Papers and Originals

THE AETIOLOGY OF COMPLETE HEART-BLOCK

BY

MAX ZOOB, M.D., M.R.C.P. K. SHIRLEY SMITH, M.D., F.R.C.P.

From the Cardiac Department, Charing Cross Hospital, London, and the Regional Thoracic Unit, Brook Hospital, London

[WITH SPECIAL PLATE]

That complete heart-block in the elderly is often due to coronary sclerosis has been, and still is, very widely assumed. The prevalence of coronary heart disease in the later decades of life, when such block commonly occurs, and the occasional appearance of complete heart-block in cardiac infarction have lent colour to this assumption. Moreover, the weighty affirmations of earlier authors have tended to discourage a critical attitude in this matter. The purpose of the present paper is to study the aetiology of complete heart-block and in particular to weigh the evidence upon which this disorder has been attributed to coronary disease. It is well first to review earlier relevant publications (Table I).

TABLE I.—*Aetiology of Complete Heart-block as Described in Four Recent Papers by Other Authors*

Cause	Ide (1950) (71 Cases) (%)	Penton *et al.* (1956) (251 Cases) (%)	Rowe and White (1958) (278 Cases) (%)	Gilchrist (1958) (46 Cases) (%)
Ischaemia	58	42	49	60
Hypertension	—	24	24	—
Rheumatism, syphilis, etc.	42	27	24	37
Unknown	—	7	3	3

Friedberg (1956) stated that coronary artery disease was the chief cause of heart-block, while Rowe and White (1958) studied 278 cases and attributed 35% to previous ischaemic heart disease; they found 24% to be due to hypertensive heart disease and 14% to acute cardiac infarction. In Ide's (1950) series of 71 patients 58% were attributed to coronary artery disease; in eight cases there was cardiac infarction. Penton *et al.* (1956), studying 251 patients, considered 42% to have a coronary and 24% a hypertensive aetiology.

Contrasting results were obtained by Gilchrist (1958), who found no conclusive example of chronic complete block following cardiac infarction and noted that angina was uncommon in patients with complete block. Also Campbell (1944) found evidence of ischaemic heart disease in only 17 of his 74 cases, and in 23 no cause for the block was evident. Gallavardin (1957) found no cardiopathy other than the block itself in 30 out of 50 cases studied, and concluded that coronary atheroma was not the usual cause of block.

Material and Methods

A group of 51 patients derived from hospital and consulting practice has been studied. Their ages ranged from 34 to 85 and averaged 65. There were 37 men and 14 women. Particular attention was paid to the question of ischaemic cardiac pain and the possibility of previous or present cardiac infarction. The patients were observed over periods ranging from a few months to several years, and when patients had been seen only on one occasion information about their subsequent course was obtained from their practitioners. Eighteen patients were followed until death ensued, but necropsy findings were available in only three cases, many of the remainder having died suddenly in their homes. In two of the three necropsies the conducting tissues were examined histologically according to the method of R. E. B. Hudson (1962, personal communication). A piece of tissue, including the A-V node, bundle of His, and bundle branches, was sectioned at 0.5 mm. intervals. This provided between 50 and 70 sections which were stained with haematoxylin and eosin.

Results

In 30 of the 51 cases no indication whatever of the aetiology existed and the arrhythmia was regarded as the primary disorder. In the remainder a recognized cause of A-V block was discovered and the incidence of the various types is shown in Table II. These groups are now considered individually.

TABLE II.—*Causes of Complete Heart-block in the 51 Cases Studied*

Cause	No. of Cases
Primary	30
Posterior infarction	8
Primary with associated ischaemic heart disease and episodic angina	4
Syphilis	4
Aortic stenosis	3
Congenital	1
Infection	1

Primary Heart-block

Patients were placed in this category when there was nothing whatever to suggest angina or infarction and when it could confidently be stated that none of the known causes of heart-block were present. There were 30 patients in this group—20 men and 10 women; the average age was 67 (range 34–86 years).

Mode of Presentation.—In 23 of the 30 patients the first symptom was syncope. In a further three consciousness was not lost and the patient complained merely of attacks of dizziness. Three patients experienced paroxysms of dyspnoea, and in one of them the symptom was observed to coincide with extreme bradycardia. Two patients complained of effort dyspnoea, and in only one was the block discovered fortuitously.

Persistence of the A-V Block.—In this group heart-block was permanent in all but four instances and was observed for periods of a few months to 11 years. Two of these four transient cases reverted to sinus rhythm, but in one of them complete block returned and proved fatal. The other two developed atrial fibrillation, one persistently with right bundle-branch block, the other temporarily with final reversion to complete A-V block.

Blood-pressure.—This was of special interest, as previous authors have thought hypertension to be an important cause. Taking 145/90 mm. Hg as the upper limit of normal, we found that only five patients had diastolic hypertension, while 15 had systolic hypertension with a large pulse pressure. The average age of the latter group was 73.8 (range 53–86 years), contrasting with 65.5 (52–75) in those with diastolic hypertension, and 60.5 (34–74) in

those with normal pressure (Table III). Diastolic hypertension therefore occurs too infrequently to be a causative factor and systolic hypertension may be merely the effect of a large stroke volume (due to bradycardia) in elderly persons with inelastic arteries.

TABLE III.—*Primary Heart-block: Blood-pressure and Age in 30 Patients*

Blood-pressure	No. of Cases	Age in Years	
		Mean	Range
Normal	10	60·5	34–74
Diastolic hypertension (>90 mm. Hg)	5	65·5	52–75
Systolic hypertension (>150 mm. Hg)	15	73·8	53–86

Murmurs.—Ten patients had systolic murmurs, of which five were thought to be related to aortic ejection and caused by the large stroke volume. In four cases the murmurs were quiet and ill-defined, and one patient had aortic stenosis and incompetence. Twenty had no murmurs.

Prognosis.—The survival of patients has been calculated either from the first syncopal attack or from the cardiographic evidence of block if this occurred first. Thirteen patients were observed until death ensued. In five heart-block proved fatal within six months, and a further two patients died of other causes within the same period. Of the remainder, two survived for two years, and one each for three, five, nine, and eleven years. The average age at death of these 13 patients was 67.4 (range 49–75 years). The period of observation of the 17 patients still living has been less than one year in six cases; of the remainder, three have survived for one year, three for two years, one for three years, and two each for six and ten years. The average age of nine patients observed for a year or more was 64.7 (range 34–84 years) at the onset and 68.7 (44–86) when last seen. It therefore seems that if the first few years are survived the patient may live for several years and the expectation of life may be but slightly reduced. One-quarter of our patients have survived for five or more years from the onset of the disorder.

Necropsy Findings.—These were as follows.

Case 1

The patient, a woman of 76, had complete block for over a year. The heart, which was studied by Dr. R. E. B. Hudson, weighed 600 g. The valves were healthy but all the chambers were dilated and there was hypertrophy of both ventricles, the right being 6 mm. thick and the left 14 mm. The coronary arteries were atheromatous and the lumen of the left anterior descending branch was reduced by one-half at a point 5 cm. from its origin. There was no occlusion anywhere, and in particular the lumen of the right coronary was easily followed to the posterior interventricular sulcus. There was a small linear scar in the antero-lateral wall of the left ventricle but no gross or microscopical evidence of infarction.

Histologically the outstanding finding was widespread increase of the interstitium of the left ventricle with small areas of replacement fibrosis and lymphocytic foci suggesting an extinct inflammatory process (Special Plate.* Fig. 1). The pacemakers and conducting tissues were studied in detail. The sino-auricular node showed severe fibrosis although its arteries were patent. The His-Tawara system was studied in 77 serial sections at 0.33-mm. intervals. The main A.-V. node was elongated and there was some increase of interstitium with intimal thickening of the arteries. In the lower part of the bundle of His the conducting tissues were interspersed with fibrous tissue and there was one small focus of lymphocytes. The left branch was completely separated from the main bundle by fibrous tissue (Special Plate, Fig. 2). The fibres of the right branch were few in number and were interspersed with fibro-fatty tissue (Special Plate, Fig. 3). The interventricular septum showed tiny scattered areas of replacement fibrosis and scattered small foci of lymphocytes. The walls of many of the smallest arteries showed hyaline necrosis with mural or occluding thrombosis.

In summary, the heart showed severe fibrotic changes in the sino-auricular node, complete interruption of the left bundle branches, and partial interruption of the right bundle branch by fibrous tissue. The relatively slight coronary disease and the presence of small fibrotic and cellular lesions throughout the myocardium as well as in the conducting tissues suggested that the process was a disseminated focal myocarditis.

Case 2

The patient, a man of 72, died after two years of heart-block. The heart weighed 465 g. There was no abnormality of the valves or pericardium, but moderate atheroma of the coronary arteries was present. The anterior descending branch of the left coronary was considerably narrowed 2 cm. below its origin, but there was no occlusion either in this vessel or in the right coronary system. The lumen of the right coronary was easily followed to the posterior interventricular sulcus and there was no obvious lesion in the vicinity of the bundle of His.

Serial microscopy sections were made of the conducting tissues. As in the previous case, the A-V node and the bundle of His appeared to be normal, but the fascicles of the left branches were separated from the main bundle by fibrous tissue. The origin of the right branch from the main bundle could not be found and it was therefore impossible to identify it in the remaining sections. There was a widespread fine fibrosis of the ventricular muscle, most pronounced in the interventricular septum, but also present in some degree throughout the heart. In this case lymphocytic foci were not found and the arterioles appeared relatively normal.

In summary, the heart showed widespread microscopical fibrotic lesions which interrupted the fascicles of the left bundle branch and did not appear to be ischaemic in origin.

Case 3

In this case a routine necropsy was performed but serial sections of the conducting tissues were not made. The patient was a woman of 69 who died after one year of complete heart-block. The heart weighed 365 g. and there was no hypertrophy of either ventricle. The pericardium was normal and the coronary arteries were widely patent. There was no sign of infarction and no macroscopic fibrosis of ventricular muscle. The cusps of the aortic valve were somewhat calcified in their central portions, but the valve appeared to be competent and not significantly stenosed. A single section of the myocardium showed a diffuse fine fibrosis which was judged not to be ischaemic by virtue of its character and the healthy state of the coronary arteries.

In these three cases of persistent complete heart-block necropsy showed scattered foci of myocardial fibrosis judged to be independent of coronary disease; these lesions could have represented an extinct inflammatory process. In each of the cases studied by serial sectioning the fibrosis involved the conducting tissues.

Ischaemic Heart-block

It was considered that heart-block could be attributed to ischaemic heart disease only if there was evidence of cardiac infarction or angina of effort. Such evidence existed in only eight patients, all of them middle-aged or elderly men. In one of them transient heart-block preceded posterior infarction by 29 years, but in the remaining seven cases the block followed more or less closely on a severe posterior infarction. In four cases it occurred during the first few days of the attack, in two it followed several months later, and in one case successive episodes of infarction culminated in heart-block 20 years after the first attack.

*The pictures illustrating this paper appear on the last page of the Special Plate.

In this instance the block has so far been present for six months, but in all the others it was transient, lasting a maximum of four days. The severity of the attack was shown by the presence of shock in three patients, severe and persistent or repeated pain in two, and a fatal outcome in two. In one patient block occurred only during the period of shock, and when the blood-pressure was raised by noradrenaline infusion the heart rate increased and the block disappeared (Fig. A). In the remaining cases sinus rhythm was resumed spontaneously, but in four right bundle branch block remained.

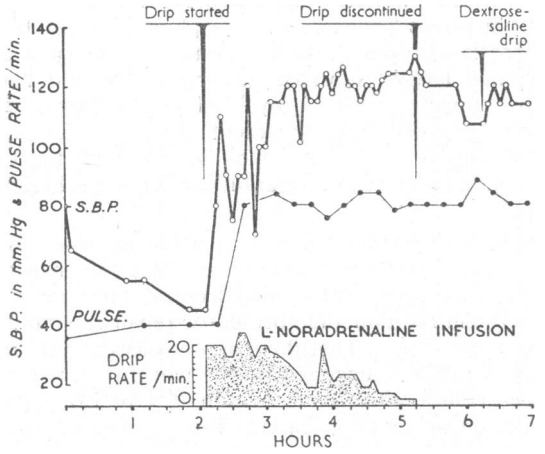

FIG. A.—Effect of an L-noradrenaline infusion on the blood-pressure and heart rate in a patient with posterior cardiac infarction and complete heart-block. Note that the abrupt rise of pressure from 45 to 110 mm. Hg is accompanied by doubling of the heart rate. The cardiogram then showed sinus rhythm.

Primary Heart-block with Associated Ischaemic Heart Disease and Episodic Angina

Four patients, three of them women, were seen with a clinical course sufficiently well defined to permit their consideration as a separate group. In all of them the conduction disturbance and evidence of ischaemia occurred independently and all four showed striking T-wave abnormalities. The appearance of complete heart-block was followed, after periods ranging from six months to nine years, by attacks of severe cardiac pain at rest always accompanied by deep inversions of the T waves in limb or chest leads or both; there were, however, no abnormal Q waves and no rise of temperature or E.S.R. to suggest infarction. One patient died shortly after such an attack but the remainder made good recoveries. In two of this group the development of block was preceded by angina of effort, but in the other two heart-block with syncope or heart failure was the presenting feature. As these four cases are of particular interest the following brief clinical details are given.

Case 4.—A woman of 66 developed angina of effort with normal sinus rhythm and no evidence of infarction. Three years later there were multiple syncopal attacks, each of which was followed by prolonged cardiac pain at rest, the cardiogram showing deep inversion of the T waves in leads I and V2-4 but no abnormal Q waves. Gradual recovery followed (Special Plate, Fig. 4), but heart-block has so far persisted for two months.

Case 5.—A man developed angina of effort at the age of 70; the T wave was low in lead II and inverted in lead III. One year later the cardiogram was normal, but six years later complete block spontaneously appeared and syncopal attacks followed. After three years of block, cardiac pain occurred at rest with very deeply inverted T waves in leads II and III but without significant Q waves. Complete block was then associated with atrial fibrillation and death followed in a few days. There was no necropsy.

Case 6.—A woman of 65 developed complete block with syncopal attacks. There was no sign of ischaemia or infarction in the cardiogram until eight years later, when a severe attack of pain at rest was accompanied by deep inversion of the T waves in leads II, III, and V2-6. These partially resolved, but a further similar episode occurred two months later and again four years later. The patient, now aged 77, remains well apart from her persistent block, which has so far been present for 12 years.

Case 7.—A woman of 66 developed congestive failure due to myxoedema and at the same time complete block was noted. Heart failure responded well to treatment with digitalis, diuretics, and thyroxine, but heart-block persisted. Six months later she developed angina of effort; shortly afterwards a prolonged attack of pain at rest was accompanied by deep T-wave inversions in leads I, II, and V2-6, but no abnormal Q waves. The transaminase (S.G.O.T.) was 11 units 24 hours after the onset. The T-wave changes gradually lessened, but the block has now been present for two and a half years.

In these four cases complete heart-block lasted 2 months, 2½ years, 3 years, and 14 years, and was punctuated by attacks of cardiac pain at rest with remarkable inversions of the T waves in limb or chest leads or both. In none of them were abnormal Q waves found. Three of these patients survived with some regression of the T-wave inversions but persistent A-V block; one died within a few days of his pain. The mechanism of this syndrome is discussed later.

Other Cases

Heart-block and Syphilis.—In four patients the W.R. was positive; one patient aged 60 had aortic incompetence and it was likely that syphilis was responsible for the block. The remaining three were aged 70, 76, and 78 years and showed no clinical evidence of syphilis. In view of this and their advanced age it seems somewhat unlikely that syphilis was responsible for the block and these patients may in fact be further examples of primary heart-block. However, as doubt remains they have been considered separately. Treatment with penicillin in three cases and prednisolone in two did not alter the block.

Aortic Stenosis with Heart-block.—Three patients had calcific aortic stenosis; in one case the murmur had been noted since childhood. Two patients presented with syncopal attacks, the third because a murmur had been discovered on routine examination. In the latter case complete block was preceded by left bundle-branch block and then 2:1 block as described by Gilchrist (1958).

Congenital Heart-block.—The solitary patient in this group was aged 22 and was known to have had a slow heart rate since the age of 10. There was an aortic ejection murmur with systolic hypertension of 160/50 at a heart rate of 44. There were no other signs of heart disease.

Discussion

In the past cardiographic abnormalities in the elderly were often attributed to ischaemic heart disease simply because of its commonness. More recently, however, it has been recognized that many disorders of rhythm and conduction occur in the absence of apparent coronary disease. In particular, both right and left bundle-branch block may occur in otherwise healthy people who seem to have a normal expectation of life (Wolfram, 1951). Despite this, the older habits of thought tend to persist and it is still widely believed that complete heart-block with its late age of onset, dramatic clinical features, and gross disturbance of cardiac physiology must inevitably be due to some serious form of heart disease such as coronary athero-

sclerosis or hypertension. The contrast between this traditional view and our own based on this study is summarized in Table IV. In our largest group, many of whom were women, we found nothing on which to base a diagnosis of ischaemic heart disease and yet the block was almost always permanent. By contrast, when coronary disease was made obvious by the occurrence of severe posterior cardiac infarction the patients were all men and the block was almost invariably transient. The clinical differences between the two groups are summarized in Table V.

TABLE IV.—*Aetiology of Complete Heart-block: Range of Opinions Expressed in the Four Recent Papers Quoted in Table I Contrasted With Those of the Present Series*

Cause	Studies by Other Authors (646 Cases) (%)	Present Study (51 Cases) (%)
Primary	—	60
Primary with associated ischaemia	—	8
Ischaemia (posterior infarction)	42–60	16
Aortic stenosis	—	6
Hypertension	0–24	—
Miscellaneous	3–42	10

TABLE V.—*Contrasting Clinical Features in 30 Cases of Primary Heart-block and Eight Cases of Ischaemic Heart-block in Present Series*

Feature	Primary	Ischaemic
Men women	20 10	8 0
Presentation	Fainting. Paroxysmal dyspnoea	Pain
Duration	Usually months or years	Usually hours or days
Permanent block	26 (out of 30)	1 (out of 8)

It is interesting to speculate on the mechanism of the conduction disturbance and the reason for its transience in cardiac infarction which Gilchrist (1958) has also noted. It seems most unlikely that direct involvement of the bundle of His by the infarct is responsible. If this were so, in at least some of the patients the necrosis would surely cause irreversible damage to the conducting tissues. Moreover, the bundle, which is only about a centimetre long, lies high in the interventricular septum just behind the membranous portion. Infarction usually occurs lower down in the region of the bundle branches, and permanent bundle-branch block is a common sequel of septal infarcts; complete block, however, would require involvement of the right branch as well as the fascicles of the left, and is apparently rare, though it may perhaps explain our solitary case of persistent complete block following successive infarctions.

It seems likely that A-V block occurring in cardiac infarction has a quite different explanation. It occurs in association with posterior cardiac infarction, which is usually a result of an occlusion of the right coronary artery. Terminal branches of this vessel are mainly responsible for the blood-supply of the A-V node, which presumably becomes ischaemic when this vessel is obstructed. Infarction in the auricle is, however, rare and the node survives by means of an alternative supply from the left coronary artery through the left ramus ostii cavae superiosus. This may be barely adequate at first, but becomes so as soon as the circulation improves; if hypotension is quickly corrected block may disappear at once (Fig. A), but more often a few days are needed. If the circulation fails to improve, the patient usually dies within a few days, the heart-block persisting to the end.

Pathology of the Primary Group

In our primary group not only was there no evidence of coronary disease but the findings did not suggest a hypertensive aetiology, although this has been proposed by earlier authors. Ten of our patients had normal blood-pressures, and a mild diastolic hypertension was found in only five cases. Moreover, A-V block occurs only rarely in the course of established hypertensive heart disease.

The nature of the pathological process in the primary group remains uncertain. Necropsy findings excluded syphilis, rheumatism, and ischaemic heart disease. Detailed histological examination in two cases showed not only an extensive fibrosis of the bundle of His and the bundle branches but also an increase of the interstitium throughout the ventricular muscle with microscopic areas of replacement fibrosis and, in one case, lymphocytic foci. In both, the fibrosis was thought to be due to a diffuse myocarditis which had largely subsided, leaving widespread damage. Of the known causes of myocarditis, diphtheria seemed improbable; there was no recent history of it, and such an explanation would be out of keeping with the known characteristics and time relationships of diphtheritic disorders.

Many infective, allergic, or toxic processes may, however, be associated with histological evidence of myocarditis (Saphir, 1960), and their microscopical appearance with monocytic infiltration and fibroblastic repair resemble those found in our cases. That these appearances are not rare in elderly subjects is suggested by the work of Schwartz and Mitchell (1962), who found them in 26% of 137 necropsies selected on a random basis. They presented evidence against an ischaemic origin for these lesions; noted that they became progressively commoner with age; and showed that, although more frequent in men than in women, the sex difference of 3 to 2 was significantly less than that found in ischaemic disease. The same three features were noted in our group of primary heart-block.

The cause of such a focal myocarditis is at present unknown. It may depend upon bacterial infection, the lesions resulting either from direct bacterial invasion or indirectly as a toxic process. Alternatively, inflammatory changes may be due to an autoimmune process, and the presence of lymphocytic infiltrations would support this. In any focal myocarditis the occurrence of block may be quite fortuitous, depending simply on the chance occurrence of one of the scattered lesions in the conducting system. As in rheumatism and diphtheria, only a small proportion of cases of myocarditis might develop block, but in contrast to the latter conditions scarring results in permanent damage. If, however, the process is one of autoimmunity or other sensitization, lesions would be expected to occur close to small blood-vessels, and the rich vascular supply of the conducting tissues would make them particularly vulnerable. Whatever the underlying aetiology of this focal myocarditis it seems to be the commonest cause of complete permanent heart-block in elderly subjects.

Although an example was not found in our series, one further cause of heart-block in the elderly deserves mention —that is, calcification of the aortic valve or mitral valve ring with involvement of the adjacent conducting tissues. This was described by Mönckeberg (1908), Mahaim (1931), and Yater and Cornell (1935).

Association with Ischaemic Disease

Finally, our group of primary heart-block with associated ischaemic disease remains to be considered. It might be argued that in these cases the block was due to infarction and that pain was obscured by syncope. This is unlikely for two reasons: first, syncope in heart-block is character-

istically transient, lasting no more than a minute or two, and full consciousness is quickly regained; secondly, the cardiograms showed no evidence of a full-thickness infarction, there were no abnormal Q waves, and neither was there a left bundle-branch block pattern to account for their absence, so a patchy necrosis is the most that could be presumed. As it has already been shown that a severe transmural posterior infarction with fully developed Q- and T-wave abnormalities produces only a transient A-V block it seems most unlikely that a patchy intramural infarction would lead to permanent interruption of conduction. Moreover, the striking abnormalities of the T waves in these cases are unusual in ischaemic heart disease.

We believe that this syndrome of recurrent cardiac pain at rest with these characteristic cardiograms may result from bouts of extreme slowing of the heart. This is supported by the papers of Froment *et al.* (1959) and Fowler (1962). Although the work of Starzl *et al.* (1955) suggests that in heart-block the coronary flow diminishes *pari passu* with the work of the heart so that angina should not occur, yet if there were a minor coronary narrowing the effect of this reduction in flow might be exaggerated. This, together with the arterial unsaturation which occurs during extreme bradycardia, might then result in focal ischaemia with T-wave abnormalities. This recurrent cardiac pain may be regarded as a variety of the episodic angina described by Shirley Smith and Papp (1962).

Summary

A group of 51 patients (14 women and 37 men) suffering from complete heart-block has been studied with particular reference to the aetiology. The average age of the group was 65 years.

The disorder was attributed to ischaemic heart disease only if the patient had at some time suffered from angina pectoris or cardiac infarction. In only eight patients, all of them men, was this the case, and in all but one of them the block was transient. It is thought that the block in these cases results from a transient ischaemia of the A-V node which is supplied by terminal branches of the right coronary artery and that recovery ensues through the development of an increased flow through the branches derived from the left coronary artery.

In four patients syphilis may have been the cause of the heart-block. One of them also showed aortic incompetence, but in the remaining three, aged 70, 76, and 78 respectively, the heart-block was the sole cardiac abnormality.

In three patients calcific aortic stenosis coexisted with complete heart-block and the conduction disturbance was presumably caused through involvement of the bundle of His by the calcifying process.

One patient appeared to have heart-block as an isolated congenital abnormality, and another showed it transiently in the course of an obscure infection.

The largest group consisted of 30 patients—20 men and 10 women—whose average age was 67. The block was permanent in all but four cases. Hypertension did not seem to play a causative part, and the absence of coronary disease was confirmed by necropsy in three cases. In two of them serial sections showed interruption of the conducting system by severe fibrosis. Throughout the myocardium there were scattered lesions suggestive of an extinct myocarditis. It is suggested that heart-block in the elderly is usually due to a myocarditis which may be of bacterial or allergic origin.

Finally, in a further group of four patients (three women and one man) it was noted that the clinical course of heart-block was punctuated by attacks of angina at rest without decisive evidence of infarction. As it had been found that severe posterior infarction resulted only in transient block it was thought unlikely that the patchy ischaemia was the cause of the permanent block in these cases. It seemed more likely that the block was of the primary variety but complicated by coronary atheroma. Episodes of extreme slowing may have caused the attacks of pain with cardiographic abnormalities.

ADDENDUM.—Since this study was completed the paper of J. Lenegre and P. Moreau, *Bull. Soc. méd. Hôp. Paris*, 1962, **113**, 767, has appeared. In 27 of their 37 cases of heart-block significant coronary artery disease was absent and the lesion was a fibrosis of obscure origin predominantly affecting both bundle branches in 24 cases and the bundle of His in 10 cases. The remainder of the myocardium was normal in all.

We are most grateful to Dr. R. E. B. Hudson for much helpful criticism and advice and also for his histological study of the conducting tissues in two of our cases. We have to thank the physicians of Charing Cross Hospital and Dr. R. H. Hartley for allowing us to study patients under their care.

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"Until recent days it had been true to say that the Luton and Dunstable Hospital, opened in 1939, had been the last general hospital to be built in this country for over twenty years. . . . Just before the inception of the National Health Service in 1948 work had commenced on the building of two new wards, but with the continued expansion of industry and the growth of population in Luton and Dunstable it soon became clear that further substantial developments were needed if the hospital was to keep pace with the demands upon it. . . . In 1956, when the Minister of Health included the project in the list of works to be centrally financed by the Ministry, it was agreed that the first stage should provide wards, *x*-ray department, out-patient department, and accident and emergency service, together with a new boiler-house and expansion of various supporting services. . . . The second phase of the development will follow almost immediately after the completion of the first stage, and the new out-patient department will provide a much-needed increase in consulting, examination, and treatment facilities. Later stages of development, which form part of the Minister's ten-year programme, include a new pathological laboratory, further expansion of out-patient facilities, a psychiatric unit of 120 beds with a day centre, a geriatric unit with 50 beds, communicable disease unit of 40 beds, and a further increase in the number of acute beds, together with the necessary expansion of residential accommodation and other services." ("Luton and Dunstable Hospital Redevelopment: The First Phase 1963.")

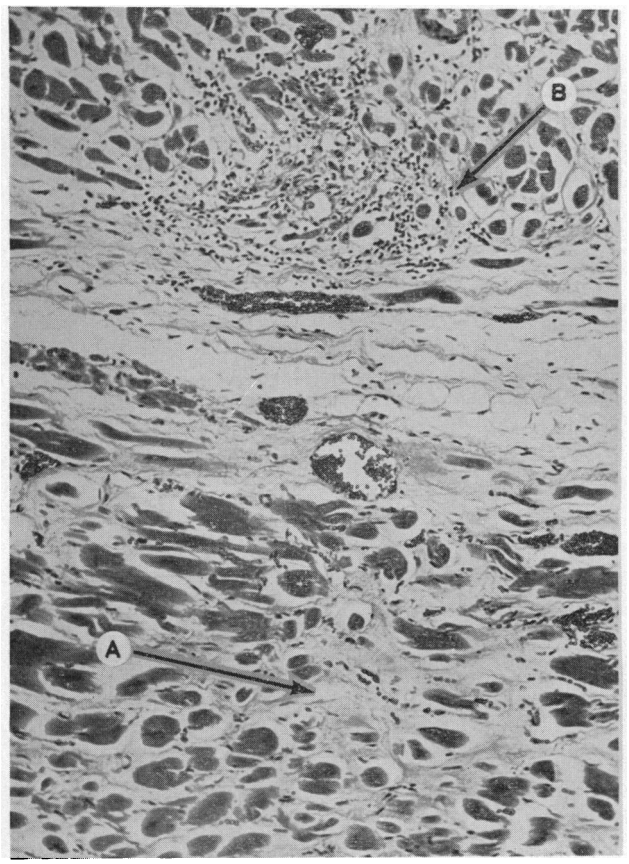

FIG. 1

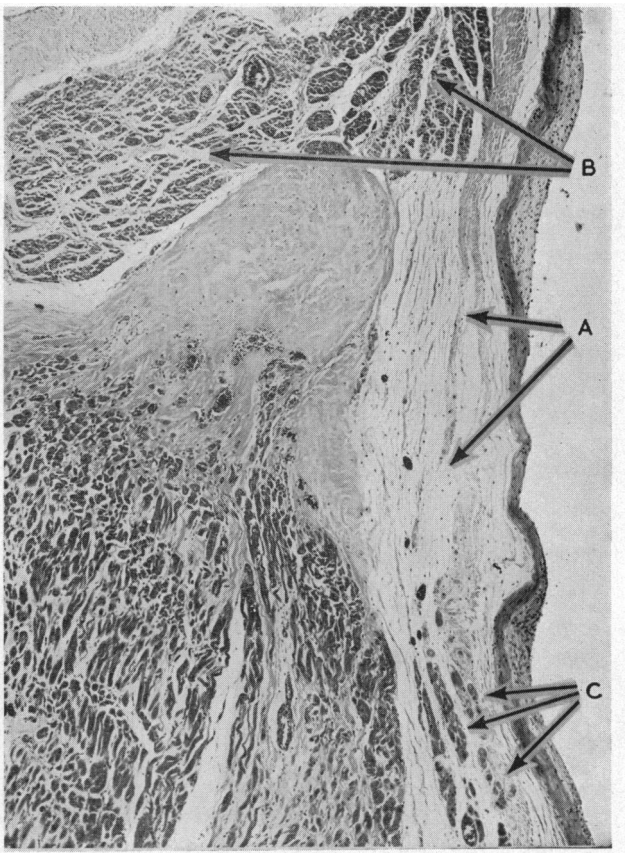

FIG. 2

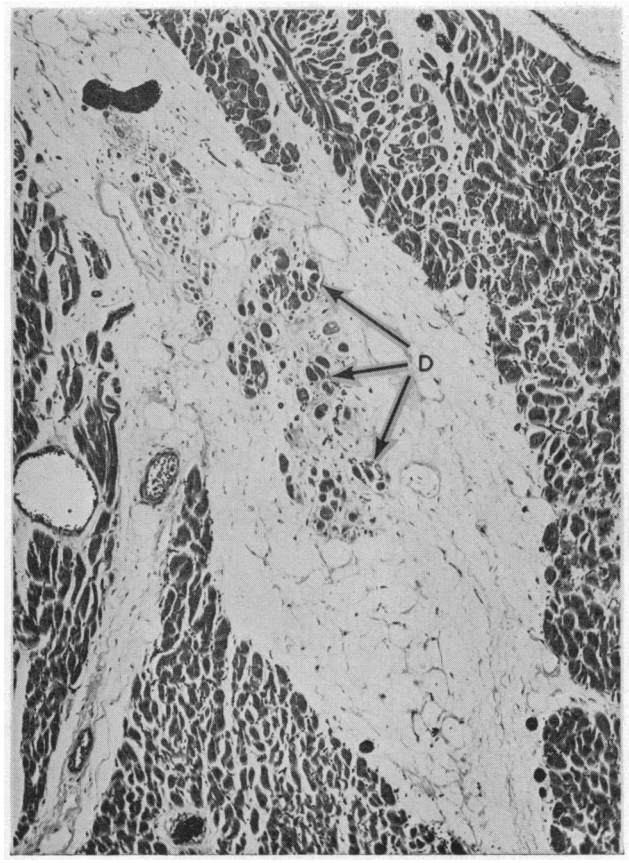

FIG. 3

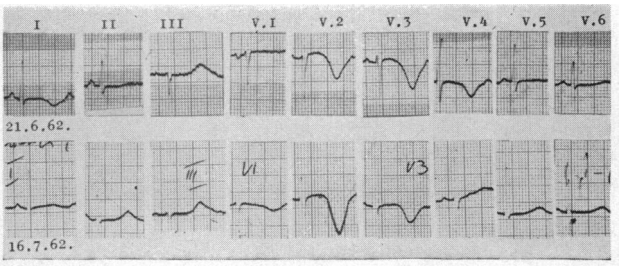

FIG. 4

FIG. 1.—Case 1. Photomicrograph of left ventricular muscle in primary complete heart-block showing an abnormally large amount of interstitial tissue (arrow A) and a cluster of lymphocytes (arrow B). The appearance suggests an extinct inflammatory process.

FIG. 2.—Case 1. Photomicrograph of conducting system. An area of fibrosis (A) separates the main bundle (B) from the left bundle-branch fascicles (C).

FIG. 3.—Case 1. Photomicrograph showing that the fibres of the right bundle branch (dark areas marked D) are scanty and interspersed with fibro-fatty tissue.

FIG. 4.—Cardiogram of a woman aged 66 who developed syncopal attacks with prolonged angina at rest. There were deep T-wave inversions but no significant Q waves. Heart-block was present; only fortuitous P waves are seen in some complexes.