 550g. left ventricle was moderately hypertrophied but the valves were normal. A recent rather soft yellowish infarct involved the anterior ⅓ of the interventricular septum, apex and adjacent ⅓ of the anterior wall.

AN /

An old well fibrosed infarct, 20mm. in diameter, was found in the posterior wall. The coronary arteries showed moderate atherome and an antemortem thrombus occluded the descending branch of the left artery 5mm. from its origin.

L.V. WALL THICKNESS Normal wall 21mm. Infarct 18mm.

CASE NO. F. 53. D. M., Male, 64 years. Registration No. 218189.

Admitted 25.6.63. Died 27.6.63. (2 Days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	2	1	7	4	4	4	22

Admitted gravely shocked with distended neck veins and slight ankle oedema, B.P. 60/30, pulse 40/minute regular.

E.C.G. Posterior infarct with complete heart block and atrioventricular dissociation.

TRANSAMINASES	Day	1	2
	S.G.O.T.	35	155
	S.G.P.T.	30	172

PROGRESS. Never recovered fully from the shock despite sedation with morphia though the B.P. did rise for a short time on the 2nd. day to 100/60 before falling away to 60/?. The heart block showed no improvement and the congestive failure became rapidly more severe despite the use of digoxin and chlorothiazide. At the time of death cardiac massage was attempted without success.

POSTMORTEM NO. 633511.

The pericardial sac contained a normal volume of straw-coloured fluid and the posterior aspect of heart was moderately injected. Heart weighed 500g., left ventricle was moderately injected and the valves appeared normal. There was a recent haemorrhagic infarct involving posterior half of inter-ventricular /

interventricular septum, the entire posterior wall and parts of lateral wall. The coronary arteries showed gross atheroma and the right was occluded by an antemortem thrombus at its origin.

L. V. WALL THICKNESS Normal wall 18mm. Infarct 14mm.

CASE NO. F. 54. A. M. Male, 74 years. Registration No. 180727

Admitted 2.3.62, Died 2.3.62. (1 Day).

	A. S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	6	5	4	0	4	22

Admitted moderately shocked, with distended neck veins but no peripheral oedema. B. P. 114/74, pulse 100/minute regular. History of myocardial infarct in 1942.

E.C.G. Anterior infarct, Q S wave changes in leads I, Avl. V1 - V4. P-R interval 0.2 seconds.

PROGRESS Did not respond to morphia or digoxin. Steroids were considered as a life saving measure but death occurred before this could be done.

CASE NO. F. 55. J. C. Male, 73 years. Registration No. 161601

Admitted 30.1.62, Died 2.2.62, (3 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	3	4	0	4	22

Admitted shocked and with distended neck veins though no obvious peripheral oedema present. B. P. 100/65, pulse 100/minute regular. Past history of myocardial infarct in 1955, and Paget's disease of pelvis in 1960.

E.C.G. Anterior infarct, Q S wave changes in leads I, V4 - V6.
P-R interval 0.18 seconds.

TRANSAMINASES	Day	2
	S.G.O.T.	277
	S.G.P.T.	42

HAEMOGLOBIN 93% (Sahli)

PROGRESS Settled well with morphia and B.P. rose to 115/20 by end of first day. Thereafter progressive congestive failure developed, digoxin and mersalyl had little obvious effect and death occurred late on the 3rd. day.

POSTMORTEM NO. 34/62.

Pericardial sac contained only a small volume of clear fluid and was obliterated anteriorly by a fibrin exudate. Heart weighed 475g., the chambers were fairly normal in size, and the valves were normal. There was a recent yellowish infarct occupying much of the anterior wall and an old fibrosed /

fibrosed infarot involved the remainder of anterior wall, apex and a small part of interventricular septum. The coronary arteries were grossly narrowed by atheroma but there was no antemortem thrombus.

L. V. WALL THICKNESS Normal wall 17mm. Infarot 15mm.

CASE NO. F. 56. W. D., Male, 72 years. Registration No. 150942

Admitted 9.2.63, Discharged 4.4.63, (54 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	4	4	23

Admitted shocked with marked oedema of legs and sacrum, B.P. 100/70, pulse 95/minute irregular. History of dyspnoea on effort for several years.

E.C.G. Anteroseptal infarct, Q S wave changes in leads I, Avl., V1 - V5. P-R interval steady at 0.20 seconds. Ventricular extrasystoles for first 4 days.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	53	28	35
	S.G.P.T.	20	8	13

HAEMOGLOBIN 90% (Cyan method).

PROGRESS Sedated with morphia and B. P. soon rose to 128/80. Ventricular extrasystoles were troublesome at first but were controlled after first 4 days by procaine amide. Despite prolonged mersalyl therapy severe peripheral oedema persisted for the first month but then slowly cleared. After discharge remained reasonably well though troubles by dyspnoea on moderate effort (Category III) and was still alive 21 months after the infarct.

CASE NO. F. 57. C.P. Female, 69 years. Registration No. 79158.

Admitted 14.4.61, Died 13.5.61, (29 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	7	4	0	3	23

Admitted gravely shocked with slight congestive failure, B.P. 90/55, pulse 90/minute regular. Past history of myocardial infarct in 1958.

E.C.G. Lateral infarct with Q R wave changes in leads II, Avf, V5 - V6. P-R interval on 1st. 8th. and 28th. days 0.18, 0.16, 0.17 seconds respectively.

TRANSAMINASES	Day	5
	S.G.O.T.	31
	S.G.P.T.	38

HAEMOGLOBIN 97% (Sahli)

PROGRESS Shock was severe and prolonged despite administration of morphia and aminophyline, and persisted until the 3rd. day. B. P. rose to around 100-125/65-75 by end of first week and never rose to above these levels. Congestive failure improved at first with digoxin, mersalyl, and later chlorothiazide, but was never completely absent and became increasingly more severe during the last week of life.

CASE NO. F. 58. D.N. Male, 60 years. Registration No. 152241

Admitted 29.4.62, Died 29.4.62, (1 Day).

Admitted in gross shock, B.P. 76/30, pulse 88/minute, triple rhythm; gross oedema of legs and sacrum and marked neck vein over filling present. History of previous myocardial infarcts in 1959 and 1961.

E.C.G. Posterior infarct, Q S wave changes in leads III, Avf. P-R interval 0.19 seconds.

PROGRESS: Morphia, digoxin and mersalyl had no effect and the patient died within 6 hours.

CASE NO. F. 59. W. C. Male, 75 years. Registration No. 196479.

Admitted 26.10.63, Died 2.11.63, (8 Days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	4	4	23

Admitted shocked and in congestive failure, B. P. 160/100 falling soon to 145/84, pulse 110/minute irregular. History of hypertension and dyspnoea on effort for 3 years with angina for one year.

E.C.G. Lateral infarct with Q S wave changes in leads II, Avf, V5 - V6. P-R interval 0.15 seconds throughout admission. Ventricular extrasystoles present throughout first 2 days.

TRANSAMINASES	Day	1	2	3
	S.G.O.T.	57	156	129
	S.G.P.T.	20	39	62

PROGRESS Did well at first after sedation with omnopon and administration of digoxin and mersalyl. On the 8th. day died suddenly following a cerebral embolus.

POSTMORTEM NO. 213/63.

Pericardial sac was normal but the posterior aspect of heart was dark red in colour. Heart weighed 525g., left ventricle was moderately prominent and the valves were normal. There was a recent necrotic yellowish infarct measuring 52 x 30 mm. lying mainly in posterior and partially in lateral wall. The /

The coronary arteries were extremely narrow due to many ulcerated and sometimes calcified athoromatous plaques.

L. V. WALL THICKNESS Normal wall 19mm. Infarct 12mm.

CASE NO. F. 60. T. R. Male, 76 years. Registration No. 210864.

Admitted 15.3.63, Died 15.3.63, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	7	4	0	3	23

Admitted grossly shocked with marked oedema of the legs, B. P. 50/?, pulse 100/minute regular. History of myocardial infarct in 1953.

E.C.G. Posterolateral infarct: Q R wave changes in leads II, III, Avf, V.5. - V6. P-R interval 0.18 seconds.

TRANSAMINASES	Day	1
	S.G.O.T.	250
	S.G.P.T.	64

ELECTROLYTES	Day	Sodium	Potassium	Chloride	CO_2
		139	5.1	106	14.0

PROGRESS Showed no improvement after administration of digoxin and mersalyl. As a possible life saving measure it was decided that hydrocortisone be given but death occurred before this was possible.

CASE NO. F. 61. A. W., Male, 75 years. Registration No. 160010

Admitted 27.10.61, Died 29.10.61, (3 Days).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	3	5	4	4	4	23

Admitted very shocked, with marked oedema of the legs and sacrum. B.P. 40/?, pulse 95/minute irregular at times. History of dyspnoea on effort for 4 years.

E.C.G. Anteroseptal infarct and left bundle branch block, with Q S wave changes in leads I, Avl., V1 - V5. P-R interval 0.24 seconds with occasional dropped beats on the first day.

PROGRESS Responded initially to morphia and mersalyl, B.P. rising to 70/20 by end of first day but thereafter his condition deteriorated steadily until the time of death.

POSTMORTEM NO. 613476.

The pericardial sac contained a normal volume of straw-coloured fluid and there was a fibrin exudate over anterior wall. Heart weighed 465g., left ventricle was slightly hypertrophied and the valves were normal. A recent infarct involved anterior $\frac{1}{3}$ of interventricular septum, apex and most of anterior wall. The coronary arteries showed moderate atheroma and an antemortem thrombus occluded the left artery 5mm. from its origin.

L. V. WALL THICKNESS Normal wall 19mm. Infarct 18mm.

CASE NO. F. 62, E. B., Male, 70 years. Registration No. 229840.

Admitted 11.11.63, Died 11.11.63, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	7	4	0	4	24

Admitted grossly shocked, with marked neck vein over filling and slight oedema of the legs, B.P. 90/?, pulse 112/minute regular. Previous infarct in 1949.

PROGRESS Showed no response to treatment with morphia, digoxin, aminophyline or mersalyl and died before any investigations could be performed.

CASE NO. F. 63. F. R., Female, 60 years. Registration No. 176113.

Admitted 18.4.62, Discharged 27.5.62, (41 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	4	4	4	25

Admitted shocked with distended neck veins but no peripheral oedema, B.P. 90/60, pulse 100/minute, irregular at times.

E.C.G. Posterior infarct; Q S wave changes in leads II, III, Avf., P-R interval 0.26 seconds. Nodal rhythm present during first day.

TRANSAMINASES	Day	2	4
	S.G.O.T.	258	95
	S.G.P.T.	64	89

HAEMOGLOBIN 102% (cyan method).

PROGRESS Reponded well to morphia and procaine amide, B. P. 130/80 early on 2nd. day, and rhythm normal late on first day. Chlorothiazide was given during the first week, and subsequent progress was uneventful. After discharge was well at first having only slight dyspnoea (Category II) but after 21 months dyspnoea became more severe (Category III) though the patient was still alive at 33 months.

CASE NO. F. 64. W. McC. Male, 61 years. Registration No. 138445.

Admitted 8.3.61, Died 12.4.61, (36 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	4	4	4	25

Admitted severely shocked and with moderate oedema of the legs.
B. P. 125/80, pulse 100/140/minute, regular. History of myocardial infarct in December, 1960.

E.C.G. Posterolateral extension of old posterior infarct, with Q S wave changes in leads II, III, Avf, V5 - V6. P-R interval on 1st., 8th. and 28th. days, 0.18, 0.18, and 0.18 seconds respectively.

TRANSAMINASES	Day	1	3
	S.G.O.T.	101	107
	S.G.P.T.	11	74

HAEMOGLOBIN 91% (Sahli)

PROGRESS Responded to morphia, digoxin, mersalyl and later chlorothiazide. Although congestive failure improved at first, B. P. never rose above 130/80 and fell during the last week to 70 - 85 / 50 - 60. Congestive failure became more severe during the 3rd. and 4th. weeks and a terminal hypostatic pneumonia developed.

CASE NO. F. 65. J. M., Male, 68 years. Registration No. 169878.

Admitted 14.10.61, Died 15.1061, (2 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL.
PROGNOSTIC INDEX	3	3	7	4	4	4	25

Admitted grossly shocked, with slight ankle oedema and distended neck veins, B. P. 70/?, pulse 130/minute regular. History of intermittent claudication for 3 years.

E.C.G. Anteroseptal infarct with left bundle branch block; Q S wave changes in leads I, Avl., V2 - V6. P-R interval 0.2 seconds. Sinus tachycardia present throughout admission.

PROGRESS Did not respond to morphia, digoxin or mersalyl. B.P. soon fell to 50/? and congestive failure steadily worsened. At the time of death cardiac massage was attempted with no lasting result.

POSTMORTEM (File No. 644265).

Pericardium and pericardial sac were normal. Heart weighed 435g., the chambers were of normal size and the valves were normal. A rather ill-defined pale infarct involved half of posterior wall and a small portion of interventricular septum. An old fibrosed infarct, 20mm. in diameter, involved anterior wall, apex and a little of the anterior end of interventricular septum. In addition to gross atheroma in both coronary arteries, the right was occluded by an antemortem thrombus near its origin. /

L.V. WALL THICKNESS Normal wall 17mm. Infarct 17mm.

CASE NO. F. 66 J. McL. Female, 54 years. Registration No. 132689.

Admitted 23.2.61, Died 3.3.61, (8 Days)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	2	6	5	4	4	4	25

Admitted gravely shocked, pulse 98/45, pulse 130/minute, with slight ankle oedema. Previous anteroseptal infarct in 1960.

E.C.G. Posterior infarct: Q S wave changes in leads II, III, Avf., P-R interval 0.16 seconds on all occasions. Sinus tachycardia on first 2 days.

TRANSAMINASES	Day	2	3
	S.G.O.T.	210	148
	S.G.P.T.	17	50

HAEMOGLOBIN 97% (Sahli).

PROGRESS Improved at first following administration of morphia, digoxin, and chlorothiazide. B.P. 110/70, at end of first day, 125/70 on 4th. day. Late on 2.3.61 again collapsed and died within a few hours.

CASE NO. F. 67. A. M., Male, 41 years. Registration No. 151683.

Admitted 21.3.61, Died 21.3.61, (1 Day).

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	0	6	7	4	4	4	25

Admitted moribund, B. P. 50/?, pulse 34/minute, with marked oedema of legs. Past history of myocardial infarcts in 1959, 1961, 1962.

E.C.G. ? Posterior infarct. Complete heart block with atrioventricular dissociation.

PROGRESS Died almost at once. Aminophyline, digoxin, mersalyl and pethidine had no effect.

CASE NO. F. 68. A. N. Male, 68 years. Registration No. 152992

Admitted 3. 11. 63. Died 3. 11. 64. (1 Day)

PROGNOSTIC INDEX	A. S.	P. H.	S.	F.	R.	E.	TOTAL
	3	6	7	4	4	4	28

Admitted severely shocked. B. P. unrecordable. Pulse at first 95/minute then 38/minute regular. Marked oedema of legs and sacrum present. Previous myocardial infarcts in 1950 and 1961.

E. C. G. Posterior infarct. QS wave changes in leads II, III, Avf. At first sinus rhythm, P-R interval 0.18 seconds with runs of ventricular extrasystoles, then half an hour after admission, complete heart block.

PROGRESS. The patient appeared to die 2 hours after admission, morphia, digoxin, mersalyl and aminophyline having had no effect. External and then internal cardiac massage were attempted without success.

POSTMORTEM NO. 633945

Pericardium and pericardial sac normal. Heart weighed 435 G. and left ventricle was slightly hypertrophied. Valves normal in appearance. There was a recent, ill defined, haemorrhagic infarct in posterior wall involving a small area of adjacent interventricular septum, and measuring 32 x 21 mm. overall. An old small well fibrosed anteroseptal infarct was present. The coronary arteries were narrowed by gross atheroma but no antemortem thrombus was present.

L.V. Wall Thickness. Normal wall 17 mm. Infarct 17 mm.

CASE NO. F. 69. L. G. Male, 65 years. Registration No. 150829

Admitted, 1.4.62, Died 1.4.62, (1 Day)

	A.S.	P.H.	S.	F.	R.	E.	TOTAL
PROGNOSTIC INDEX	3	6	7	4	4	4	28

Admitted moribund, B. P. unrecordable, pulse 130/minute irregular and very weak with marked pulmonary and slight leg oedema. History of myocardial infarcts in 1960 and 1961, the latter being included in this survey as Case No. F. 14.

PROGRESS Died before laboratory investigations could be undertaken. No response to pethidine, digoxin, mersalyl or aminophyline.

CASE NO. G. I. J. M. Male, 66 years. Registration No. 204409

Admitted 22.12.62. Discharged 26. 1. 63. (35 Days).

PROGNOSTIC INDEX	A. S.	P. H.	S.	F.	R.	E.	TOTAL
	3	3	5	4	0	4	19

Admitted shocked with slight ankle oedema. B.P. 105/70, pulse 95/minute regular. History of intermittent claudication and of angina for several years.

E. C. G. Anteroseptal infarct with QS wave changes in leads I, V1 - V4. P.R. interval 0.18 seconds on admission.

TRANSAMINASES	day	1	2	3	4
	S.G.O.T.	77	39	29	35
	S.G.P.T.	22	76	36	26

PROGRESS Responded to morphia, and chlorothiazide and at first seemed quite well. On the 6th day collapsed with ? ventricular fibrillation, which was converted to auricular fibrillation after 3 shocks from a defibrillator. Hydrocortisone was given at this time as a life saving measure and subsequent progress was uneventful. Patient alive at 18 months.

CASE NO. G. 2. E. B. Female, 73 years. Registration No. 201250

Admitted 16. 11. 52. Died 29. 11. 62. (13 Days).

PROGNOSTIC INDEX	A. S.	P. H.	S.	F.	R.	E.	TOTAL
	3	6	1	1	4	4	19

Admitted slightly shocked, with basal crepitations but no peripheral oedema. B. P. 120/75, pulse 90/minute irregular. History of myocardial infarct in 1954.

E. C. G. Anterior infarct, QS wave changes in leads I, Avl, V3 - V6. P. R. interval on 1st and 8th days 0.18 and 0.17 seconds. Ventricular extrasystoles on admission.

TRANSAMINASES	day	1	2	3
	S.G.O.T.	240	365	170
	S.G.P.T.	52	67	175

PROGRESS Responded initially to morpia, aminophyline, digoxin and mersalyl. On 4th day collapsed and was then started on a hydrocortisone regime similar to that given in Series A. Responded initially, B.P. 135/80 on 6th day, but collapsed again on 13th day and died almost at once.

CASE NO. G. 3 J. M. Female, 63 years. Registration No. 173824.

Admitted 2. 12. 61. Died 9. 12. 61. (8 Days).

PROGNOSTIC INDEX	A. S.	P. H.	S.	F.	R.	E.	TOTAL
	2	6	0	4	4	4	20

Admitted with distended neck veins, slight peripheral oedema, but not in shock. B.P. 140/95, pulse 105/minute irregularly irregular. History of myocardial infarct in 1959, and of hypertension for several years.

E.C.G. Anteroseptal infarct, with QS waves in leads I, Avl, Vl - V4. P. R. interval 0.19 seconds.

TRANSAMINASES	day	1	3
	S.G.O.T.	160	315
	S.G.P.T.	60	335

PROGRESS Despite digoxin and mersalyl, went into congestive failure. On 4th day collapsed and received hydrocortisone 100 mg. i.v. Improved a little that day but thereafter failure became progressively worse until the time of death.

CASE NO. G. 4. A. M. Male, 46 years. Registration No. 167560

Admitted 19. 9. 61. Discharged 20. 10. 61. (31 Days).

PROGNOSTIC INDEX	A. S.	P. H.	S.	F.	R.	E.	TOTAL
	0	3	5	4	4	4	20

Admitted shocked, in congestive failure. B.P. 128/74, pulse 100/minute irregular. History of dyspnoea on effort for some years.

E.C.G. Anteroseptal infarct with QS wave changes in leads I, Avl, V1 - V4. Auricular fibrillation present on admission.

TRANSAMINASES day 2

S.G.O.T. 73

S.G.P.T. 40

PROGRESS Improved at first with morphia, digoxin, and mersalyl but at the end of the 2nd day, collapsed, and received a total of 500 mg. hydrocortisone i.v. Improved until 23. 9. 61. when there was an outburst of ventricular tachycardia, controlled speedily with procainamide. Subsequent progress uneventful but died 2 years later following a further infarct.

CASE NO. G. 5. C. H. Male, 41 years. Registration No. 203683

Admitted 11. 12. 62. Discharged 19. 1. 63. (38 Days).

PROGNOSTIC INDEX	A. S.	P. H.	S.	F.	R.	E.	TOTAL
	0	3	7	4	4	4	22

Admitted gravely shocked. B. P. and pulse unrecordable, with marked oedema of the legs. History of hypertension recently.

E. C. G. Anteroseptal infarct. QS wave changes in leads I, Avl, Vl - V 6. P.R. interval 0.19 seconds.

TRANSAMINASES	day	1
	S.G.O.T.	24
	S.G.P.T.	10

PROGRESS Did not recover from shock despite sedation with morphia and early on 3rd day collapsed. E.C.G. showed ventricular fibrillation and therapeutic measures included internal cardiac massage, the use of a defibrillator, and the administration of both procaine amide and hydrocortisone. Thereafter progress was uneventful and the patient was alive 2 years later.

CASE NO. G. 6. S. D. Male, 74 years. Registration No. 151650

Admitted 24. 11. 62. Discharged 5. 1. 63. (42 Days).

PROGNOSTIC INDEX	A. S.	P. H.	S.	F.	R.	E.	TOTAL
	3	3	7	4	4	4	25

Admitted gravely shocked. B.P. 80/50, pulse 85/minute irregular, with moderate peripheral oedema.

E.C.G. Posterolateral infarct. QS wave changes in leads II, III, Avf, V5 - V6. P. R. interval 0.24 seconds with a number of dropped beats.

TRANSAMINASES	day	1	2	3
	S.G.O.T.	45	52	170
	S.G.P.T.	27	23	30

PROGRESS Did not recover from shock till late on 2nd day but initial recovery was unremarkable. Collapsed early on 4th day and received hydrocortisone (as in Series A) with temporary improvement. On 12th day, complete heart block supervened, and despite hydrocortisone therapy sinus rythm was not restored for a further 5 days. Thereafter recovery was without incident.

CASE NO. H. 1 H. B. Male, 56 years Registration No. 139000

Admitted 18. 1. 61. Died 19. 1. 61. (1 Day)

Admitted collapsed with slight ankle oedema. B. P. 120/70, pulse 90/minute regular. History of dyspnoea and angina on effort for 3 years, and of recent melaena.

E. C. G. Anterior infarct with QS wave changes in leads I, Avl, V4 - V6. P. R. interval 0.18 seconds.

PROGRESS Never recovered fully from initial shock despite sedation with morphia. 20 hours after admission B.P. began to fall to imperceptible levels and death occurred soon after.

POSTMORTEM NO. 13/61.

Pericardium and pericardial sac were normal. Heart weighed 400 G., left ventricle was a little hypertrophied, but the valves were normal. A rather ill defined pale infarct involved apex and much of anterior wall, but not interventricular septum. The first 15 mm. of both coronary arteries were virtually occluded by large soft ulcerated atheromatous plaques but there were no antemortem thrombi.

L.V. Wall Thickness. Normal wall 17 mm. Infarct 17 mm.

A simple ulcer was present in first part of duodenum and this showed evidence of recent haemorrhage, altered blood being present in small, but not large bowel.

CASE NO. H. 2. A.Y. Female, 77 years. Registration No. 197085

Admitted 1.10.62. Died 2. 10. 62. (1 Day)

CLINICAL SUMMARY.

Admitted collapsed, barely conscious. B.P. 160/120, pulse 120/minute regular, with marked oedema of the legs. Her condition deteriorated rapidly and there was no obvious response to morphia, aminophylline, digoxin or mersalyl.

POSTMORTEM No. 209/62.

Pericardial sac contained 80 cc. of clear straw coloured fluid, and anterior wall of heart appeared injected. Heart weighed 600 G., both ventricles were hypertrophied though the valves were normal. A recent haemorrhagic infarct involved apex, lower anterior quarter of interior wall. The coronary arteries show marked atheroma with some calcified areas and there was no antemortem thrombus.

L.V. Wall Thickness. Normal wall 20 mm. Infarct 20 mm.

CASE No. H. 3. M. H. Female, 59 years. Registration No. 208105

Admitted 19. 2. 63. Died 19. 2. 63. (1 Day)

Had been in surgical ward with probable ear infection when she developed a typical myocardial infarct and was transferred to a medical ward. Admitted there collapsed. B. P. unrecordable, pulse 130/minute, very weak and at times imperceptible. History of myocardial infarct 2 years before.

PROGRESS. Died almost at once.

POSTMORTEM No. 55/63.

Pericardium and pericardial sac normal. Heart weighed 420 G. left ventricle was hypertrophied, and the valves were normal. A rather ill-defined infarct was present in apex, and much of anterior wall. Adjacent areas of interventricular septum showed patchy fibrosis. The coronary arteries showed little atheroma, no antemortem thrombus was present, and the lumina seemed patent.

L.V. Wall Thickness. Normal wall 20 mm. Infarct 20 mm.

CASE NO. H. 4. L. 8. Male, 73 years. Registration No. 209199

Admitted 24. 2. 63. Died 25. 2. 63. (1 Day)

Admitted semicomatose, with moderate oedema of legs, B. P. 140/80, pulse 84/minute irregular. History of previous myocardial infarct 18 months before.

E. C.G. Anterior infarct with auricular fibrillation.

PROGRESS. Died within 15 hours.

POSTMORTEM No. 63/63

Pericardial sac contained a normal volume of straw coloured fluid but the lateral aspect of heart was injected. Heart weighed 550 G., left ventricle was moderately hypertrophied and valves were normal. A recent infarct involved anterior 1/3 of interventricular septum, apex, anterior wall, and part of lateral wall. Much of posterior wall was replaced by a densely fibrosed infarct. The coronary arteries showed marked atheroma with areas of ulceration and calcification but no ante-mortem thrombus was seen.

L. V. Wall Thickness. Normal wall 25 mm. New infarct 25 mm. Old infarct 15 mm.

CASE NO. H. 5. J. J. Female, 66 years. Registration No. 219356

Admitted 13. 6. 63. Died 14. 6. 63. (2 Days)

CLINICAL SUMMARY.

Admitted to surgical department with upper abdominal pain but later transferred to medical ward. Shocked and in slight congestive failure on admission. B.P. 110/70, pulse 120/minute regular. Began to recover from shock after receiving morphia, but then collapsed and died almost at once.

POSTMORTEM NO. 133/63. Pericardial sac contained 115 ml. of straw coloured fluid and a fibrin exudate was present over posterior wall. Heart weighed 475 G., left ventricle was very prominent, but the valves were normal. A recent haemorrhagic infarct occupied most of posterior wall and the posterior 1/3 of interventricular septum. The coronary arteries were greatly narrowed by atheroma but no antemortem thrombus was present.

L.V. Wall Thickness. Normal wall 24 mm. Infarct 23 mm.

CASE NO. H. 6. A. P. Male, 65 years. Registration No. 205203

Admitted 5. 1. 63. Died 8. 1. 63. (3 Days)

Admitted slightly shocked, with neck vein overfilling but no peripheral oedema, B.P. 100/60, pulse 125/minute, irregular at times.

E. C.G. Anteroseptal infarct. QS wave changes in leads I, Avl, V1 - V 6. P-R interval 0.17 seconds. Ventricular extrasystoles at times in first day.

PROGRESS. Despite administration of digoxin and mersalyl congestive failure gradually became more severe and the B. P. never rose above 115/80. On the 3rd day the patient collapsed and died despite prolonged external cardiac massage.

POSTMORTEM NO. 10/63.

Pericardial sac contained 1/3 pint of liquid blood, the anterolateral aspect of heart was injected and a slit-like rupture 15 mm. long was noted in anterior wall close to interventricular septum 40 mm. above apex. A massive necrotic infarct involved anterior half of interventricular septum, apex, and the greater part of both anterior and lateral walls of left ventricle. The coronary arteries showed considerable atheroma and the left was occluded at its origin by an antemortem thrombus.

L.V. Wall Thickness. Normal wall 18 mm. Infarct 0 mm.

CASE NO. H. 7. P.F. Male, 72 years, Registration No. 206530.
Admitted 10.2.63, Died 12.2.63, (3 Days).

On admission moderately shocked, with pulmonary oedema but no peripheral oedema, B.P. 110/70, pulse 100/minute regular. Possible myocardial infarct some months earlier.

E.C.G. Anterior infarct with Q.S. wave changes in leads I, Avl, V1-V6.

PROGRESS Improved steadily at first with pethidine and aminophyline, but late on 3rd day collapsed and died at once.

POST MORTEM NO. 51/63.

The pericardial sac was obliterated anteriorly by fine fibrin adhesions and the anterior wall of heart was haemorrhagic and soft. Heart weighed 500 g., left ventricle was greatly hypertrophied and the valves were normal. Virtually the entire myocardium of both ventricles was infarcted being soft and yellowish in appearance the only normal areas being part of posterior wall near apex and a small adjacent area of right ventricle. The coronary arteries were greatly narrowed by atheroma but no antemortem thrombus was present.

L.V. WALL THICKNESS Normal wall 24 mm, Infarct 22 mm.

CASE NO. H. 8. F.McC. Male, 65 years, Registration No. 88631.
Admitted 25.2.61, Died 28.2.61, (4 Days).

Had been in congestive failure for some time, and was slightly shocked, B.P. 140/80, pulse 95/minute regular.

E.C.G. Recent anterior infarct, Q.S. waves in leads I, V1-V5. P.R. interval 0.16 seconds.

PROGRESS Improved a little at first with morphia, digoxin and mersalyl but periphoral oedema remained severe. Collapsed and died on 4th day following a ? cerebral thrombosis.

POST MORTEM NO. 56/61.

Pericardial sac contained a normal volume of clear fluid but anterior wall was covered by a fibrin exadate. Heart weighed 610 g., left ventricle was markedly hypertrophied, aortic valve being slightly incompetent due to calcification of the cusps. A recent infarct involved apex and the lower part of anterior and lateral walls. The coronary arteries showed marked atheroma with calcification, were greatly narrowed, but not occluded.

L.V. Wall thickness Normal wall 24 mm, Infarct 22 mm.
Brain showed a recent softening of left cerebrum.

CASE NO. H. 9. A.O. Male, 70 years, Registration No. 205613.
Admitted 20.2.63, Died 24.2.63, (4 Days).

Admitted moderately shocked with moderate ankle oedema, B.P. 100/65, pulse 110/minute regular. History of previous infarct 4 years earlier.

E.C.G. Posterior infarct, Q.S. waves in leads II, III, Avf. P.R. interval 0.19 seconds.

PROGRESS Recovered from shock after receiving morphia but oedema became more severe despite administration of digoxin and chlorothiazide B.P. rose to 135/80 on 2nd day but on the 4th day fell again to 90/60 and death occurred soon after.

POST MORTEM NO. 60/63.

The pericardial sac was obliterated by a recent fibrin exudate and no fluid remained. Heart weighed 600 g., and left ventricle showed considerable hyperplasia though the valves were normal. A recent infarct involved entire posterior wall and a small part of interventricular septum. An old well fibrosed infarct, 20 mm in diameter lay in lateral wall. The coronary arteries showed gross atheoma and were greatly narrowed but no antemortem thrombus was present.

L.V. WALL THICKNESS Normal wall 24 mm, Infarct 24 mm.

CASE NO. H. 10. M.F. Female, 57 years, Registration No. 139671.
Admitted 20.2.61, Died 24.2.61, (5 Days).

Admitted collapsed and only partially conscious with a recent myocardial infarct and probable cerebral embolus; B.P. 170/110, pulse 115/minute regular. History of severe angina on effort for some years.

E.C.G. Posterolateral infarct with Q.S. wave changes in leads II, III, Avf, V5 - V6.

PROGRESS The patient was never well experiencing considerable chest pain throughout the first two days. The patient became fully conscious after the first few hours, but a right hemiplegia was present at all times. Later slight congestive failure developed, and on the 4th day the patient gradually became unconscious, death occurring on the next day.

POST MORTEM NO. 53/61.

Pericardial sac contained a normal volume of straw coloured fluid but posterior aspect of heart was dull red in colour. Heart weighed 385 g., left ventricle was slightly hypertrophied, though the valves were normal. An extensive haemorrhagic infarct involved most of posterior wall, small areas of lateral wall, but not interventricular septum. The coronary arteries showed gross atheroma and calcification and were greatly narrowed although no complete occlusion was present.

L.V. WALL THICKNESS Normal wall 20 mm, Infarct 18 mm.

Contd...

contd./

Brain showed an area of softening in left internal capsule and several small petechial haemorrhages were present in left cerebral hemisphere.

CASE NO. H. 11. R. McN. Male, 56 years. Registration No. 214100

Admitted 1. 5. 63. Died 6. 5. 63. (6 Days)

CLINICAL SUMMARY.

Admitted shocked, with neck vein overfilling. B.P. 100/70, pulse 100/minute regular. History of dyspnoea on effort for 1 year. E. C. G. showed a postero-lateral infarct. The patient was never well, the B. P. fell slowly to around 100/60, and congestive failure increased in degree despite administration of digoxin and chlorothiazide. A hypostatic pneumonia was present during the last 2 days of life.

POSTMORTEM NO. 109/63.

The pericardial surfaces were joined by a fibrin exudate over most of the surface of the heart. Heart weighed 440 G, the left ventricle was slightly hypertrophied, and the valves were normal. A massive infarct involved the entire posterior wall, much of lateral wall, together with parts of anterior wall and the posterior half of interventricular septum. The coronary arteries showed gross atheroma and were markedly narrowed throughout their length.

L. V. Wall Thickness. Normal 25 mm. Infarct 20 mm.

SERIES NO. H. 12. D. McD. Male, 56 years. Registration No. 158696

Admitted 17. 3. 62. Died 25. 3. 62. (8 Days)

Admitted shocked with slight peripheral oedema. B. P. 110/75, pulse 88/minute regular. Past history of hypertension for some years, and thyrotoxicosis treated with radioactive iodine in 1960.

E.C.G. Anteroseptal infarct with QS wave changes in leads I, V1 - V4.

PROGRESS. Never well; recovered from shock after 24 hours after sedation with omnopon but B. P. never rose above 115/65. Despite administration of digoxin and chlorothiazide, progressive congestive failure developed and sacral oedema was present at the time of death.

POSTMORTEM No. 72/62.

Pericardial sac contained 10 cc. of serous fluid, the anterior aspect of heart was haemorrhagic, appeared very soft and on the point of rupture. Heart weighed 675 G. and left ventricle was markedly hypertrophied, and the valves seemed normal. A yellowish necrotic infarct involved anterior half of interventricular septum, apex, and the greater part of anterior wall. Some parts of anterior wall were markedly thinned and two areas seemed on the point of rupture. The coronary arteries showed moderate atheroma and on antemortem thrombus occluded the left descending branch close to its origin.

L. V. Wall Thickness. Normal wall 24 mm. Infarct 2 mm.

CASE NO. H. 13. H. H. Female, 64 years. Registration No. 138711

Admitted 1. 1. 61. Died 10. 1. 61. (10 Days)

Admitted shocked, with slight ankle and leg oedema. B.P. 110/70, pulse 100/minute irregular. History of dyspnoea on effort for 3 years. Ventricular extrasystoles on 1st day.

E. C. G. Anteroseptal infarct with QS wave changes in leads I, Avl, Vl - V 5.

PROGRESS. Despite administration of digoxin and mersalyl, congestive failure developed gradually. On the 6th day auricular fibrillation was noted but was not controlled with quinidine and fibrillation continued until death on the 10th day.

POSTMORTEM No. 10/61.

The pericardial sac was obliterated by fine fibrin adhesions over anterior wall, which was markedly injected in appearance. Heart weighed 390 G., left ventricle was minimally hypertrophied and the valves appeared normal. A rather haemorrhagic yellowish infarct involved the lower anterior quarter of interventricular septum, apex, and lower half of anterior walls. The coronary arteries showed gross atheroma with areas of ulceration and calcification, and the lumina were greatly narrowed. No antemortem thrombus was present.

L. V. Wall Thickness. Normal wall 20 mm. Infarct 17 mm.

CASE NO. H. 14. W. S. Male, 67 years. Registration No. 89837

Admitted 12. 4. 61. Died 26. 4. 61. (14 Days)

Admitted slightly shocked with slight congestive failure. B. P. 140/70, pulse 80/minute regular. History of dyspnoea on effort for many years.

E. C. G. Anterolateral infarct; QS wave changes limited to leads I, V 4 - V6. P -R interval 0.15 seconds.

PROGRESS. Was fairly well at first but then slipped into congestive failure despite digoxin and chlorothiazide. A chest infection developed in the last days of life and failed to respond to tetracycline.

POSTMORTEM No. 88/61

Pericardial sac contained a normal volume of fluid but anterior wall of heart was injected and dull red in colour. Heart weighed 400 G., the chambers were of normal size and the valves were normal. There was a recent necrotic infarct involving anterior 1/3 of interventricular septum, apex, and most of anterior wall. The coronary arteries showed moderately severe atheroma and the descending branch of the left artery was occluded by an antemortem thrombus near its origin.

L.V. Wall Thickness. Normal wall 18 mm. Infarct 15 mm.

CASE NO. H. 15. J. H. Male, 62 years. Registration No. 139607

Admitted 6. 2. 61. Died 20. 2. 61. (15 Days)

Admitted moderately shocked, with slight ankle oedema. B. P. 105/85, pulse 96/minute regular. Previous infarct in 1957.

E. C. G. Posterior infarct, QS wave changes in leads 11 and 111. P-R interval 0.18 seconds.

PROGRESS. Sedated with morphia and responded well to chlorothiazide. On 3rd day B.P. 155/95 and oedema was absent. Subsequent progress was uneventful until the 15th day when he collapsed and died at once.

POSTMORTEM No. 48/61

The pericardial sac contained a slightly increased volume of straw coloured fluid and the posterior aspect of heart was dull red in colour, and showed slight aneurysmal bulging. Heart weighed 425 G., and left ventricle was markedly prominent though the valves were normal. A rather necrotic infarct occupied the entire posterior wall and an old fibrotic infarct was seen in adjacent parts of the interventricular septum. Friable antemortem thrombus was adherent to the inner aspect of the infarct and partially filled the cavity of left ventricle. The coronary arteries were narrowed by gross atheroma and an antemortem thrombus occluded the right vessel 10 mm. from its origin.

L.V. Wall Thickness. Normal wall 25 mm. Infarct 5 mm.

Brain showed a small softening in left cerebral hemisphere.

CASE NO. H. 16. G. G. Male, 69 years. Registration No. 157660

Infarct 1. 8. 61. Died 18. 8. 61. (18 Days)

Was in urology department for treatment of simple hyperplasia of prostate, but collapsed with severe chest pain, and was treated thereafter by Dr. Murray's unit. B. P. 105/70, pulse 125/minute regular.

E. C. G. Anteroseptal infarct. QS wave changes in leads I, Avl, Vl - V6. P R interval 0.16 seconds.

PROGRESS Never well. Recovered somewhat after sedation with omnopon. B. P. 130/80 on 2nd day. Despite administration of digoxin and chlorothiazide congestive failure was present intermittently but death on the 18th day was sudden and rather unexpected.

POSTMORTEM NO. 138/61

Pericardial sac contained 255 cc. of blood clot and external examination of heart showed an apical aneurysm 50 mm. in diameter showing a slit rupture 5 mm. long in the posterior aspect. Heart weighed 430 G., and left ventricle was slightly hypertrophied though the valves were normal. There was an extensive haemorrhagic necrotic infarct involving anterior 1/3 of interventricular septum, apex, and most of anterior wall. The apex showed marked thinning with aneurysm formation. The coronary arteries showed gross atheroma with areas of ulceration and marked resultant narrowing. No antemortem thrombus was present.

L. V. Wall Thickness. Normal wall 15 mm. Infarct 0 mm.

CASE NO. H 17. G. S. Male, 72 years. Registration No. 139780

Admitted 22. 2. 61. Died 12. 3. 61 (19 Days)

Not shocked on admission, but with marked oedema of legs and sacrum. Blood pressure 118/85, pulse 85/minute irregular. Dyspnoea on effort for 5 years.

E. C. G. Anteroseptal infarct, with QS wave changes in leads I, V1 - V5. P R interval 0.15 seconds. Ventricular extrasystoles present on admission.

PROGRESS. Despite treatment with digoxin, chlorothiazide and tetracycline, congestive failure worsened and a hypostatic pneumonia developed.

POSTMORTEM 65/61

Pericardial sac contained a normal volume of clear fluid but the surface of heart over apex was dull and roughened. Heart weighed 600 G., both ventricles were markedly hypertrophied, and valves were normal. Relatively recent whitish infarcts involved anterior half of interventricular septum and apex. The descending branch of left coronary artery, and the main trunk of the right artery were occluded by large softened atheromatous plaques.

L. V. Wall Thickness. Normal wall 25 mm. Infarct 20 mm.

CASE NO. H. 18 J.J. Female, 72 years, Registration No. 179001

Admitted 3.2.62, Died 23.2.62. (21 Days)

Admitted moderately shocked and in slight congestive failure, B.P. 105/77, pulse 105/minute. History of dyspnoea on effort for 5 years.

E.C.G. Posterior infarct. Q.S. wave changes in leads II, III, Avf. P.R. interval 0.18 seconds.

PROGRESS Responded well initially to treatment with pethidine, digoxin and mersalyl but during the 3rd week congestive failure returned and proved resistant to all treatment.

POST MORTEM NO. 47/62.

Pericardial sac contained a normal volume of straw coloured fluid, but posterior wall of heart was injected. Heart weighed 300 g., all chambers were of normal size and the valves were normal. A recent whitish infarct occupied most of posterior wall but did not involve interventricular septum. The coronary arteries showed severe ulcerated atheroma but no complete occlusion and no antemortem thrombus was seen.

L.V. WALL THICKNESS Normal wall 14 mm, Infarct 13 mm.

CASE NO. H. 19 A.C. Male, 51 years, Registration No. 206292.

Admitted 7.9.63, Died 4.10.63 (27 Days).

Admitted moderately shocked with moderate oedema of legs, B.P. 100/50, pulse 80/minute irregular. History of myocardial infarct 10 months earlier.

E.C.G. Recent anterior and old posterior infarcts. Q.S. wave changes in leads I, Avl, V4 - V6. Auricular fibrillation on admission.

PROGRESS Recovered from shock after sedation with morphia, and oedema responded to treatment with digoxin and chlorothiazide. Subsequent progress was uneventful until the 20th day when a left hemiplegia developed over a period of 2 - 3 hours. Death occurred a few hours later.

POST MORTEM NO. 193/63.

The greater part of pericardial sac was obliterated by old adhesions. Heart weighed 455 g., the left ventricle was slightly hypertrophied, and the valves were normal. A rather haemorrhagic infarct occupied part of anterior wall and most of lateral wall, and was continuous with a densely fibrosed infarct occupying most of the posterior wall. The coronary arteries showed considerable atheroma and softened plaques occluded both arteries near their origin. No antemortem thrombus was seen.

L.V. WALL THICKNESS normal wall 18 mm, New infarct 13 mm.

S E R I E S J

Histological material only (see Table 48)

S E R I E S K

Histological material only (see Table 49)

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