HEART BLOCK IN MYOCARDIAL INFARCTION: A HISTOPATHOLOGICAL STUDY

by

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THE CASE with apoplectic attacks described by Robert Adams (1827) and also one of the cases reported by Stokes (1846), had yellow discolouration and friability of the left ventricular wall, almost certainly had organising myocardial infarcts. Recently Davies et al (1967) studied the histology of the conducting tissue in fourteen cases with complete heart block complicating myocardial infarction, but in view of the therapeutics advantages of endocardial pacing (Scott et al 1967), a further histopathological study of nine cases was undertaken.

METHODS

The hearts were examined in the routine manner at the time of necropsy. Later the formalin-fixed specimens were prepared for examination of the conducting tissue using the method described by Hudson (1965). Partial serial sections at 0.5 mm were made through a block of the upper interventricular septum to display the main AV bundle, the AV node and bundle branches. The histological sections were routinely stained with haematoxylin and eosin, and also with Hart’s modification of Weigert’s elastic tissue stain, counterstaining with van Gieson’s picro-fuschin. Sections displaying the AV node, the main bundle and the bundle branches were examined in order to assess the relative amounts of conducting fibres, fibro-elastic tissue and the presence of fatty tissue (Sims, 1968). The examination was made without a knowledge of the clinical details in nine cases with heart block and also in sixteen cases with normal conduction. The results of this were compared statistically using an exact probability computer programme.

RESULTS

The details of the nine cases with complete heart block are presented in Table I. The ages ranged from 38 to 76 years (average 65 years. The duration of the heart block, as estimated from the first electrocardiograph showing heart block until the time of death, was less than six hours in five cases. The heart rate was between 40–50 beats per minute, but one case had AV dissociation with a rate of 100/minute. Heart failure was present in seven of the nine cases.

In five cases a recent coronary occlusion was demonstrable, while in seven cases recent infarcts were present, of which four were anterior and three posterior in position. Recent infarction involving the conducting tissue was present histologically in four cases, and in these the duration of the block was longer than six hours. In three instances, with one anterior and two posterior recent infarcts, there was infarction of the bundle branches, and in case No. 3 (Figure) only the right bundle branch showed evidence of infarction. Case No. 6, a 38-year-old male, showed recent infarction of the AV node and main bundle associated with the unusual

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TABLE I
Cases of Complete Heart Block with Myocardial Infarction

| Case | Age & Sex | Duration of Block | Heart Failure | Occlusion Site & Age | Infarct Site | Infarct Age | Histological findings in conducting Tissue |
|------|-----------|-------------------|--------------|---------------------|-------------|------------|------------------------------------------|
| 1    | 60 M     | Several hours     | + LAD & CA  | Antero-Septal       | Recent      |            | Bundle branches: slight fibrosis          |
| 2    | 76 M     | 2 days            | + RCA      | Recent              | Posterior   | Recent     | Bundle branches: Fibrosis and recent infarction |
| 3    | 75 M     | 24 hours          | + LAD & CA | Antero-Septal       | Healed      |            | Bundle branches: Fibrosis and Recent infarction R. branch |
| 4    | 74 F     | 1 week            | + RCA      | Old                 | Posterior   | Healed     | Bundle branches: Fibrosis                  |
| 5    | 71 M     | 3-6 hours         | + LAD      | Old                 | Antero-Lat | Recent     | Bundle branches: Recent infarction         |
| 6    | 38 M     | 1 week            | + RCA      | Recent              | Antero-Lat | Recent     | Bundle branches: AV node & AV bundle       |
| 7    | 74 M     | Several hours     | + LAD      | Old                 | Posterior   | Healed     | Bundle branches: Recent infarction         |
| 8    | 65 M     | Several hours     | LAD         | Posterior           | Recent      |            | Bundle branches: AV bundle and            |
| 9    | 57 M     | 2 hours           | LAD & RCA  | Old                 |             |            | Fibrosis                                  |

LAD = Left anterior descending artery  CA = Circumflex artery (left)  RCA = Right coronary artery

TABLE II
A comparison of the histological features of conducting tissues in cases with complete heart block complicating myocardial infarction and cases with normal conduction.  (Age comparison of groups P > 0.25)

| Conducting Tissue | Number of Cases | Amount of Conducting Tissue | Histological Assessments |
|-------------------|-----------------|----------------------------|--------------------------|
|                   |                 | Fibrosis | Elastosis | Fat |
|                   |                 | + + + + | + + + + | + + |
| AV Node           | Heart Block 9   | 0 3 4 6 | 4 3 2 0 0 6 3 0 | 4 5 |
|                   | Normal 16       | 0 10 6 2 10 4 0 0 3 9 4 | 4 12 |
|                   | P = 0.23        | P = 0.29 | P = 0.034 | P = 0.39 |
| AV Bundle         | Heart Block 9   | 0 4 5 2 4 3 0 0 6 3 0 | 4 5 |
|                   | Normal 16       | 0 7 9 0 9 4 3 0 5 6 5 | 8 8 |
|                   | P = 1.0         | P = 0.16 | P = 0.11 | P = 1.0 |
| Bundle Branches   | Heart Block 9   | 2 4 3 0 4 1 4 0 6 3 0 | 6 3 |
|                   | Normal 16       | 0 9 7 1 10 3 2 0 3 8 5 | 10 6 |
|                   | P = 0.23        | P = 0.38 | P = 0.032 | P = 0.99 |

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finding of a recent occlusion of the right coronary artery and an anterior infarct. In several cases there was fibrosis of the bundle branches. When the histological features were compared statistically with assessments from a similar age group with normal conduction (Table II), no significant difference is detected, where P is never <0.02.

**DISCUSSION**

Since myocardial infarction cannot be recognised using haematoxylin and eosin staining technique for about 10–12 hours after the onset of ischaemia, changes could not be expected in three cases where heart block was present for less than six hours. In these cases histological examination was therefore not helpful.

From a knowledge of the conducting tissue of the human heart and its blood supply (James and Burch, 1958) it could be predicted that occlusion of the right coronary artery would produce posterior infarction with ischaemia of the AV node in 90 per cent of cases, producing perhaps heart block for a short time until a satisfactory collateral blood supply developed. Occlusion of the anterior descending branch of the left coronary artery will lead to anterior infarction, which may produce heart block by extension into the septum to involve the bundle branches. The heart block so produced is more likely to be associated with a large infarct and to be permanent. This association of small posterior infarcts with transient heart block and of large anterior infarcts with permanent block is much modified by the individual variation in blood supply, the distribution of atherosclerosis, and the development of the collateral circulation in the heart. This is demonstrated by case No. 2, in whom there was extension of the posterior infarct forwards into the septum to involve both bundle branches, while in case No. 6 the recent infarction of the AV node and bundle was associated with a recent anterior infarct.
Another factor which would appear to be important is previous damage of the conducting tissue, so that the remaining tissue is more likely to be vulnerable to further ischaemia. This would seem to be the case in four of the present cases where scarring was present in the bundle branches. In two of these cases the bundle branches were infarcted but in the other cases no infarction was discernable. However, when the histology of cases of similar age with normal conduction was compared, no significant increase of fibrosis in the bundle branches was found. This apparent fibrosis may therefore represent an ageing change, and in this state ischaemia could easily produce either a transient or persistent failure of conduction. In case No. 3 there was infarction of the septal myocardium and right bundle branch. After 24 hours normal AV conduction returned, but with residual right bundle branch block. Davies et al (1967) in a study of fourteen cases with transitory complete heart block complicating acute myocardial infarction, showed that focal necrosis of the AV node was present in three instances, four had partial and one had total necrosis of the left fascicles, and two had bilateral branch necrosis when chronic heart block was more likely to ensue.

Seven of the nine cases studied were also included in a clinical investigation of the treatment of complete heart block complicating myocardial infarction with either endocardial pacing or drugs (Scott et al, 1967). Of the fifty patients in that study, twenty-nine had inferior (pathologically referred to as posterior) infarcts and eighteen had anterior infarcts, while two had both. The mortality was found to be 59 per cent in patients with anterior but only 39 per cent in those with inferior infarcts. The reason for the greater mortality with anterior infarcts complicated by complete heart block, is that infarcts must be large to involve both bundle branches, and are therefore more likely to be accompanied by shock and heart failure. It is in this group that endocardial pacing was found to be of value, since by raising the heart rate to 85–90 beats per minute, the cardiac output could be maintained and the circulatory failure corrected.

Complete heart block occurs in 5–8 per cent of cases with myocardial infarction but only in about 10 per cent of these does it become chronic (Lancet, 1968). In the study of Scott et al (1967), the heart block lasted an average of three days and in only one case did Grade I heart block persist. Clinically they found that the prognosis depended upon the age of the patient, the site of infarction and the time of onset of complete heart block.

**Summary**

The conducting tissue of the heart has been examined in nine cases with complete heart block complicating myocardial infarction. There was evidence of recent infarction in the bundle branches in three cases and of the AV node and main bundle in another case, but the distribution of necrosis has little relationship to the site of arterial occlusion or the location of the infarct. This is accounted for by the widespread nature of the arterial disease and the extensive collateral circulation. Previous damage with subsequent fibrosis, or the degeneration due to ageing of the conducting tissue, may also make the development of heart block more likely with any further episodes of ischaemia. These findings are discussed in the light of a contemporary clinical investigation.
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