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DIAGNOSIS OF CARDIAC INFARCTION

BY

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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

exertion after meals, but so mildly that he did not have to stop walking. He was able to play golf, and lost his pain after five or six holes—first-effort angina (Price, 1951). He had never had pain at rest, and it had never been prolonged. He was hypertensive; his blood pressure was 190/110. An electrocardiogram showed evidence of posterior cardiac infarction. At the follow-up he had only slight occasional effort-pain.

Clinical History

Analysis of the histories shows little or no significant difference, as between the groups, in such factors as age and sex, history of previous anginal pain, associated diseases such as diabetes mellitus or intermittent claudication, or precipitating factors, whether physical or emotional. The circumstances and mode of onset reveal some differences between the groups. In groups N and A there is a slight preponderance of attacks with onset at rest compared with attacks coincident with or apparently consequent upon physical or mental activity; usually, however, the onset was unmistakable on the history alone. In group B six patients related their symptoms to effort and five to various periods of effort, rest, or emotion (it being impossible to decide, retrospectively, which had been the most significant occasions); in six others the onset was insidious—three with heart failure, a fourth masked by symptoms of gastric carcinoma, while in two the onset remains obscure; four patients suffered recurrent attacks of nocturnal dyspnoea; one persisted in misinterpreting the symptoms as due to dyspepsia; and in one case the first sign of illness was the sudden occurrence of a complication—pulmonary embolism, itself misconstrued as acute bronchitis. In group C, 11 patients related their symptoms solely to exertion; in four others there were symptoms chiefly on exertion, but also short mild attacks at rest; while in one there were effort-pains and brief (five-minute) attacks of angina decubitus; in one case there was pain not only on walking but also on change of posture although not, otherwise, at rest; the majority in this group supplied clues to the true diagnosis, such as sudden onset of angina, increasing severity thereof, or progressive diminution of the distance covered without pain.

Pain was the presenting symptom in 231 cases (92%) in the series. Of 26 different descriptions of pain, varying from a "fireball in the navel working up to the throat," to "rough cinders behind the breast-plate," words indicating severity appear in every fourth history in group N but only in every eighth in group A and in none in the other two groups. Conversely, painless attacks occurred in 2.5% of group N, 5.5% of group A, and 30% of group B (in which, also, 21% of attacks were masked by the more prominent symptoms of other diseases—carcinoma, biliary colic, sudden hemiplegia—or revealed by the development of complications).

Duration of pain was brief in all cases of group C, and is known in only 10 of the 23 in group B. But in the other two groups we have comparable figures: 133 cases in group N, and 44 in group A (Table II). Except for the two ex-

TABLE II

Group	Duration of Pain					Total
	½ hr.	1 hr.	2-6 hrs.	12-24 hrs.	Days	
N	2	19	58	47	7	133
A	4	6	18	14	5	44

amples of very short or very long attacks the duration is closely comparable in these groups. It might seem, therefore, judged by this series, that one in every four persons suffering typical pain of cardiac infarction would not be correctly diagnosed within seven days.

It had been thought possible that the common interpretation by patients of their symptoms as due to "indigestion" would explain delay in diagnosis in many cases; the diagnosis was delayed either by patients or by doctors (or both)

on this account in some cases, but in fewer than had been anticipated. Coexistent digestive disorders, peptic ulcers, cholecystitis, etc., gave rise to considerable diagnostic difficulty in a few instances.

Nausea, vomiting, collapse, giddiness, and faintness were all noted in a much greater proportion of cases in group N compared with missed cases.

Physical Signs.—Three physical signs merit comment. Consistently with the preceding paragraph, fall of blood pressure following the attack is known to have occurred in relatively more cases of group N than among missed cases. In group N 87 cases are known to have had fever (of an average duration of 5.9 days), whereas in only 16 was there definitely no rise of temperature and in 53 there was no evidence either way. In all the other groups there are only six cases, in group A, in which there was known to have been a rise. Of 136 letters from doctors, the temperature was mentioned in only 5. But reference in such letters to the presence or absence of pericardial friction suggests that this is a sign on which great, perhaps undue, reliance is placed. The overall incidence in this series was 5.6%.

Electrocardiography

The only confirmatory test of value in retrospective diagnosis is electrocardiography. There were 282 known incidents of infarction among the 250 cases. Electrocardiograms are available for 279 attacks. The geographical details of these records have been simplified to show predominantly anterior or posterior patterns. In Table III it is shown that the trend of the ratio of anterior to posterior (102 : 77) in group N is reversed or modified in all missed groups.

TABLE III.—E.C.G. Site of Infarction

Group	Anterior	Posterior
N	102	77
A	28	28
B	12	14
C	3	15

Complications

The incidence of thrombo-embolism was 7% in groups N and A, 13% in group B, and nil in group C. (Nearly half of the cases in group N were treated with anticoagulants.)

Excluding patients who died during a first attack, the known duration of post-infarction angina of effort to the date of follow-up or last consultation or death was calculated. Some degree of post-infarction angina was found in 41% of group N, 70% of group A, and 55% of group B, while no case in group C was known to have become symptom-free.

TABLE IV.—Post-infarction Angina

Group	Not Known	No Angina	Angina of Effort
N	23%	36%	41%
A	12%	18%	70%
B	20%	25%	55%
C	10%	—	90%

The symptom was noted for longer than eighteen months in about 22.5% of group N, 25% of groups A and B, and 56% of group C. These figures may indicate a trend but not necessarily a true picture, because in group N the percentage of cases in which evidence is lacking is double that in group A. Inadequate rest is the most probable explanation for the high incidence and persistence of this symptom in missed cases.

No case of cardiac aneurysm was proved.

Marked neurotic symptoms followed infarction in 15 patients of group N and 11 in the missed groups; but more than half of these were known to have had neurotic manifestations before this illness.

Mortality

Sixty-eight patients are known to have died, but not all from cardiac infarction. The corrected figures, after excluding deaths from other or unknown causes, are shown in Table V.

TABLE V.—Deaths

	From All Causes	From Coronary Artery Disease
Whole series	27%	23.2%
Group N	27%	23.7%
" A	14.8%	13%
" B	61%	48%
" C	23.5%	18%

Discussion

Accurate early diagnosis of cardiac infarction is of increasing importance not only individually but socially because the incidence of coronary artery disease is increasing (Cassidy, 1946; Morris, 1951). Cassidy showed that in England and Wales the crude death rate from this disease was 48 per million living in 1926, and 473 per million living in 1939. The Registrar-General (1952, personal communication) has written that the latter figure would now be adjusted to 433, but that the comparable figure for 1949 was 996. The disease takes a particularly crushing toll of the lives of professional and executive workers at the peak of their usefulness (Ryle and Russell, 1949; Morris *et al.*, 1952).

It has been suggested above that the diagnosis would be less often missed if patients' temperatures were observed carefully for the first few days after any suspicious incident. Parkinson and Bedford (1928) found fever to be a more useful diagnostic sign than leucocytosis. Yater *et al.* (1948) studied the charts in 274 cases: 82% showed fever, which started to rise in 4 to 48 hours from onset and lasted on an average for six days. In the present series temperatures were mentioned in only 3.7% of 136 letters from practitioners.

Pericardial friction is not so common as seems to be supposed. This may be due to the high incidence observed by some earlier authors (Gordinier, 1924; Coombs, 1927), although Levine (1929) noted the sign in only 13.8% of 145 cases. Recent estimates of its incidence vary from 7% (Yater *et al.*, 1948) to 10% (Wood, 1950).

Confusion persists about terminology. Parkinson (1932) wrote: "Our terminology has been tangled by confusing angina (disease) with anginal pain (specific symptom)." Too often one sees the terms "coronary occlusion" and "cardiac (or even coronary!) infarction" used synonymously. Acute coronary insufficiency (Master, 1947) has been ably summarized by Wood (1950), but, in my experience, the term is too often used as an alternative to coronary occlusion or cardiac infarction, and apparently as a reason for not enforcing sufficiently prolonged treatment.

A point that arises in connexion with the missed cases is whether the reversal of the normal preponderance of anterior over posterior infarctions (see Wartman and Hellerstein, 1948; Yater *et al.*, 1951) is significant or whether it is fortuitous. In their series of 100 ambulant cases Fagin and Chapnick (1950) found that 52 patients with typical histories had cardiographic evidence of 34 anterior and 18 posterior infarcts; among 41 atypical cases (including some with angina of effort only) there were 26 anterior and 15 posterior infarcts, whereas in seven patients who were said to be symptomless two had evidence of anterior and five of posterior lesions. Although there were more anterior than posterior infarcts in the small series of cases of effort angina reported by Stein (1950), yet Wade and Morgan Jones (1951), in a series of 17 cases of this type, found 12 posterior and 5 anterior tracings. In group C of the present series the ratio was 3 anterior to 15 posterior infarcts.

Group A includes a majority of cases with mild clinical course and high survival rate, but with long ultimate dis-

ability as judged by the persistence of angina. Nevertheless, the mortality is not negligible, and we can only judge of those who survive long enough to reach us in the outpatient department. But in a large series Yater *et al.* (1948) reported that in 83% of fatal cases the patient died within the first twenty-four hours. However, Papp and Shirley Smith (1951) were able to show a close relationship between mortality rate and severity of attack, which is clearly borne out in the present series. Mild cases were foreseen by Herrick (1912). Levy (1931, 1932, 1936) stressed the rapid recovery from the initial attack, the fact that fever was found, that serial electrocardiograms might be necessary, and that the heart should be safeguarded from further damage. Howard (1933) observed that these cases should be diagnosed on clinical grounds alone and emphasized the rise in temperature, which Papp (1949) also noted in all cases of moderate severity and in a few of the most minor.

It has been recognized for at least seventy years that coronary occlusion and myocardial infarction may be associated with paroxysmal dyspnoea without anginal pain (Kidd, 1882; Obrastzow and Straschesko, 1910). The painful symptoms of infarction may be absent in the failing heart. An incidence of about 7% of painless attacks seems to be fairly generally accepted; the painless onset is said to be one of the principal sources of diagnostic errors (Bean, 1938), and it is notable that they form such a high proportion as 30% of group B of missed cases.

Gallavardin (1921) discussed the possibility of an infarction initiating the syndrome of angina of effort in a person previously immune from angina. Parkinson and Bedford (1928) observed that coronary thrombosis might initiate angina pectoris, complicate its course, or prove the fatal termination. Morgan Jones and Wade (1949) reviewed 62 patients with angina pectoris, with pain confined to exertion, invariably relieved by rest, and never lasting longer than fifteen minutes: cardiographic changes diagnostic of infarction were found in 28% and suggestive of infarction in another 20%. In France a series of 354 cases of angina of effort revealed cardiographic evidence of infarction in nearly 10% (Heller *et al.*, 1951). The corollary to these reports appears in the present paper, in which similar criteria are shown to have characterized nearly 7% of 250 cases of infarction.

I have considered the several ways in which precise diagnosis may elude recognition. Various authors (e.g., Bedford, 1933; Levine, 1938) have shown how a careful history may bring to light some forgotten incident of pain or dyspnoea. But there is increasing evidence that infarcts may occur silently (Moor and Rogers, 1932; Brill, 1938; Keith Simpson, 1939; French and Dock, 1944; Cassidy, 1946; Mathur and Wahi, 1951). Nevertheless, other authors insist that the incident of even minor infarctions revealed at necropsy can always be reconstructed by a careful history (Laubry and Soulié, 1950).

Summary

Of 250 hospital cases with cardiographic evidence of cardiac infarction, 156 (62.4%) had been diagnosed within seven days, group N. In this group attacks were often severe; 2.5% were painless.

The other 94 cases (37.6%) had been missed as defined, and are considered in three groups.

In 54 missed cases, group A, it was possible to make an accurate retrospective diagnosis from the history; the majority of attacks were relatively mild; 5.5% were painless.

In 23 missed cases, group B, it was difficult (for example, because of multiple attacks of pain) to judge exactly when the incident of infarction occurred; 30% of these attacks were painless and 21% masked. Mortality was high in this group; the underlying cause of painless paroxysmal dyspnoea is too often overlooked.

In 17 missed cases, group C, the patients suffered angina pectoris virtually confined to effort.

The diagnosis depends, above all, on a careful case history. Fever, perhaps the most useful, is the most neglected physical sign; by contrast, excessive reliance is placed on the presence or absence of pericardial friction.

There is evidence that disability was more prolonged in (even mild) missed cases than in those diagnosed and treated early.

The significance of and reasons for delayed diagnosis, and the cardiographic changes, are discussed.

It is a pleasure to record my thanks to the consultant physicians at the Royal Sussex County Hospital, who allowed me to use their case notes, and particularly to Dr. R. Kemball Price, who advised me and encouraged me to use the records in the cardiac department; also to Sir Lionel Whitby, Regius Professor of Physic, for permission to publish this abstract from a thesis submitted for the degree of M.D. of the University of Cambridge.

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The Hyderabad health museum, one of the best of its kind in the Eastern Hemisphere, was recently rehoused in a new building in Hyderabad City, where it attracts many visitors. The high illiteracy rate in India makes the visual method of presentation and instruction all the more important in public health education. The museum includes models, posters, photographs, charts, and other visual aids. Films and lantern slides are shown regularly, and a van equipped with projectors and printed literature makes tours throughout the State of Hyderabad. The resources of the museum are fully described and catalogued in the second edition of an attractive 48-page illustrated handbook which has just been published by the Bureau of Health Education, Public Health Department, Hyderabad.

FAMILIAL HYPERTROPHIC PYLORIC STENOSIS

BY

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Congenital pyloric stenosis in the adult, first reported by Maier in 1885, is not so very rare, and most surgeons will encounter a case from time to time. The congenital condition occurring in infants is relatively common, and the literature contains a number of reports of cases occurring in two or more siblings. Gailey (1948) reports four proved cases in one family and mentions another family with four affected members reported by Finkelshtein and quoted by Sauer (1924). There are occasional reports of cases occurring in successive generations, and Schrieber, quoted by Laubscher and Smith (1947), described a patient whose father and grandfather both suffered from pyloric stenosis in adult life. In the family reported here hypertrophic pyloric stenosis was discovered at operation in a man aged 39; he subsequently revealed that two of his four sons had had Ramstedt's operation performed upon them in infancy, and he had a nephew treated medically for the condition in infancy.

In addition his eldest brother had died in infancy from persistent vomiting. The accompanying genealogical table shows the incidence of pyloric stenosis in this family.

Case 1

A man aged 39 first attended hospital on January 21, 1952, complaining of recurrent attacks of epigastric pain and heaviness after meals for 10 years. He had been investigated in 1944, when x-ray examination was said to show a duodenal ulcer and medical treatment was advised. Records of that period during the war are unfortunately missing. He had had no other serious illnesses, no previous operations, and gave no history of gastric trouble in childhood. His pain usually came on soon after meals, but was sometimes delayed for two and a half to three hours. It was situated in the epigastrium, did not radiate, and lasted 20 minutes to one hour. Often it would "make him lie down exhausted." Attacks would occur over a period of a few weeks, once or twice a year, but without seasonal incidence. Heaviness after meals was usual, and he was always a small eater. He often suffered from acid regurgitation, but vomited only occasionally. When he did vomit his pain and heaviness were always relieved. There had been no haematemesis or melaena, and weight loss had amounted to 7 lb. (3.2 kg.). His last attack had been more severe than any before and he had been off work for two weeks. He had lost an average of one to two weeks' work a year during previous attacks.

On examination he appeared to be a stable type of patient, rather thin but in fair general condition. In the abdomen there was only tenderness in the epigastrium; no tumour was palpable and succussion splash was not obtained. Barium-meal examination showed a large stomach with good tone, but it was empty in four hours. The duodenal cap was tender and irritable, and there was a suspicion of an ulcer crater in the right fornix. At this time the pyloric condition was not considered, and the x-ray appearance of the pylorus