Complete heart block

Studies of atrial and ventricular pacemaker site and function

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Atrial and ventricular pacemaker function was studied in 20 patients with idiopathic chronic complete heart block using the rate response to an intravenous bolus dose of isoprenaline (5 µg/70 kg bodyweight). Pacemaker responses were compared with those of 16 normal control subjects. None of the patients was having syncopal attacks at the time of admission and they were therefore selected in that none required immediate pacing.

Ten of the patients had His bundle electrograms; all were shown to have a pre-His type of atrioventricular block.

Two major groups emerge from the responses to isoprenaline.

(a) High risk group: 11 of the 14 patients with reduced ventricular pacemaker responses had frequent syncopal attacks; 8 of the patients with Adams-Stokes syncope had a bundle-branch block pattern, while 3 had a narrow QRS. These patients require pacing. (b) Low risk group: a low risk asymptomatic group (5 patients) was identified with atrial and ventricular responses to isoprenaline within normal range. One of these patients had a bundle-branch block pattern, while 4 had a narrow QRS. These patients might be managed without pacing.

The atrial response to isoprenaline was reduced in 12 of the 20 cases, 10 of whom also had reduced ventricular responses. All 9 patients with bundle-branch block had reduced ventricular responses, while 7 had reduced atrial responses. This evidence indicates that cardiac conducting tissue pathophysiology is widespread in complete heart block.

The present work suggests that consideration of the ventricular pacemaker function is important in assessing liability to syncope in complete heart block.

While patients with Adams-Stokes attacks require pacing it is suggested that all asymptomatic patients with complete heart block and those with minor symptoms are assessed using studies of both ventricular pacemaker function and site. A low risk group not requiring a pacemaker may emerge after sufficient follow-up assessment.

Most patients with chronic complete heart block present with sudden syncope. There are some, however, who remain in complete heart block for many years without significant symptoms. The objects of the present work have been to investigate both atrial and ventricular pacemaker function in chronic complete heart block, to relate these findings to symptoms, and if possible to characterize groups with either a high or a low risk of syncope.

When a symptomatic adult presents with complete

heart block the aetiology is often unknown. If the patient has had no major symptoms liability to syncope in the individual case will also be unknown. Though it is usually assumed that the risk of syncope in every case of complete heart block is such that prophylactic if not therapeutic pacing is generally recommended, little work has been done on the assessment of individual risks.

The QRS width and ventricular rate in congenital complete heart block are used by some as important prognostic factors; a fast rate and a narrow QRS indicating a good prognosis, a slow rate and a wide QRS indicating a poor symptomatic

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prognosis (Ayers, Boineau, and Spach, 1966; Molthan *et al.*, 1962). More fundamental factors probably exist. The present investigation is based on the hypothesis that syncope occurs in patients who fail to maintain their cerebral blood flow because their ventricular rate is governed by a pacemaking site which at times fails to maintain an adequate rate or may be subject to total failure (asystole). Ventricular escape rhythms may occur as a consequence of a slow ventricular rate. A relation has been shown to exist between syncope and the presence of reduced atrial pacemaker function in sinus bradycardia (Mandel *et al.*, 1972; Dighton, 1974) and sinoatrial block (Dighton, 1975). It is postulated that an analogous situation exists in complete heart block, with syncope occurring in patients with reduced ventricular pacemaker function. In this study, atrial and ventricular pacemaker function in complete heart block has been assessed from the rate response to an intravenous bolus dose of isoprenaline (Dighton, 1974).

A further factor of fundamental importance to the occurrence of syncope in complete heart block is the site of origin of the ventricular pacemaker (Rosen *et al.*, 1971, 1972). From animal electrophysiological experiments it may be assumed that the more distal the site of the ventricular pacemaker, the slower will be the intrinsic rate, and the less will be the response to sympathetic drive (Hoffman and Cranefield, 1960). The likelihood of asystole, pacemaker failure, and syncope may, therefore, be greatest in complete heart block when the ventricular pacemaker is distal in site. In this study an attempt has been made using His bundle electrograms to define the site of the ventricular pacemaker.

Patients and methods

Twenty patients with chronic complete heart block were investigated after obtaining fully informed consent. There were 11 men and 9 women of mean age 60 years.

Sixteen volunteer control subjects without any evidence of cardiac disease, mean age 52 years, were investigated using the same test of pacemaker function. The responses of these subjects were used as controls for the atrial pacemaker responses in complete heart block. While these responses cannot strictly be used as controls for ventricular pacemaker responses, they have been used in the absence of any more suitable control data. The possibility of increased sympathetic activity in complete heart block might also be taken to detract from the suitability of the present control group (Finucane and Gialafos, 1974). However, the present work fails to support this view. His bundle electrograms were not performed on the control subjects since sufficient normal data have now been published.

All subjects and patients were investigated using a single intravenous bolus dose of 5 µg isoprenaline per 70 kg bodyweight, the injection being delivered into an

arm vein. A continuous electrocardiographic recording was started 15 seconds before injection and continued during and for one minute after injection. The records were analysed using a standard rate ruler averaging 2 consecutive RR intervals for ventricular rate and 2 consecutive PP intervals for atrial rate. Five estimates of atrial and ventricular rate were made before injection and taken as the control resting rates. Further estimates were made at 3-second intervals after injection and continued for 2 minutes. A pilot-study of reproducibility using repeat infusions of isoprenaline had shown these were not necessary in the main study.

No patient had been paced at the time of investigation, and all drugs had been withdrawn for at least 48 hours. In no patient was the aetiology of the complete heart block known with certainty. In 10 of the 20 patients His bundle electrograms were obtained, a bipolar catheter being introduced either percutaneously through the femoral vein or alternatively through the antecubital vein and positioned across the tricuspid valve. From the records obtained the level of atrioventricular block could be ascertained. With a His deflection seen to follow each P wave, a diagnosis of post-His complete heart block can be made. When the His deflection is seen to precede each QRS a diagnosis of atrioventricular block at a pre-His level can be made. The His to ventricular activation time was measured in each case.

Results

The resting atrial and ventricular rates together with QRS configurations of all subjects are shown in Table 1. No significant difference was found between the resting atrial rates of either control or complete heart block subjects.

In Table 2 clinical features are correlated with the results of this investigation.

TABLE 1 *Clinical and electrocardiographic features of control subjects and patients with chronic complete atrioventricular block*

	Controls	Complete heart block
Total number	16	20
Mean age (yr)	52	60
Men	8	11
Women	8	9
Mean resting rates:		
Atrial	74.5/min	79/min (63-100)
Ventricular	—	40/min (29-54)
Electrocardiographic features:		
Narrow QRS	16	11
Right bundle-branch block	0	5
Left bundle-branch block	0	2
Alternating right and left bundle-branch block	0	1
Intermittent left bundle-branch block	0	1

TABLE 2 Analysis of responses to isoprenaline and associated clinical features of 20 patients with complete heart block

	Adams-Stokes syncope	Vasomotor syncope	Reduced atrial responses	Reduced ventricular responses	Bundle- branch block	Narrow QRS
Total no. with various clinical features	12	3	11	14	9	11
Reduced ventricular responses (N=14)	11	0	10	14	9	5
Reduced atrial responses (N=12)	9	1	12	10	7	4
Normal ventricular responses (N=6)	1	3	1	0	0	6
Normal atrial responses (N=8)	3	2	0	3	1	7

a) Atrial responses (Fig.)

The control subjects increased their atrial rates above resting rate by a mean of 30.5 beats per minute, with 5 µg isoprenaline (per 70 kg body-weight). The responses ranged from an increase of 22 to 39 beats per minute.

In patients with complete heart block the range of increase in atrial rate was from 9 to 40 beats per minute (mean 18.8 beats per minute). The mean increase in atrial rate of those with complete heart block was found to be significantly less than in control subjects ($P < 0.01$) (t-test).

b) Ventricular responses (Fig.)

With 5 µg isoprenaline (per 70 kg bodyweight i.v.) the increase in ventricular rate of those in complete heart block ranged between 1 and 42 beats per minute above control rate (mean response being 12.2 beats per minute). The increase of ventricular rate in complete heart block was found to be significantly less ($P < 0.01$) than control atrial responses.

c) His Bundle electrograms

His bundle electrograms were performed on 10 patients, all of whom had a history of syncope. Despite the presence of bundle-branch block in 9, there was no evidence of any abnormal prolongation of the His to ventricular activation time in any case (HV: 40–50 ms in all cases). In each case the QRS complex was preceded by a His deflection indicating block proximal to the site from which the His deflection was recorded.

Clinical features

Analysis of the 14 patients with ventricular responses below the control (atrial) range revealed that 11 patients were subject to Adams–Stokes syncope (high risk group), 9 had a bundle-branch block pattern, while 5 had a narrow QRS. Of the 14 patients, 10 also had reduced atrial responses, while 3 had normal atrial responses.

Of the 12 patients with Adams–Stokes attacks, 8

had a bundle-branch block pattern on electrocardiogram, 4 had a narrow QRS; 11 of the 12 had reduced ventricular responses. Of the 9 patients with a bundle-branch block pattern, 8 had Adams–Stokes attacks and all 9 had reduced ventricular rate responses. Of the 14 that had reduced ventricular responses, 11 had had Adams–Stokes attacks.

Six patients in complete heart block had ventricular responses within control atrial response range. All 6 had a narrow QRS complex, 1 had a history of Adams–Stokes syncope, while 3 were subject to what was taken to be vasomotor syncope on historical evidence. Only 1 of these 6 had a subnormal atrial response to isoprenaline.

Of the 11 cases with a narrow QRS complex, 5

FIG. Increase in rate above resting rate with isoprenaline 5 µg per 70 kg bodyweight.

had 'subnormal' ventricular responses and a liability to Adams-Stokes syncope (high risk group). Of the remaining 6, 5 had normal ventricular responses and no liability to Adams-Stokes syncope (low risk group). The remaining patient had normal ventricular responses and syncopal attacks which may have been a vasomotor type on historical evidence.

Discussion

In interpreting the results of this work, attention must be drawn to the fact that none of the patients required urgent pacing. No patient included in this series had frequent syncope requiring urgent pacing. In all cases the prevailing rhythm was seen to be stable, giving no cause for concern clinically, and making time available for the performance of the investigations presented.

This method of selection probably accounts for the unusually high incidence in complete heart block of 11 cases out of 20 with a narrow QRS complex. Patients having frequent syncope and requiring urgent pacing are not represented in this series. This may account for the fact that 10 of the 15 with a history of syncope had a ventricular pacemaker sited proximal to the bundle of His.

It has been suggested that sympathetic drive predominates in complete heart block (Finucane and Gialafos, 1974). This was concluded after finding a drop in ventricular rate in response to an infusion of atropine and propranolol together. Their results probably indicate that there is sympathetic but no vagal influence in the His-Purkinje system. One cannot conclude from their work that there is a greater than normal sympathetic drive in the presence of a lesser vagal influence in complete heart block. It is unlikely, therefore, that responses to isoprenaline are reduced in some cases of complete heart block just because of excess sympathetic drive.

Results of the present work help to define a high risk group and indicate a definite association between Adams-Stokes attacks, a bundle-branch block pattern, and the inability of both the ventricular and atrial pacemakers in complete heart block, to respond to a small standard intravenous bolus dose of isoprenaline. In the group studied there was no helpful relation between resting ventricular rate and symptoms. Of the 12 patients having experienced Adams-Stokes attacks, 11 had a reduced ventricular response to this test. Three further patients with reduced ventricular responses were asymptomatic. On this evidence sudden syncope seems to be commonly related to the inability of the ventricular pacemaker to speed sufficiently on

demand. It may be that the lack of response is associated with unreliability, the dominant pacemaker at times producing subthreshold, non-conducted impulses. Should this occur, a long asystolic period might occur before an idioventricular distal pacemaker took over; alternatively the period might be sufficiently long to allow an escape rhythm to supervene. Though speculative, these considerations are supported by the direct observations made by Parkinson, Papp, and Evans (1941).

The 3 patients thought to have vasomotor syncope had a proven proximal type of block and normal ventricular responses. In these cases the presence of effective autonomic influences was demonstrated by 'normal' ventricular rate changes in response to both carotid sinus pressure and a Valsalva manoeuvre (Dighton, 1974). While not excluding the possibility of other mechanisms of syncope, this evidence makes vagal suppression a possibility. Clinical evidence for a vagal mechanism is suggested in each case by syncope of gradual onset, associated with pallor and a feeling of subjective temperature change. Circumstances in each case had been conducive to 'fainting'. The rhythm associated with syncope was not known in any case.

Twelve patients had both reduced atrial and ventricular responses in comparison with controls. In addition all 9 patients with bundle-branch block had reduced ventricular responses, while 7 of them had reduced atrial responses. This evidence suggests that cardiac pacemaker and conducting tissue pathophysiology is extensive in complete heart block. The commonest cause of atrioventricular conducting tissue pathology is known to be idiopathic fibrosis, a pathological process which may be widespread, affecting the sinoatrial node and atrium, the atrioventricular node and upper His bundle in addition to the bundle-branches (Davies, 1971; Rossi, 1969).

His bundle electrograms revealed a proximally arising pacemaker in all tested. Those with a proximally arising pacemaker and normal ventricular function probably have their pacemaker sited in the atrioventricular node since it is likely that no site distal to the atrioventricular node is capable of responding in a manner comparable to the normal sinoatrial node (Hoffman and Cranefield, 1960). Those with a proximal pacemaker with reduced responses might have their pacemaker sited either in an atrioventricular node affected by a pathological process or in the His bundle. The latter statement is based on the assumption that the normal atrioventricular node is as capable of responding to isoprenaline as the sinoatrial node though starting from a slower rate. The assumption is supported by

the finding here of 'normal' ventricular responses in 6 patients, all of whom had a narrow QRS complex.

There were 4 patients who had atrial and ventricular responses that differed. Three of them had normal atrial responses together with reduced ventricular responses showing a possible ventricular conducting tissue abnormality in the presence of apparently normal atrial pacemaker function. The fourth patient had a normal ventricular response with a subnormal atrial response. This result suggests sinoatrial disease associated with a normal atrioventricular pacemaker, perhaps caused by acquired atrial pathology in association with congenital atrioventricular block (on historical evidence). So far this patient has not been paced.

Some patients (the minority) have both normal atrial and ventricular pacemaker responses to isoprenaline and are asymptomatic, having presumably accommodated their cardiovascular system to a slow rate. These patients seem to have a low risk of syncope and may not require pacing. Close observation and perhaps long-acting or sublingual isoprenaline therapy may be all that is required. Repeated estimation of their ventricular pacemaking function is simple and seems advisable.

It is suggested that all asymptomatic patients, together with those that have minor symptoms and chronic complete heart block, should be assessed by both intracardiac His bundle electrograms, together with ventricular pacemaking function studies as described above. If both atrial and ventricular pacemaking function are found to be normal, a case can be made for treating the patient conservatively unless Adams-Stokes attacks supervene. Should the ventricular pacemaking function be found defective the case for pacing is supported. Many more cases and longer follow-up will be required before an electrophysiologically based policy statement about the treatment of individual cases of complete heart block can be made. Nevertheless results of the

present work suggest the possibility of there being a small group of patients with chronic complete heart block that may not require a pacemaker.

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