CARDIAC PACING AFTER MYOCARDIAL INFARCTION IN THE PRESENCE OF BUNDLE BRANCH BLOCK

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Bundle branch block (BBB) accompanying acute myocardial infarction carries a high mortality. Infarction is usually extensive and death is commonly due to cardiac failure and shock, but in a significant number of patients sudden ventricular standstill due to complete heart block (CHB) is the mode of death. While little can be done to alter the size of the myocardial infarction, prophylactic pacing might be expected to reduce the death rate from asystole.

Following reports of the use of pacemakers in such patients with myocardial infarction complicated by BBB, we adopted a policy of pacing such patients in early 1969. Preliminary analysis of our results in mid-1970 led us to abandon pacing patients with unifascicular BBB, by which is meant uncomplicated left bundle branch block (LBBB) or right bundle branch block (RBBB) without associated hemiblock or evidence of first or second degree A–V block, while we continued to pace bifascicular cases.

The patients reported in this study were seen between July 1968 and March 1972. Sixty of them were in the coronary care unit for at least some of their hospital stay under the care of one of us (BMK). The remaining 28 patients were in the general medical wards, unmonitored, and were either referred for cardiology consultation or were detected at review of all in-patient electrocardiographic diagnosis at weekly intervals. This report embraces 90 instances of BBB accompanying the acute phase of myocardial infarction in 88 patients, of whom 15 were women. The average age of the patients was 60.8 years. Fig. 1 shows the distribution of cases seen. Conventional criteria for the diagnosis of LBBB and RBBB were used and these were deemed unifascicular blocks. It is conceded that LBBB may be the result of interruption of both anterior and posterior divisions (post-divisional), and is in effect therefore a bifascicular block. Once established, however, pre- and post-divisional LBBB are indistinguishable electrocardiographically and in all cases were arbitrarily regarded as pre-divisional and consequently unifascicular in type. LBBB was observed in nine patients and RBBB in 13 patients. LBBB or RBBB, in association with first degree, or Mobitz II second degree block was observed in 13 patients in whom five had a LBBB pattern and eight a RBBB pattern. All 13 patients were classified as bifascicular block. Without His bundle electrograms, however, it cannot be stated categorically that the first or second degree block was due to conduction disturbance in the contralateral bundle[1].

RBBB with left anterior hemiblock (LAH) and RBBB with left posterior hemiblock (LPH) were seen in 28 and 20 patients respectively. Masquerading BBB (LBBB pattern in limb leads and RBBB in precordial leads) was seen in one instance. A further five patients had QRS complexes $\geqslant$ 0.12 seconds with pathological left or right axis deviation, some with features of incomplete or complete LBBB. In several, the QRS widening may best have been attributed to intra-infarction block in the presence of LAH or LPH, but a certain diagnosis was not possible. These were called "indeterminate" BBB and were classified as bifascicular in type because it was believed that they most often represent incomplete post-divisional conduction disturbances in both fascicles of the left bundle. A single patient was classified as undetermined BBB as he developed marked QRS widening plus second degree A–V block on the monitor and succumbed in asystole before a 12-lead ECG could be done.

A transvenous temporary pacemaker was inserted prophylactically in 39 instances. The relatively infrequent use of prophylactic pacing is partly because the 1968 patients in this group were seen prior to the change in policy of management regarding pacing in BBB, partly because we abandoned pacing patients with unifascicular BBB in mid-1970 once it became apparent that they showed no tendency to develop CHB and finally, because some patients included in this study were not referred for opinion, and were therefore not paced despite the presence of bifascicular BBB. In order to compare the relative risk of the patients in the group who were paced prophylactically with those who were not, the Norris Prognostic Index[2] was calculated for each group. There was no significant difference between the paced group with an average Index of 9.03 (range 3.52 – 19.02) units, and the non-paced group with an average Index of 10.00 (range 3.58 – 19.10) units.

The overall hospital mortality of the 88 patients was 50%. Twenty-four patients died in shock and/or severe low output cardiac failure with or without secondary ventricular fibrillation, two died of cardiac rupture, while one patient died in low output cardiac failure aggravated by ventricular septal rupture. Three patients died of primary ventricular flutter-fibrillation, two of these patients succumbing outside the Coronary Care Unit. Five patients died from ventricular asystole and a further ten died suddenly outside the Coronary Care Unit, seven of them without demand pacemakers in situ.

Twenty-three of the 90 instances of infarction were accompanied by the development of CHB, with or without abrupt ventricular asystole. All 23 of these instances occurred among the 68 patients with antecedent bifascicular block. Ten of the 22 patients with unifascicular BBB died (45% mortality) while 35 of 68 patients with bifascicular BBB died (51% mortality). This suggests that complete heart block, which was seen only among the patients with bifascicular BBB, did not significantly increase the mortality of the group, or that it was balanced by an equally serious complication peculiar to the patients with unifascicular BBB. This is not supported by the analysis which follows, and the similar mortality in the two groups, may to some extent have been influenced by the intervention of pacing in the patients with bifascicular BBB.

Of 45 patients with bifascicular BBB who did not develop CHB (mean Norris Index = 9.95 units) 20 died, whereas of the 23 patients who developed CHB (mean Norris Index = 9.97 units) 15 died. Despite almost identical initial prognosis therefore, and despite the use of prophylactic pacing in many patients, the group which developed CHB fared worse (65% versus 44% mortality).

Apart from antecedent bifascicular BBB, are there any other factors which can help predict which patients run the risk of developing CHB? The highest rate of progression to CHB occurred in those patients with RBBB and LPH, (Table I) of whom 10 out of 20 went on to CHB. Three of eight patients with RBBB plus first or second A–V block developed CHB and seven of 28 patients with RBBB and LAH developed CHB. None of the five patients with LBBB and first or second degree A–V block and one of the five patients with "indeterminate" BBB developed CHB. The single patient with masquerading BBB as well as the single

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TABLE I

Conduction Disturbance	No. of Instances	CHB	Died
LBBB	9	0	5
RBBB	13	0	5
LBBB + 1st or 2nd° AV block	5	0	1
RBBB + 1st or 2nd° AV block	8	3	4
RBBB + LAH	22	6	10
RBBB + LAH + 1st or 2nd° AV block	6	1	3
RBBB + LPH	15	8	9
RBBB + LPH + 1st or 2nd° AV block	5	2	2
'MASQUERADING' BBB	1	1	1
'INDETERMINATE' BBB	1	0	1
'INDETERMINATE' BBB + 1st or 2nd° AV block	4	1	3
UNDETERMINED BBB	1	1	1
TOTAL	90	23	45

patient with undetermined BBB plus second degree A–V block developed CHB.

Does prior myocardial infarction increase the risk of developing CHB? Of the 68 instances of bifascicular BBB 20 had had no previous infarction and ten of these developed CHB (50%); 39 had had a previous infarction and nine of these developed CHB (23%); while in the remainder there was uncertainty about previous infarction. From this evidence it would appear that patients who have had previous infarction have a lower risk of developing CHB, although this would appear contrary to expectations.

Does the site of the infarct in any way influence the development of CHB? The most common site of infarction among our 68 patients with bifascicular BBB was anterior. Among 48 such patients 16 (33%) developed CHB; of the eight patients with inferior infarction, two developed CHB (25%); and of four patients with anterior plus inferior infarction two developed CHB (50%). In the remaining eight patients the site of the infarct was not determined, with certainty.

Does pre-existing BBB increase the risk of CHB? Of the 68 instances of bifascicular BBB, 38 had no preceding BBB and 17 of these developed CHB (45%); nine had previous BBB and two of these developed CHB (22%); the pattern of conduction prior to infarction in the remainder was unknown. Allowing for the small number of patients under comparison, it would seem that CHB is likelier when the BBB occurs as a result of the fresh infarction than when it antedates the infarction.

Does the time of onset of BBB following infarction influence the occurrence of CHB? No patient developed BBB later than 72 hours after infarction, and the time of onset of the BBB did not correlate with the development of CHB, which occurred between 4 and 108 hours after infarction, with an average time of 37 hours. It would appear that there is no more accurate way of anticipating the development of CHB than the mere presence of bifascicular BBB plus myocardial infarction of less than 72 hours' duration.

If prophylactic pacing is to be of advantage it should carry no inherent risk and should reduce the mortality of those patients developing CHB. In the 39 cases where pacing was employed, the procedure was complicated by life-threatening arrhythmias in four instances, all of which were readily controlled without ill-effect.

Of 23 patients who developed CHB, 17 were paced and nine died, whereas the remaining 6 patients who were not paced all died. The cause of death among the 6 patients was documented as complete heart block and asystole in five. It should be pointed out, however, that four of these five patients were in severe pump failure at the time of death and would probably not have survived even with the pacemaker. The remaining patient who died unpaced, however, was doing well at the time of arrest, and although he was resuscitated, he died several days later without regaining consciousness from anoxic cerebral damage.

Among the unmonitored deaths in patients with bifascicular BBB, seven died without a prophylactic pacemaker. Five of the seven were apparently doing well at the time of the sudden, unheralded demise, and it seems possible that some of these may have died from preventable asytole. Of the 21 patients with bifascicular BBB who were paced and survived, eight were shown to develop complete heart block. Five of these eight patients developed sudden asystole, and it seems likely that their survival may have been attributed to by the use of a pacemaker.

This study differs from previous similar reports in the following respects. Firstly, we had an unusually large number of patients with a combination of RBBB plus LPH. This has previously been regarded as one of the less common conduction problems in myocardial infarction and the reason for our twenty patients in a series of 90 consecutive cases, is not explained. Secondly, prophylactic pacemaker insertion was not accompanied by any serious hazard to the patient and transient arrhythmias in four patients were readily controlled, in contrast to reports of other authors[3].

Finally, none of our patients with unifascicular BBB developed CHB. Table II compares our cases of unifascicular and bifascicular BBB with those of other series. Many series have been excluded from the grouped analysis because of their failure to differentiate the entity of RBBB plus LPH as differing from uncomplicated RBBB; or due to failure to separate patients with RBBB or LBBB alone from those with associated first or second degree A–V block. Despite the exclusion of these studies from the analysis, it will be seen that other authors have in fact found that CHB complicates a significant number of patients with unifascicular BBB.

The explanation of this may partly lie in failure of some workers to observe the progression of unifascicular BBB to bifascicular BBB prior to the onset of CHB. Of our 68 bifascicular BBB patients, 52 presented with bifascicular BBB at the time of their conduction disturbance first being noted, and of these 21 developed CHB. Sixteen patients, however, presented with unifascicular BBB initially, prior to going on to bifascicular BBB and of these two patients developed CHB as would a case of manifest bifascicular classified by other workers as unifascicular BBB progressed unnoticed to bifascicular BBB before developing CHB. We agree, however, that some patients with apparently uncomplicated LBBB in the presence of a normal P–R interval, can have delay in the contralateral right bundle, as demonstrated by His bundle electrograms[4], and are therefore, potentially at risk of CHB as would a case of manifest bifascicular BBB have been. In addition, it seems only rational that a patient with pre-existent LBBB or RBBB, whatever its aetiology, could develop acute myocardial infarction complicated by CHB at A–V nodal level, yet such patients would usually be seen to pass through a phase of first or second degree A–V block prior to the development of CHB and would therefore, fall into the category of bifascicular BBB prior to the development of CHB, although they are in actual fact not truly bifascicular in such cases.

Finally we have compared our mortality rate and incidence of CHB and bifascicular BBB with the combined results of other acceptable series (Fig. 2); it will be seen that there is a fair agreement in the high mortality and risk of development of CHB in this condition. We believe, therefore, because of the risk of

TABLE II

	LBBB			RBBB			BIFASCICULAR BBB		
	No.	Mortality	CHB	No.	Mortality	CHB	No.	Mortality	CHB
Present Authors	9	5/9 (56%)	0/9	13	5/13 (38%)	0/13	68	35/68 (51%)	23/68 (34%)
Combined Other Series	36	14/21 (67%)	9/29 (31%)	9	6/9 (67%)	3/3	329	95/170 (56%)	119/283 (42%)

CHB which contributes to some extent to the mortality of these high risk patients, and the safety of temporary transvenous pacing, that patients with a combination of bifascicular BBB and acute myocardial infarction of less than 72 hours' duration should have a prophylactic temporary pacemaker inserted as soon as the conduction disturbance is detected. Patients with unifascicular BBB may in rare instances develop CHB although this has not been seen in our series of patients and we therefore, do not consider the risk sufficient to justify prophylactic pacing, in such patients.

REFERENCES

1. Rosen, K. M., Rahimtoola, S. H., Chuquimia, R., Loeb, H. S. and Gunnar, R. M.: "Electrophysiological significance of first degree atrioventricular block with intraventricular conduction disturbance." Circulation, *XLIII*, 491, 1971.
2. Norris, R. M., Brandt, P. W. T., Caughey, D. E., Lee, A. J. and Scott, P. J.: "A new coronary prognostic index." Lancet, *I*, 274, 1969.
3. Godman, M. J., Lassers, B. W. and Julian, D. G.: "Complete bundle branch block complicating acute myocardial infarction." New Engl. J. Med., *282*, 237, 1970.
4. Ranganathan, N., Dhurandhar, R., Phillips, J. H. and Wigle, E. D.: "His bundle electrogram in bundle branch block." Circulation, *XLV*, 282, 1972.