ATRIOVENTRICULAR BLOCK: A CLINICAL STUDY*

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Atrioventricular block is a disorder of cardiac rhythm frequently encountered in practice. Its higher degrees, particularly complete atrioventricular block, constitute serious, and at times lethal, disorders of the cardiac mechanism. Since the advent of open heart surgery, and operations for repair of ventricular septal defects and endocardial-cushion defects, atrioventricular block induced by surgical trauma is not infrequently seen. Usually the block is transient, disappearing minutes, hours, or at times several days, after the operation.[1] Permanent atrioventricular block after surgery is rare and can usually be avoided by careful placing of sutures.[2] The patients developing atrioventricular block after operation have afforded clinicians an opportunity for detailed observation of this disorder, and much has been learned concerning its clinical and haemodynamic features and the effects of various therapeutic measures.

With the knowledge so gained, it was felt that a clearer understanding of the disorder might be achieved if a fresh study were made of cases that had not been subjected to surgical operation. As mere prolongation of atrioventricular conduction does not interfere with cardiac mechanism, this study was confined to patients with atrioventricular block of greater than grade-I degree.

Material

Thirty-two patients personally seen by the authors in private practice were studied. The age and sex distributions (Table I) were in general agreement with the data

TABLE I. AGE AND SEX DISTRIBUTION IN 32 CASES OF ATRIOVENTRICULAR BLOCK

Range: 18 - 88 years

Average: 60 years

Decades:	2nd	— 1 case		6th	— 3 cases
	3rd	— 3 cases		7th	— 7 ,,
	4th	— 0 ,,		8th	— 10 ,,
	5th	— 6 ,,		9th	— 2 ,,

Males — 19
Females — 13

in other series.[3-6] The ratio of males to females was 1·5 to 1, the same ratio as that reported by Wright *et al.*[6]

Aetiology

The aetiological factors are listed in Table II. In 10 patients the aetiology was not certainly known. Of these 10, one was a young policeman in whom both bilharzial and hydatid complement-fixation tests were positive, and in the other 9 we have presumed that coronary heart disease was the cause since no other cause was manifest. These 9 were all over 50 years old and all showed elevated systolic blood pressure, but the presumption may well be inaccurate.[10] Six other patients not only fulfilled these same criteria but in addition experienced angina pectoris or had had a previous myocardial infarction but without block at the time of the infarction.

TABLE II. AETIOLOGY OF 32 CASES OF ATRIOVENTRICULAR BLOCK

Cause	*No. of cases*	
Rheumatic fever	2	
Quinidine	1	
Unknown (? bilharzia or hydatid)	1	
Unknown (presumed coronary heart disease)	9	— 5 females 4 males
Coronary heart disease	6	— 1 female 5 males
Myocardial infarction	13	— 5 females 8 males

In the remaining 13 patients, atrioventricular block appeared at the time of the development of a myocardial infarction, or a few days afterwards. Of some interest is the fact that 5 of these 13 were females, of whom 2 were pre-menopausal (but one of the two was diabetic and the other had essential hypercholesteraemia).

In 2 patients block occurred during an attack of rheumatic fever, and in one patient quinidine intoxication was undoubtedly responsible. Digitalis intoxication was a contributory aetiological factor in one of the patients with myocardial infarction. The paucity of cases of digitalis intoxication in our series is largely explicable by the fact that we have excluded cases where atrioventricular block is associated with supraventricular arrhythmias, e.g. paroxysmal atrial tachycardia with block.

Type of Block

The types of block seen are listed in Table III. From this it may be seen that a number of patients had different degrees of block at different times. Varying degrees of block are illustrated in Figs. 1 and 2. It is im-

TABLE III. PATIENTS ACCORDING TO THEIR TYPE OF BLOCK, SHOWING ALSO THE VARIATIONS IN DEGREE AT DIFFERENT TIMES

Type	*No. of patients*	*Variations in degrees*
Sino-atrial block	4	3 with complete atrioventricular block 1 with incomplete atrioventricular block
Complete atrioventricular block	21	8 with other degrees
Incomplete (2:1 or greater) atrioventricular block	12	8 with other degrees
Wenckebach phenomenon	9	6 with other degrees

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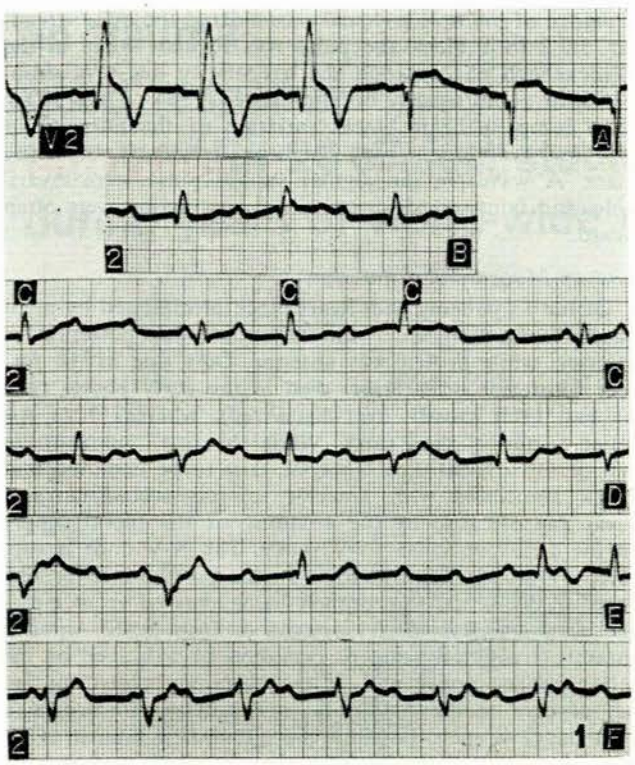

Fig. 1. Tracings from a patient with anterior myocardial infarction. A = Intermittent right bundle-branch block. B = 2:1 atrioventricular block. C = Complete heart block, with 3 possibly conducted beats (C). D = 2:1 atrioventricular block with alternating bundle-branch block. E = Complete heart block with intermittent bundle-branch block and irregular ventricular activity. F = Complete heart block with bundle-branch block. All these tracings were taken within 24 hours.

portant to note, however, that two-thirds of the patients had complete atrioventricular block at one time or another. It is of interest that 4 of the patients, at one time or another, showed sino-atrial block, i.e. sinus arrest or complete cardiac standstill (Fig. 3).

Electrocardiographic Findings

Conventional 12-lead ECGs were performed on all the patients. For analysis of the arrhythmia, standard lead 2 and lead V1 proved the most helpful. Long strips of continuous recording are necessary for correct analysis, and only by recording such long strips can it be shown occasionally that what appears superficially to be 2 to 1 block may in fact be complete block with the atrial rate nearly double the ventricular rate.

We are in agreement with Wright *et al.*[6] that in patients with acute myocardial infarction definite ECG features of the infarction are usually present, and that the presence of broad notched QRS complexes does not necessarily indicate a very low pacemaker but more commonly a coexisting bundle-branch block. Thus, the case illustrated in Fig. 1 showed intermittent bundle-branch block (Fig. 1A) before the development of atrioventricular block with apparently varying site of pacemaker (Fig. 1E). It is possible that at least some cases of apparent varying site of pacemaker (Fig. 4) may represent alternating bundle-branch block.

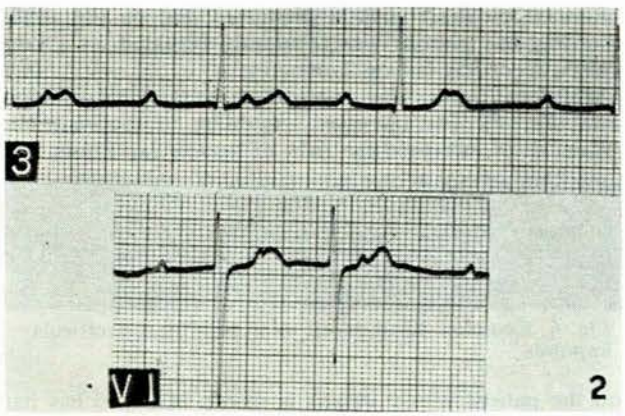

Fig. 2. Upper tracing showing 2:1 block with variable P-R interval. Lower tracing showing Wenckebach phenomenon.

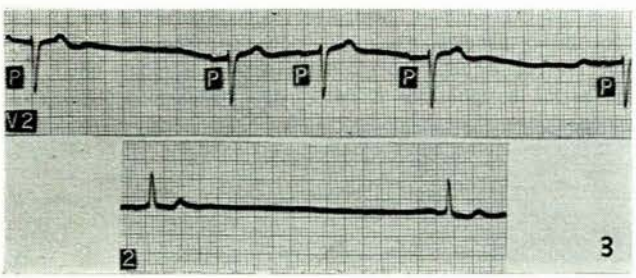

Fig. 3. Sinus arrest. Upper tracing shows intermittent grade-I atrioventricular block. This patient at other times showed 2:1 atrioventricular block.

Occasionally, heart block remained partial despite remarkably long delays in atrioventricular conduction. In Fig. 5, the top tracing shows grade-I heart block with a period of reversed reciprocal rhythm. The bottom tracing, recorded 3 months later, shows a Wenckebach phenomenon with PR intervals reaching 0·6 second. For con-

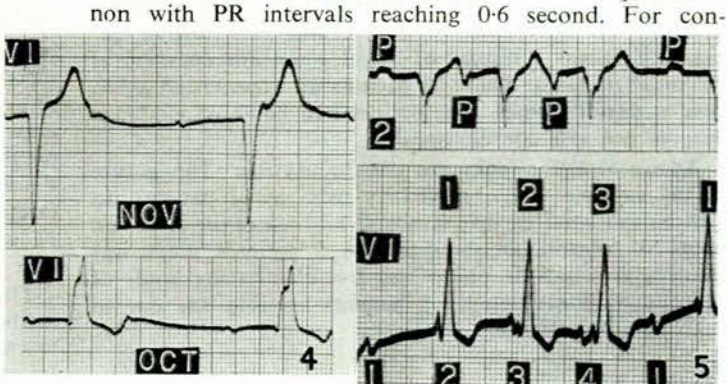

Fig. 4. Varying site of ventricular pacemaker, or possibly varying type of bundle-branch block with complete heart block. *Fig. 5.* Upper tracings showing reversed reciprocal rhythm. Lower tracing showing Wenckebach phenomenon with remarkably long P-R interval. P-waves numbered below and QRS-waves above record. The 4th P-wave is blocked.

venience, one short strip is illustrated, but on the actual recording this pattern was constantly repeated on a continuous recording lasting 2 minutes.

Multifocal ventricular pacemakers were noted in some cases (Fig. 6). The increased irritability of the ventricles in such cases increased the risks of ventricular fibrillation,

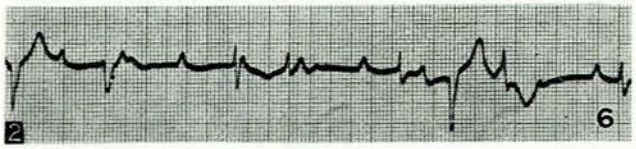

Fig. 6. Complete heart block with multifocal ventricular impulses.

and the patient whose tracing is shown in Fig. 6 has had numerous Adams-Stokes attacks due to periods of self-limited ventricular fibrillation. She is still alive, 11 years after the onset of the block.

In the presence of complete heart block, the atrial and ventricular pacemakers may discharge irregularly (Fig. 7). The reason for the ventricular irregularity is not clear, but the atrial irregularity is often due to earlier atrial con-

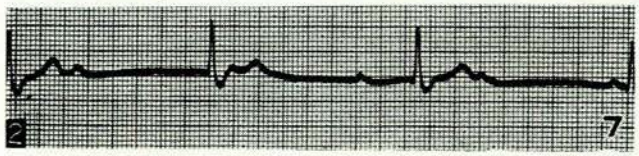

Fig. 7. Complete heart block with variation of atrial and ventricular rate.

traction immediately following a ventricular complex. It is believed that increased flow of blood to the sino-atrial node following ventricular systole may result in earlier discharge from this node. While filling of the main coronary vessels occurs predominantly in diastole, the smaller branches to the atria fill best during systole.

Symptoms

Pre-existing angina or cardiac failure were aggravated by the slow heart rate.

Symptoms referable to the atrioventricular block itself were present in 24 cases (75%). In 18 (56%) Adams-Stokes attacks with convulsions occurred, and in 5 further patients there was a history of 'fainting' on one occasion at the onset of block. These 23 patients all experienced giddiness at various times. One patient had bouts of giddiness but did not lose consciousness.

Adams-Stokes attacks are due to asystole. The nature of the asystole was observed by ECG in 11 patients. In 3 there was total cardiac standstill, and one of the three had bouts of ventricular standstill. In 5 patients there was ventricular standstill, one of the five also having bouts of ventricular fibrillation. In 3 patients asystole was due to ventricular fibrillation.

Physical Signs

The pulse was slow in all cases, usually below 40 per minute. In complete heart block the pulse was usually regular, although minor degrees of irregularity of the ventricular pacemaker could not be detected clinically. The presence of multiple pacemakers rendered the pulse irregular. In 2 to 1 block the pulse was regular, while in the Wenckebach phenomenon the irregularity due to a dropped beat could usually be appreciated.

In complete heart block, variation of the intensity of the first heart sound was frequent, irregularly occurring giant 'A'-waves in the jugular venous pulse were invariable, and faint sounds due to atrial contraction were often heard.

Site of Myocardial Infarction

Of the 13 patients with heart block precipitated by myocardial infarction, in 7 the infarction was posterior or inferior, while in 6 it was anterior. Only one of the former (posterior or inferior) died in the early phases (and he had been grossly over-digitalized), whereas 3 of the latter (anterior) died early. While the small numbers do not permit of statistical analysis, it is to be expected that block associated with anterior infarcts would carry a great risk because of the anatomy of the blood supply to the atrioventricular (A-V) node. The A-V node is supplied by an ascending branch on the postero-inferior surface of the heart, this branch being derived from either the right or left coronary arteries or from both. Anterior infarcts are usually due to occlusion of a branch of the left coronary artery and, if this were to involve cutting off of flow to the A-V node, it would require occlusion of a large branch, possibly the circumflex branch at or near its origin. Posterior infarction is due to occlusion of more distal ramifications of the circumflex coronary artery or of branches of the right coronary artery, and in either case the area of myocardium affected is likely to be less.

Of some interest is the one patient with complete heart block at the onset of an infarction in whom subsequent ECGs showed only minor features of an inferior infarct (S - T segment depressions with biphasic T-waves in a VF). It is postulated that infarction was due to occlusion of the most distal branches of the coronary artery supplying the A-V node.

Prognosis

It is not possible to prognosticate accurately in these cases. Thus, a patient may successfully tolerate numerous bouts of asystole over many years and then suddenly succumb; another patient may succumb to the first bout of asystole. Moreover, the underlying disease may be more important than the conduction defect in deciding the prognosis. In this series, there were 15 deaths. Of these 4 were early deaths, all in patients with myocardial infarction, of whom one died of Adams-Stokes attacks; and 11 were late deaths occurring between 3 months and 7 years after onset of block (average 3·6 years).

The 11 late deaths are analysed in Table IV. Four of them were due to Adams-Stokes attacks and 2 were due to cardiac failure, with block present at the time of death. In one patient the cause of death was not known. Thus, the block was directly responsible for death in 4 patients and indirectly contributory in 2 patients (and possibly a third). In 4 (possibly 5) patients, the block was not contributory to death, and in only one (possibly 2) of these was block still present at death. Excluding the 4 early

TABLE IV. ANALYSIS OF 11 LATE DEATHS

Cause of death	*Number*	*Block present at death*
Adams-Stokes attack	4	yes (block 2 - 5 years)
Congestive cardiac failure	4	2 yes 2 no
Unknown	1	yes
'Stroke'	1	yes
Ruptured aortic aneurysm	1	no

deaths, of 12 patients with permanent block 8 (66%) are dead. Of 16 patients with intermittent or temporary block 4 (25%) are dead. The presence of permanent block is thus a bad prognostic sign. However, 2 patients with permanent complete heart block, who have had frequent Adams-Stokes attacks, are alive and well 11 and 12 years respectively after the onset of block.

Management

The management of atrioventricular block has undergone radical changes in recent years, and it may be stated with confidence that we are now for the first time able to offer a reasonably effective programme of management for this condition.

If heart block is permanent and stable, without Adams-Stokes attacks, and there is no cardiac failure, treatment is probably not necessary. For the management and prevention of Adams-Stokes attacks, drugs such as atropine, ephedrine and adrenalin have generally proved disappointing, and personally we have abandoned that therapy. The use of isopropyl-noradrenalin (isoproterenol) has been reported on favourably in recent years,[7-9] but some have found it disappointing.[10] We have used this drug sublingually in this series in 22 patients and we have not been convinced of its efficacy. After initial doses, increased rate of an idioventricular pacemaker may occur, but the effect soon wears off. Moreover, multifocal ventricular beats may sometimes occur after the use of the drug (see tracings B and C in Fig. 8). Recently Linenthal and Zoll[11] have convincingly demonstrated the efficacy of intravenous isoproterenol in preventing attacks of ventricular fibrillation in patients with complete atrioventricular block, but they mention that in smaller doses ventricular excitability may be increased. This would appear to explain the findings in some of our cases. We have had no personal experience of intravenous isoproterenol.

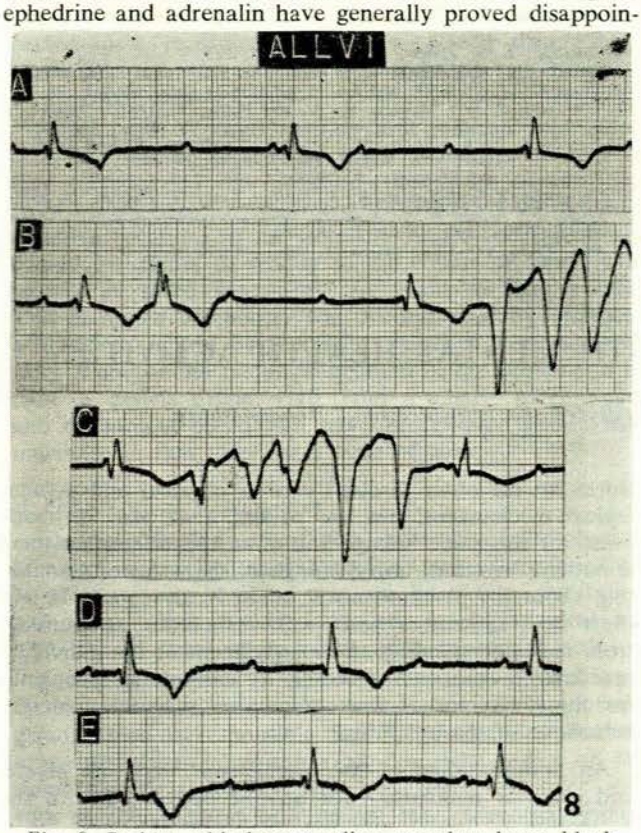

Fig. 8. Patient with long-standing complete heart block (see text). A = Resting tracing. B = Tracing 4 minutes after commencing isuprel 15 mg. sublingually. C = Tracing 4½ minutes after commencing isuprel. D = Resting tracing. E = Effect of carotid-sinus pressure.

The greatest advance in the therapy of atrioventricular block has been the introduction of electrical methods of pacemaking in the heart. With the use of internally placed electrodes, effective pacemaking may be continued for long periods with no discomfort.[1] Externally placed electrodes are effective, but the voltage needs to be so high that it produces intense discomfort. External pacemaking has been used effectively in 3 cases in this series, and has been on stand-by in 2 cases, allowing the physician a feeling of some degree of security.

More effective and less disturbing to the patient is pacemaking by means of an internal electrode introduced at the tip of a cardiac catheter, although the method is not without its hazards.[10] It may successfully tide the patient over an emergency. For 'permanent' pacemaking, internally placed electrodes are essential, and numerous battery-operated transistorized units are now commercially available for such cases. The power unit can be embedded in the rectus sheath, and may operate for several years off the same batteries. The establishment of a heart-block management unit at each large centre is probably desirable. Such a unit could easily apply these effective and life-saving measures.

In 1954 Prinzmetal and Kennamer[12] described the successful use of corticotrophin in a case of complete heart block following myocardial infarction, and stated that the rationale of the treatment was that the conducting tissues may not have been destroyed by infarction, but may be involved in inflammatory reaction at the edges of the infarct. Several reports have since appeared on the efficacy of corticosteroids in atrioventricular block, whether following myocardial infarction or not.[13-20] In addition to its anti-inflammatory effect, Lown *et al.*[21] state that the corticosteroid may have a facilitating effect on atrioventricular conduction,[21] while Friedberg *et al.*[18] mention an 'arousal of the ventricular pacemaker', resulting in abolition of Adams-Stokes attacks despite continued heart block.

In our series, of the 22 patients in whom atrioventricular block was believed to be due to coronary heart disease (including those patients with myocardial infarction) 4 died early. Of the remaining 18, 7 received steroids and in 5 (71%) the block proved to be temporary, while of the 11 who did not receive steroids in only 3 was the block temporary (27%). Of the 10 patients with perma-

nent block, 2 received steroids and are alive and well; while 8 did not receive steroids and 6 of them have died, 4 of Adams-Stokes attacks. In this series, the steroids were largely given by mouth, whereas Dall and Buchanan[19] stated that they were much more efficient if given intravenously.

It is to be stressed that in our series there is no true control, and it is possible that chance may be responsible for the apparent efficacy of steroids. Nevertheless, the reports in the literature lend considerable support to our belief in the efficacy of this therapy, although Zoll and Linenthal[22] have not been able to confirm it.

Apart from 'specific' therapy, certain general measures may be of value. As emotional factors may be responsible for precipitating Adams-Stokes attacks, sedation may be necessary. Digitalis may certainly be used, if indicated for cardiac failure, provided the block is not due to digitalis. Carotid-sinus pressure is best avoided, in view of the danger in complete block of depressing the ventricular pacemaker, or increasing the degree of block when it is incomplete. However, paradoxical acceleration of a ventricular pacemaker may be produced by carotid-sinus pressure (see tracing E in Fig. 8). The reason for this is not known.

In patients in whom the blood pressure is low, its elevation with pressor agents may facilitate conversion to sinus rhythm, probably by increasing coronary flow. We have not used chlorothiazide, or molar sodium lactate. The former has been reported as being ineffective,[22] while the latter may precipitate ventricular fibrillation.

Finally, we feel it important to stress the grave dangers inherent in the use of quinidine and procaine-amide in patients with heart block. These drugs may produce cardiac standstill and are contraindicated, even in the presence of multiple ventricular ectopic beats, which are better treated by producing a more rapid idioventricular rhythm with isoproterenol[11] or a pacemaker.[10]

SUMMARY

The condition of atrioventricular block has been studied in a series of 32 patients. The aetiology, ECG findings, symptoms, signs and prognosis are discussed. The site of myocardial infarction associated with block may be important in the prognosis, which may be worse in anterior myocardial infarctions. A possible anatomical explanation for this is discussed. The management of atrioventricular block is discussed and the value of two methods, viz. electrical pacemaking and the use of corticosteroids, is stressed. Mention is made of the dangers of quinidine and procaine-amide.

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