THE EARLY COMPLICATIONS OF CARDIAC INFARCTION

BY

H. E. S. PEARSON, M.D., M.R.C.P.

Physician, Whittington Hospital, London

If a person survives the sudden loss by ischaemic necrosis of part of the ventricular muscle there follows a period in which special dangers threaten each of the physical systems and the personality itself. These dangers arise from two principal sources; on the one hand, from the local effects of the lesion and the circulatory depression that follows; and, on the other, from the immobility that must be imposed by the physician, together with the hazards that may attend more active treatment.

The purpose of this article is to enumerate the complications that have arisen in this way in a large series of cases of cardiac infarction, to assess their influence on prognosis, and to discuss their prevention and treatment.

The 567 patients in this series (370 males, 197 females) have all been treated as in-patients at Whittington Hospital during a period of four years following February, 1949. They comprise 338 consecutive cases under my care and 229 patients treated in other departments of the hospital. They include cases in which cardiac infarction developed in the course of other disease. The exclusion of patients in whom domiciliary treatment was possible and the usual proportion of beds for such emergencies have made the series a selective one, with a relatively high average age (64.5 years) and a bias towards greater than average severity.

The gross mortality, including all patients reaching the receiving wards alive, was 212 (37.4%). Of this number, only 64 (11.3%) died without the supervision of any of the complications which form the subject of this article.

Outline of Treatment

The intravenous use of morphine was generally preferred, when this drug was required, in order to avoid the establishment of inactive morphine depots in the ischaemic tissues of the shocked patient. Drugs of the sympathicomimetic group were avoided as stimulants except in the gravest emergency, but, where a steadily falling blood pressure was accompanied by clinical deterioration, intravenous saline or plasma infusions as described by Sampson and Singer (1949) were given. Intra-arterial infusions and transfusions (Berman and Akman, 1952) were also used occasionally to sustain a more rapidly falling blood pressure.

Anticoagulant treatment was given during the period of recumbency in 406 cases. In the remaining 161 cases contraindications were present or the diagnosis was delayed or missed. The first 163 patients received dicoumarol preceded by heparin, 100 mg. six-hourly, by Olavson needle, for 48 hours. The remaining 243 cases received ethyl biscoumacetate, with heparin for 24 hours unless the infarct was more than two days old. Prothrombin times were estimated by Toohey's (1950) method and the results expressed as the ratio between the patient's prothrombin time and that of a normal control. The aim in the first 100 cases was to keep this prothrombin time ratio (P.T.R.) between the limits

of 1.5:1 and 2.5:1 during the period of treatment, but in the later cases it was considered safe (Pearson, 1952) to raise this therapeutic range to between 2:1 and 3:1.

Passive movements and graduated exercises were given from as early a date as the patient's condition allowed, and the treatment and convalescence otherwise followed the generally accepted course.

In the following paragraphs the rough division into anterior-wall and posterior-wall infarcts is made where localization is significant. Lateral-wall infarcts are included in the posterior group because it is considered, in spite of Bosco's (1943) view, that their arrhythmogenic characteristics are shared with those of infarcts of the right circumflex bed. Thus all infarcts described as anterior have followed occlusion of the anterior descending branch.

Ventricular Rupture

Fortunately, this accident was rare. It did not occur under the age of 61, and the average age of the 14 patients (2.5%) was 69.0 years. Women outnumbered men by nine to five, a similar sex distribution to that reported by Gans (1951). In eight cases the anterior wall of the left ventricle was involved, the posterior wall in five, and the septum in one.

The cases fall into two groups. In the first (11 cases) the blood had dissected a passage between the necrotic muscle planes through the thickness of the ventricular wall, reaching the epicardium in one or more linear splits. In 10 instances it was possible to date the infarct, this form of rupture occurring after intervals of 1, 2, 3, 5, 4, 6, 5, 5, 3, and 14 days respectively. Nine

TABLE I.—Instances of Vascular Occlusion, Haemorrhage, Gangrene, and Cardiac Rupture Tabulated According to Prothrombin Time Ratio (P.T.R.) on Day of Incidence. Therapeutic Range in Bold Type.

	Anticoagulant Treatment Given (406 Cases)			Anti-coagulant Treatment Withheld (161 Cases)	Totals
	P.T.R. Values				
	<2	2-3	>3		
Reinfarction or extension ..	8	11	1	15	35
Systemic venous thrombosis ..	4	3	0	25	32
Pulmonary embolism ..	4	9	1	32	46
" arterial thrombosis ..	1	0	0	4	5
Systemic arterial thrombosis or embolism:					
Aorta ..	0	0	0	4	4
Carotid ..	0	0	0	3	3
Cerebral ..	0	0	0	12	12
Retinal ..	0	0	0	1	1
Abdominal ..	1	1	0	7	9
Limbs ..	1	1	1	6	9
Total ..	19	25	3	109	156
Haemorrhage (sites):					
Nose ..	3	4	1	0	8
Lungs ..	1	0	0	3	4
Stomach or duodenum ..	2	0	1	1	4
(peptic ulcer) ..	0	0	0	3*	3
Faeces ..	4	3	1	4	12
(piles)j..	1	4	0	0	5
Urine ..	3	4	1	1	9
Joints ..	0	1	0	1	2
Pericardium ..	0	2	0	1	3
Myocardium ..	0	1	0	0	1
Subcutaneous tissues ..	0	0	1*	0	1
Eye (subconjunctival) ..	1	1	0	0	2
Multiple ..	0	1*	0	0	1
Total ..	15	21	5	14	55
Gangrene of extremities ..	0	1	0	8	9
Ventricular rupture ..	4	5	0	5	14

* One case of serious haemorrhage.

of the patients were receiving anticoagulant treatment (see Table I), and, as mentioned in the section on haemorrhage, it is possible that the interference with clotting had allowed a haematoma within the infarct to force its way into the ventricular cavity. The explosive effect of the luminal blood must obscure all traces of this preliminary dissection, and the doubt can be resolved only by careful study of comparative figures (*Lancet*, 1951). Those of the present series do not indicate an increased risk (cf. Gans, 1951; Friedman and White, 1944).

The other type of rupture (three cases) was a "blow-out" through a thinned or aneurysmal ventricular wall. In one of these cases it was impossible to date the infarct, but in the others 18 and 20 days were known to have elapsed between infarct and rupture. The aneurysmal wall in each case had failed to develop the remarkable tensile strength that ultimately may appear (Codounis, 1948; Moyer and Hiller, 1951), but the risk of this type of rupture is clearly less than that of the dissecting type occurring during the early necrotic stage of the infarct, as was noted in the large series of Krumbhaar and Crowell (1925). The early development of ventricular aneurysm without subsequent rupture was observed in seven cases of the series.

A search for common factors which might help to serve as a warning of this disaster showed nothing but the evidence of transmural necrosis in all the cases—that is, of gross electrocardiographic changes and a high leucocytosis, with a raised E.S.R. as a less reliable indication. Hypertension, or left ventricular hypertrophy at necropsy, was present in no more than seven of the cases, and a temporary rise in blood pressure could be held to account for only two of these ruptures, one following defaecation and another after an injection of aminophyllin. In one patient (H125) the systolic blood pressure had never risen above 90 mm. Hg. A usual post-mortem observation with bearing on the immediate severity of the local ischaemic damage was the presence of severe atheroma in the coronary arteries that had remained patent. This was found in 10 of the cases, and in two others the surviving vessels were unusually small (the records of the two remaining cases are incomplete on this point). It was rarely possible to anticipate this finding during life, but its importance seems even greater than that of hypertension, because transmural necrosis is the *sine qua non* of the commoner dissecting form of rupture and a similar failure of marginal irrigation may well account for the comparatively great area of the infarcts which "blow out" through their weakened centre.

A raised blood pressure was shown by Prinzmetal and his associates (1949) to reduce the extent of an experimental infarct and, paradoxically, to lessen the degree of bulging of the ventricular wall by improving the perfusion of the marginal areas, so hypertension may exert some beneficial effect to counterbalance its greater mechanical disruptive force. It cannot be assumed, however, that the conditions are the same in human subjects with arteriosclerotic vessels, and most published work on the subject stresses the danger of hypertension (Howell and Turnbull, 1950).

The curious preponderance of women among these cases of rupture continues a tendency that is apparent in the post-mortem records of the Central Histological Laboratories over the last nine years (Bryson, 1952, personal communication).

Preventive Treatment

The traditional treatment of the post-infarction state by carefully graduated rest has largely been dictated by the possibility of ventricular rupture, and there is no doubt that special precautions should be observed during the first week, although the danger of the "blow-out" type may persist for much longer. The elderly patient with deep topical Q waves and a leucocytosis is particularly at risk, and the risk is proportionately greater in the presence of hypertension.

The management of the patient's bowels is of particular importance in this type of case, and some form of paraffin emulsion is nearly always necessary. The experimental evidence (Benton *et al.*, 1950) in favour of the bedside commode as against the bed-pan has not altogether been borne out in practice. A light patient can conveniently and safely be lifted from the bed to a commode or wheeled sanitary chair, but the heavy person is likely to try to assist the nurses in their struggle. For these, and generally where there is special risk of rupture, the skilful use of the bed-pan has been preferred, with particular care that the patient's body is supported on pillows and not balanced on the pan. In occasional cases not even a bed-pan can be allowed in the first few days.

Digitalis has acquired a bad reputation as a possible precipitant of cardiac rupture (Gans, 1951), but there are more cogent reasons for caution in the use of this drug after recent infarction, and the measure of its danger is also the measure of its efficacy in arresting the vicious circle of falling blood pressure and marginal ischaemia.

Vascular Accidents

The original infarct has been followed by further vascular occlusion in 156 instances, occurring in 122 patients (21.5% of the total). The average age of this group (64.8 years) was not significantly over the general average. Death occurred in 67 (11.8%) cases, and in 38 (6.7%) it was considered that the complication was an important contributory cause of death.

The importance of this type of accident and the desirability of prophylaxis with anticoagulant drugs are questions which cannot yet be regarded as settled. It is probably true to say that the general climate of opinion has moved from the whole-hearted advocacy of Wright *et al.* (1948) and Gilchrist (1952) towards the *via media* of Papp and Shirley Smith (1951a) and Russek *et al.* (1952), but there has always been a strong body of opposition to the use of anticoagulants on the ground of their unnecessary danger to the patient. It is difficult, however, to avoid the conclusion that the very wide variation in the incidence of thrombo-embolism in different published series is connected with the equally wide variation in the methods of recruitment of cases and the availability of beds for them.

In offering the figures in Table I as a contribution to this problem, I wish to emphasize that this series is a selective one in which these complications are especially likely to occur, and also that the relative safety shown is the result of team-work and long practice on the part of the hospital staff.

The last column shows the occurrence of thrombosis, embolism, and haemorrhage among those 161 patients in whom anticoagulants had not been given up to the time of the accident because of contraindications or delayed diagnosis. The fact that in some cases the complication rather than the original infarct brought the patient into hospital, and the existence of the contraindications (liver and kidney disease, peptic ulcer, etc.) in others, makes the figures unsuitable for control purposes, but the relative freedom of the 406 patients treated with anticoagulants is striking.

Of the three columns of P.T.R. values, the middle one, in bold type, lists the same accidents occurring within the

selected P.T.R. range in which the treated patients were maintained as closely as possible during their period of recumbency (a slightly lower range was used in the first 100 patients). It therefore represents the greater part of the patient-time in 406 cases. The flanking columns show the instances that occurred during unintentional under- or over-dosage with anticoagulant drugs.

Maintenance within the chosen therapeutic range will be seen to give a measure of protection against systemic venous thrombosis, pulmonary embolism, and arterial occlusions. The figures for reinfarction and extension, the most important of all, are less satisfactory, probably because of the part played by atheromatous closure as against intravascular clotting. A statistical analysis is inappropriate for the reasons given above.

Haemorrhage

The incidence of haemorrhage is similarly listed according to the P.T.R. state at the time of the occurrence. The list includes all trivial bleedings, and the three cases in which the haemorrhage was sufficient to influence the prognosis are marked with asterisks. Bleeding occurred in 13 cases in which no anticoagulant had been given, and one of these was a serious haematemesis. In 15 other cases at a sub-therapeutic P.T.R. level the influence of anticoagulants is open to doubt.

The average age of the 32 patients who bled during anticoagulant treatment was 66.4 years, a little higher than the general figure, and the exclusion of trivial haemorrhage would raise this average considerably. Two cases in this group bled seriously: a woman aged 75 who developed a large haematoma of the abdominal wall, and a woman of 81 with infarction of two-thirds of the ventricular mass, who bled freely into the intestine through multiple minute erosions.

Unexplained blood in the sputum never occurred at P.T.R. levels of therapeutic range and above. It was regarded as resulting from small lung infarcts, and anticoagulant treatment was continued in such cases without ill effect.

Two sites of haemorrhage require special mention. The first is below an atheromatous plaque in a coronary vessel, resulting in occlusion of the lumen, as described by Boyd (1928) and Paterson (1936). Wartman (1950) thought that this accounted for 10% of all coronary occlusions; and Gerlis (personal communication, 1952), using special methods of examination, believed that the incidence might be higher. This possibility, with particular bearing on the prophylactic use of anticoagulants in threatened infarction, as suggested by Papp and Shirley Smith (1951b), clearly requires further investigation. It does not appear to have occurred as a complication in this series, although a number of the original infarcts were caused in this way.

The second is into the infarct itself, with possible dissection of the muscle layers by the haematoma, leading to ventricular rupture. One case in this series showed such a haematoma in an early stage. This risk has been discussed above. Bleeding from the epicardial surface of the infarct with formation of a haemorrhagic pericardial effusion occurred in three patients, two of whom had been treated with dicoumarol. In neither instance was the bleeding serious, but this possible hazard of anticoagulant treatment is emphasized by Goldstein and Wolff (1951), who report three cases, and a further case has been reported by M. Toohey (1952, personal communication).

In summary, among the cases composing this particular series the therapeutic risk of this form of treatment was low, and has diminished with increasing experience. The treatment gave a considerable degree of protection against a type of complication of frequent occurrence and with an important effect on mortality.

Gangrene

Gangrene of the extremities is mentioned by Fishberg (1940) as a rare complication of cardiac infarction; and Swan and Henderson (1951), describing two cases, attributed

its occurrence to intense and prolonged vascular spasm. This mechanism appeared to account for lower-extremity gangrene in five of the nine cases that occurred in the present series, but in the others there was complete occlusion of a main artery by thrombosis in two instances and by embolism in two others.

Only one of the nine patients was receiving anticoagulant treatment (see Table I), and it is reasonable to infer that this form of treatment will generally protect the patient from this rare but serious complication.

In none of the cases could any question of amputation be entertained. No occluded vessels have been explored surgically, and, in retrospect, it does not appear that anything would have been gained by so doing, as diffuse lesions were present in all.

Arrhythmias

Disturbances of rhythm have been unusually frequent (cf. Tatibouet and Mathivat, 1950), with an important effect on the mortality (Table II). Whereas with vascular accidents

TABLE II.—Incidence and Mortality of Arrhythmia in 567 Cases of Cardiac Infarction

	No. of Instances	Percentage of Total Cases	Deaths	Group Mortality %
Auricular fibrillation	61	10.8	26	42.7
" " flutter	7	1.2	4	57.2
Paroxysmal tachycardia { Supraventricular	6	1.1	2	—
Ventricular	7	1.2	4	—
Unknown origin	3	0.5	2	—
Total	16	2.8	8	50.0
Heart-block { Partial	9	1.6	6	—
Complete	11	1.9	5	—
Total	20	3.5	11	55.0
Multiple ectopic beats	78	13.7	33	41.3
Sinus disturbances	12	2.1	6	50.0
Unknown nature	8	1.4	4	—
Total	202	35.6	92	—

the emphasis is on prophylaxis, here it is more a question of the correct management of the complication as it appears, and the readiness with which post-infarction arrhythmias can be influenced by drugs imposes special responsibilities. Of the two main sources of difficulty and danger, the first lies in the fact that the cellular pathology underlying the arrhythmia may be constantly changing either in the direction of necrosis and scarring or towards recovery. The disturbance is therefore likely to be fugitive, and one form may give place spontaneously to another or to a succession of changes, making the reaction to a given drug unpredictable. Secondly, the injured muscle often shows increased automatism, and this dangerous excitability may be provoked by digitalis, quinidine, or even procaine (Schaffer *et al.*, 1950; Miller *et al.*, 1952) into ventricular tachycardia or fibrillation.

Not counting cases of bundle-branch block or of sporadic ectopic beats 202 instances of rhythm disturbance have occurred in 144 patients (25.4%) of this series, and of these 50 (8.8%) showed more than one variety of arrhythmia in the course of the illness and 7 (1.2%) more than two.

Auricular Fibrillation

The general impression from the 61 instances with 26 deaths was that this arrhythmia tended to occur in the relatively grave case but did not of itself add much to the danger. The use of digitalis was restricted to those patients, 38 in all, in whom it was necessary to control a rapid ventricular rate, and small doses of quinidine were generally given concurrently to minimize the effect of the former drug on ventricular excitability. This combination restored normal rhythm quite readily as a rule, particularly where coarse f waves had been visible in the E.C.G., but in instances where the baseline showed fine or absent f waves the arrhythmia tended to persist. In the remaining cases either spontaneous reversion to normal rhythm or an already

slow ventricular rate made digitalis unnecessary, but there was no significant difference in the severity and mortality among those who required digitalis and received it and those who did not.

The proportion of anterior-wall infarcts to posterior was roughly the same among the patients who developed auricular fibrillation as in the series as a whole, 1.6:1, but it is of interest that the two definite cases of auricular infarction both showed this arrhythmia.

Auricular Flutter

The seven instances of this rhythm were all in gravely ill patients, of whom four died. A posterior infarct was known to be present in six of the cases, suggesting that auricular ischaemia rather than altered dynamics caused this rare and transient disturbance. The arrhythmia was persistent only in the single case with anterior infarction.

Paroxysmal Tachycardia

In this series paroxysmal supraventricular tachycardia resembled auricular flutter in that five of the six cases had posterior-wall infarcts. This arrhythmia showed a somewhat greater tendency to persistence, but four of the cases were converted to normal rhythm by oral quinidine and the other two reverted spontaneously. As an illustration of the variable response to quinidine in the post-infarction state it should be recorded that in one of the spontaneously reverting cases the supraventricular tachycardia had developed twice during attempts to restore with this drug normal rhythm in a case of auricular fibrillation.

Tachycardias of ventricular origin were more urgently dangerous and less readily controlled. Four of the seven cases responded to quinidine and one to intravenous procaine, but in two patients the persistence of this arrhythmia played a principal part in causing death. In spite of its danger, intravenous quinidine, given slowly with continuous E.C.G. control, stood out as the most effective agent available in what may be a desperate situation.

Heart-block

At least 80% of this group had posterior-wall infarcts, and while the overall mortality was high the combination of anterior-wall infarct and complete heart-block was invariably fatal. The results in this series provide a precise clinical illustration of the anatomists' findings (Gross, 1921; Glomset and Birge, 1945) that, although the main irrigation of the bundle is through the ramus septi fibrosi of the right coronary artery, there is a subsidiary supply from the ramus limbi dextri of the left anterior descending branch, with free mutual anastomosis (Lascano, 1943). Ischaemic changes in the bundle therefore imply inadequacy of both important vessels and widespread damage elsewhere. Moreover, if the heart-block follows occlusion of the left anterior descending branch it means that the right coronary artery, or less commonly the left circumflex (Bosco, 1943) if it passes the crux, is already occluded.

Nine of the 20 patients in this group showed other arrhythmias as well as heart-block, four instances of multifocal ectopic beats, three of auricular fibrillation, two of ventricular tachycardia, and one instance each of auricular flutter and ventricular fibrillation. This evidence of increased automatism of the low centres is a warning against misguided attempts to increase the ventricular rate with adrenaline or barium.

The slow idioventricular rate does not appear to be disadvantageous in itself when securely established. Where, however, there is fluctuation between partial and complete block, with dangerous periods of asystole, the use of sympathicomimetic drugs seems justified on the expectation of improvement in the nutrition of the ischaemic conducting tissues. Ephedrine has been used in two such cases of this series, with satisfactory abolition of Stokes-Adams attacks in each.

Multiple Ectopic Beats

Among the 78 patients in whom normal rhythm was disturbed by ectopic beats nine also developed auricular fibrillation and seven paroxysmal tachycardia. Heart-block occurred in four of the cases. There was a slight preponderance of posterior over anterior infarcts, but otherwise the only feature to distinguish this group from the series as a whole was a mortality that appeared incommensurate with the average severity of the initial lesions. Moreover, 18 of these patients died suddenly and unexpectedly, and, although ventricular fibrillation was demonstrated only once, the post-mortem findings in 16 of these cases suggested, by default of other explanation, that these deaths were due to a change of rhythm at a time when the danger from the infarct appeared to be receding.

It is true that a similar termination does occur in many cases without such previous disturbances, but I feel with Rubsamen (1951) that even unifocal ectopic beats represent a potentially dangerous ventricular excitability, and that where these multiply into runs of tachycardia or arise from different foci prompt action should be taken to suppress them.

Ectopic beats following cardiac infarction are readily abolished by oral quinidine or by intravenous procaine, but increasing reliance has been placed on the latter drug, which has been found safe and reliable.

Sinus Disturbances

The main artery to the sinus node arises from the longer circumflex vessel, usually the right (Lascano, 1943; Bosco, 1943) and so may be involved in posterior-wall infarctions. This has occurred in six cases of the series, the slower rhythm of the auriculo-ventricular node then replacing that of the inactive sinus node. Retrograde conduction to the auricle, sometimes delayed by block, was usual. Only one of these patients recovered. In another case of posterior-wall infarction (M59) the damage to the normal pacemaker was incomplete, and its discharge became intermittent for some days, finally recovering, and there was one instance (M106) of 2/1 sino-auricular block. Oscillation of the pacemaker and abnormal bradycardia also occurred.

Three cases of anterior-wall infarction showed a curiously exaggerated respiratory sinus arrhythmia during the initial stages. The one case (A41) of these examined at necropsy had absence of the left circumflex vessel, a very large atheromatous right artery, and thrombotic occlusion of the small left anterior descending branch.

Pericarditis

A general pericarditis appeared as a complication in at least 12 patients (2.1%), of whom 9 were men. The average age of the group was 66.2 years, and, with one exception, all were cases of great severity and only three patients recovered. Uraemia was a predisposing factor in one case and another had diabetes.

In nine cases (eight necropsies) the pericarditis was of the fibrinous type, with early purulent change in two and beginning fibrous adhesions in two others. Haemorrhagic fluid appeared in three instances, including one patient (H188) with a small posterior-wall infarct from whom 46 oz. (1,300 ml.) of blood-stained fluid was withdrawn in three tapings. This patient had not received anticoagulant drugs, but the other two were within the therapeutic range of dicumarol treatment, the possible influence of which is discussed under the section on haemorrhage.

Four of these patients had disturbances of rhythm. In one, auricular fibrillation was probably due to coexisting mitral stenosis, but in two others posterior-wall infarcts were thought to account for complete heart-block in one and sinus arrest followed by ventricular tachycardia in the second. In one patient only (H252) the late appearance of pericarditis coincided with a resistant ventricular tachycardia, but this relationship was suspected in a further case that was unconfirmed by necropsy.

In all the fatal cases gross general coronary atheroma was present to a degree that would make survival unlikely, but there was no clinical doubt that in four patients the onset of acute pericarditis hastened the end.

In the absence of effusion the diagnosis presented great difficulty except where the infarct was known to be confined to the posterior wall, because this complication usually arose during the first week, together with the usual plastic pericarditis over the infarcted area. It is therefore probable that its incidence in the series has been higher than that recorded, but it is difficult to assess the value of treatment. In two cases, however, penicillin appeared to effect control.

Respiratory Infections

There were 97 cases (17.1%) complicated by respiratory infections of significant gravity, and on 28 occasions there was clinical or post-mortem evidence that such a complication had played a material part in preventing recovery from the attack of cardiac infarction.

Chronic bronchitis is notoriously common in this age group of the London population, and in a large number of cases it was clear that the cardiac catastrophe had provoked an exacerbation of long-standing disease. The circulatory depression, the use of morphine, and the immobility during the first days are obvious dangers in such cases, and unless particular care is taken the shocked patient is easily chilled during the initial examination. Exogenous respiratory infections have usually arisen in the second or third week, and here it is clearly a matter of cross-infection in the ward. There are serious psychological reasons against the segregation of patients with coronary disease into separate units, but an open medical ward is likely to contain, especially during the winter and early spring, many bad companions. The possibility of droplet infection can be minimized by careful arrangement of the beds, bearing in mind the particular danger of the virus pneumonias and *H. influenzae* infections.

Respiratory complications following cardiac infarction often arise in so insidious a manner that the altered physical signs may be attributed to pulmonary congestion or oedema due to progressive left ventricular failure and the essential treatment withheld until purulent sputum appears. The experience of these cases suggests that asthenic bronchopneumonia should always be suspected if at any time during the course of the illness there appears a vaguely defined deterioration in the general condition, unaccompanied by pain or a fall in blood pressure. It may be necessary to recover the sputum by tracheal aspiration, as the temperature chart may well be undisturbed and the tell-tale flush fail to appear on the face.

Treatment with the appropriate antibiotic is generally so effective that the presence of pus in the bronchi at necropsy must be accepted as a reproach to the physician. A course of penicillin has also been found of great protective value in chronic bronchitic subjects who develop cardiac infarction.

Mental Changes

The dramatic interruption of the settled life of a middle-aged person by an attack of cardiac infarction is a severe test of the character and mental stability of the patient. In great physical distress he has suddenly to confront the immediate possibility of death or the economic consequence of a long illness. His mental clarity may be dulled at first by sedatives and an impaired cerebral circulation. Later, he is likely to ruminate over the sudden deaths of his contemporaries or juniors from coronary disease, and to lose confidence both in his chances of recovery and in the good faith of those who encourage him, regarding any innocent somatic or digestive pain as the forerunner of the disaster that he sees as inevitable.

In the present series there were 14 cases (2.5%) of psychosis resulting from the attacks. The number of cases of cardiac neurosis that developed later is considerable, but

the abnormal anxiety shades imperceptibly into the justifiable, and precise figures cannot be given.

The psychotic cases comprised eight men and six women, and, while the average age of the men was 65.5 years, that of the women was over a decade higher at 77.3 years. Confusion, delirium, and stupor occurred in eight of these subjects, hallucinations and delusions in five, and reactive depression in one. One patient (H25), a charming and courteous old gentleman before and after the attack, spent two days in a wild bottle-throwing frenzy. The prognosis of the mental state in all these patients was identical with that of the physical condition.

Anxiety states following cardiac infarction occurred almost exclusively in men. They differ essentially from the usual cardiac neuroses in that the fears on which they are based are unfortunately not unlikely to be justified by future events, and it is not often possible to give the candid reassurance that might dispel them. The reaction to the fear is, however, abnormal, and the patient can be helped by sharing with him the available information about the sound healing of the original infarct and explaining the innocent nature of the symptoms that his heightened sensibility notices and his anxious mind tends to misinterpret. Prophylactically, it has been found valuable to give the patient at an early stage a rough outline of the proposed treatment and estimated length of the illness, without precise promises, so that his mind is directed at the onset away from the discomforts of the present towards the expected recovery. For the rest, prevention seems to be more a matter of what is left unsaid than of what is explained. The anxious patient shows an extraordinary propensity to seize on a chance remark by doctor, nurse, or physiotherapist that seems to confirm his fears and to erect an unassailable neurosis upon it.

Thyrototoxicosis.—Four male patients (0.7%) developed thyrototoxicosis during their stay in hospital. Each was of a conscientious and over-anxious personality, but showed no evidence of thyroid dysfunction on admission. In each case, too, the attack of infarction was a severe one, with a great deal of pain. All four patients, whose average age was 53.5 years, made good progress on diminishing doses of propylthiouracil, and it seems certain that the temporary disturbance resulted from the acute anxiety experienced in the attack.

Other Complications

Bacterial infection of the urinary tract occurred in 18 (3.2%) cases, but this complication was usually easily recognized and treated and had little prognostic significance. Apart from four men with prostatic enlargement the patients were all elderly women.

Arteriosclerotic renal disease is a common concomitant of cardiac infarction, and 17% of these cases had on admission a urinary specific gravity lower than 1012. In four cases the cardiac infarction precipitated death in uraemic coma.

As this series represents over 40 years of patient-time in hospital there has been a variety of coincidental disease. In particular, an unrelated acute abdominal emergency developed in nine (1.6%) of the patients, two of whom died as a result. Such double catastrophes may present clinical problems of the utmost delicacy.

Summary

The early complications that occurred in a consecutive series of 567 cases of cardiac infarction are enumerated and discussed under separate headings, with special reference to their influence on prognosis and to their prevention and treatment.

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DIAGNOSIS OF CARDIAC INFARCTION

BY

R. S. STEVENS, M.D., M.R.C.P.

S.H.M.O. (Physician), Royal Sussex County Hospital, Brighton

John Hunter suffered a severe and prolonged attack of angina pectoris in 1773, the year following the publication of Heberden's original account of the disease. When Hunter died, in 1793, Edward Jenner found post-mortem evidence of old posterior-wall myocardial infarction. More than a century elapsed before the clinical diagnosis of coronary artery occlusion and cardiac infarction became established: major advances included the work of Dock (1896), Marie (1896), Obrastzow and Straschesko (1910), Herrick (1912, 1919), and F. M. Smith (1918). Parkinson and Bedford (1928) published an account of the disease that remains fundamental and comprehensive, as a result of which the criteria for the clinical diagnosis have been widely understood in this country for over twenty years, while further advances have been chiefly technical, in the field of electrocardiography. It has been stated that there is a tendency to make this diagnosis too readily or unjustifiably (White, 1937; Wood, 1950), and that "errors of diagnosis do occur in both directions, and some are unavoidable" (Levine, 1950).

It was observed that several cases of cardiac infarction came ambulant and undiagnosed to the out-patient department—a fact which prompted investigation of the circumstances of diagnosis in a series of cases. It is suggested that the diagnosis may be regarded as "missed" when this is delayed for longer than an arbitrary period

of seven days (Stevens, 1952). Cardiographic confirmation might be delayed for longer; the essential is that firm clinical diagnosis should ensure early treatment.

This paper is founded on detailed analysis of the records of 250 consecutive patients with cardiographic (and, where applicable, post-mortem) evidence of cardiac infarction seen at the Royal Sussex County Hospital between 1947 and August, 1951. Surviving cases were followed up on October 31, 1951.

From the histories of cases in which the diagnosis had been delayed it was seen that these fell into two groups: those in which it was possible to judge, from the history, when the attack had occurred; and those in which the incident of infarction was ill defined. From these two it was possible to select a third group: cases in which symptoms were related solely or chiefly to effort, and in which symptoms, if occurring at rest, were of brief duration.

The series comprised 216 males and 34 females. The cases were divided as shown in Table I: those diagnosed within seven days (group N, 156 cases) and three groups

TABLE I

	No. of Cases
Cases diagnosed within 7 days (group N)	156 (72.4%)
Missed cases in which the date of onset is known (group A)	54 (25.1%)
Missed cases in which the date of onset is uncertain (group B)	23 (10.6%)
Missed cases with angina solely or chiefly on effort (group C)	17 (7.9%)

of missed cases, 94 in all, or 38% of the series. The latter are subdivided into group A, in which the exact interval (varying from eight days to six years) between onset of infarction and diagnosis can be assessed; group B, in which the date of infarction is uncertain (except within wide limits varying from a few days up to six years); and group C, cases of angina of effort (of one month's to thirteen years' duration), some of which could have been included in group A and the others in group B.

Illustrative Case Histories

Group A (Case 76).—A hairdresser aged 63 was sent as an out-patient. Eighteen days previously, while at breakfast, he had a sudden onset of pain across the chest and down the left arm; this lasted in a mild form all day. He remained in bed for two days, since when he had noticed similar pain on walking uphill. On examination the pulse was regular, the heart sounds were closed, and the blood pressure was 130/80. An electrocardiogram showed posterior-wall infarction. He was advised to return to bed for one month; he recovered and was well two years later.

Group B (Case 18).—A retired farm manager aged 58 was admitted for surgical investigation. He had been well until two years previously, when increasing lassitude and weakness forced him to retire. For six months he had noticed epigastric pain and distension, with a feeling that food was "sticking at the bottom of the chest." This had become worse but was intermittent. His capacity for exercise had become greatly reduced. He had developed pain in the left arm; he occasionally had shooting pains in the left chest, radiating to the elbow and "taking away the use of the arm." This had become worse recently. Among other investigations the electrocardiogram showed evidence of posterior cardiac infarction. At laparotomy an inoperable carcinoma of the stomach was found, and he died six weeks later.

Group C (Case 147).—An insurance superintendent aged 50 consulted his doctor because he had recently been rejected for life assurance by two companies. For two years, and especially for the past six months, angina occurred on