Changes in the QRS complex after aortic valve replacement

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The electrocardiograms of 50 patients after isolated aortic valve replacement were examined. Two main types of postoperative QRS changes were found. In 13 patients intraventricular conduction defects developed, predominantly in the form of a left anterior hemiblock as an isolated lesion or combined with other focal blocks. The other prominent finding was the appearance of abnormal Q waves suggesting inferior wall infarction in 8 patients, and anterior wall infarction in one.

Intraventricular conduction defects were interpreted as evidence of involvement of the conducting system during removal of the abnormal valve, whereas uneven myocardial blood flow during coronary perfusion was thought to be responsible for the appearance of abnormal Q waves. The clinical importance of these findings is discussed.

Interest in intraventricular conduction defects has recently been renewed by Rosenbaum et al. (1969a). They showed the trifascicular character of the conduction system and introduced the term 'hemiblock' to describe conduction disturbances in one of the two divisions of the left bundle. Left anterior hemiblock was defined by left axis deviation between -30° and -90°, initial QRS forces directed inferiorly and to the right, and slight prolongation of the QRS duration by 0.02 sec or less (Rosenbaum et al., 1969c). Left posterior hemiblock was characterized by right axis deviation to +120°, superiorly directed initial forces, and terminal QRS forces to the right and inferiorly (Rosenbaum et al., 1969b; Castellanos et al., 1969).

Focal conduction defects not only change the QRS complex profoundly, thus obscuring the signs of ventricular hypertrophy and myocardial infarction, but they may be the first warning of complete atrioventricular block which may threaten life. Considering the anatomical relation of the bundle of His and its main branches to the ventricular septum and the aortic valve (Fig. 1), mechanical injury to these structures seems likely to develop during cardiac surgery. Atrioventricular block and bundle-branch block are well-known complications after closure of ventricular septal defects (McGoon, Ongley, and Kirklin, 1964; Kulbertus, Coyne, and Hallidie-Smith, 1969) and the frequent development of left parietal block after transventricular aortic valvotomy has also been reported (Samson and Bruce, 1962). Aortic valve replacement, when coronary perfusion and cross-clamping of the aorta present additional hazards to the myocardium, seems to carry an increased risk of intraventricular conduction defects. In recent reports on valve replacement, the occasional production of atrioventricular block has been described (Kloster, Bristow and Griswold, 1965; Gannon et al., 1965) but the incidence of bundle-branch block or changes of the electrical axis were not mentioned.

In this paper, the electrocardiographic changes were analysed in 50 patients in order to investigate postoperative abnormalities of the QRS complex.

Patients and methods

Fifty patients undergoing isolated aortic valve replacement between 1967 and 1970 were included in this study (42 Starr-Edwards valves, 7 homografts, and 1 fascia lata repair). The only criterion for inclusion in this study was the availability of a 12 lead pre- and postoperative electrocardiogram. Patients who died in the immediate

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Results

The electrocardiographic changes in the 50 patients after aortic valve replacement are listed in the Table.

(a) Preoperative abnormalities In 7 patients there was evidence of pre-existing intraventricular conduction defect. In one there was complete heart block which regressed after operation to right bundle-branch block with left anterior hemiblock. Three patients had complete left bundle-branch block before operation; 1 developed right bundle-branch block with left anterior hemiblock after operation, another regressed to left anterior hemiblock and in the other patient left bundle-branch block persisted after operation. Three further patients had left anterior hemiblock before operation and this conduction defect persisted after valve replacement.

In 1 patient there were abnormal Q waves in leads II, III, and aVf before operation. This patient developed changes suggesting anterior wall infarction after operation, the details of which are discussed later.

(b) Postoperative development of intraventricular conduction defects The different types of intraventricular conduction defect occurring after operation are schematically represented in Fig. 2.

Damage to all three fascicles (trifascicular block) was found in 2 patients. The first developed complete AV block during operation, needing artificial pacing for three days. The electrocardiogram subsequently changed to left bundle-branch block with first degree

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postoperative period without having a complete electrocardiogram recorded were thus excluded from the series. There were no late deaths among the patients.

The electrocardiograms were analysed for changes of the QRS complex with special emphasis on the following criteria. (1) Initial ventricular activation (0–02 sec vector); (2) frontal plane QRS axis; (3) terminal QRS forces; (4) duration of the QRS complex. Serial postoperative electrocardiograms were reviewed to examine the evolution of the different changes.

The routine procedure at valve replacement consisted of (1) initiation of cardiac bypass and cooling to 32°C; (2) aorta cross-clamped and opened; (3) perfusion of both coronary arteries to maintain a pressure of 70–90 mmHg (mean); (4) fibrillation of the heart; (5) excision of the abnormal valve and valve replacement.

Operative data relating to coronary perfusion and aortic clamping, the state of the aortic valve, and the site of calcification and its subvalvular extension, were analysed. The early postoperative course was also studied in each patient.
AV block and later to left anterior hemiblock (Fig. 3). During follow-up, left bundle-branch block returned several times with tachycardia. The second patient had left bundle-branch block before operation which changed to right bundle-branch block with left anterior hemiblock after valve replacement (Fig. 4).

Bifascicular block developed in 2 patients. Normal intraventricular conduction changed to right bundle-branch block with left anterior hemiblock in one (Fig. 5) and to left bundle-branch block in the other.

Left anterior hemiblock was the most frequent finding. In 6 patients, there was abnormal left axis deviation with an inferiorly directed initial vector and minimal widening of the QRS complex (<0.12), thus fulfilling the necessary criteria (Fig. 6). In 3 other patients, the electrocardiographic patterns were considered to be compatible with incomplete left anterior hemiblock showing left axis deviation to $-15^\circ$ and a change of the initial QRS vector only (Fig. 7).

(c) Postoperative development of abnormal Q waves This group of patients showed initial QRS vectors suggesting myocardial infarction. In 8 patients, abnormal Q waves developed in leads II, III, and aVF (Fig. 8).

In these patients, there was no significant change in the mean QRS axis, with the exception of one patient who also developed left axis deviation to $-45^\circ$. One additional patient showed loss of the praecordial R waves and changes of left anterior hemiblock (Fig. 9). That axis shift was not the cause for this appearance was substantiated when praecordial leads taken higher on the chest wall still revealed the same pattern suggesting anterior wall infarction.

(d) No postoperative change In a group of 20 patients, the QRS complex was normal before operation and remained so after valve replacement. At operation there was no evidence of subvalvar calcification and both coronary arteries were perfused without difficulty. The postoperative course in these patients was uneventful.

(e) Correlation of electrocardiographic changes with operative data

(1) Type of valve replacement A total of 42 patients had Starr-Edwards valves and, of these, 12 developed postoperative conduction defects, 8 developed postoperative Q waves, and in the remaining 22 patients there was no change in the postoperative cardiogram.

Seven patients had homograft valve replacements and, of these, 1 developed a postoperative conduction defect, 1 developed postoperative Q waves, and in the remaining 5 patients there was no change in the postoperative electrocardiogram.

One further patient had a fascia lata valve replacement without any change in the cardiogram.

**FIG. 2** Graphic representation of the intraventricular conduction defects.
(2) Coronary perfusion Of the 50 patients, 42 had both right and left coronary arteries perfused during operation. Thirteen of these patients developed postoperative conduction defects, i.e. all patients with conduction defects had both coronaries perfused. A further 5 developed postoperative Q waves, including 2 patients in whom blood flow in the right coronary artery was inadequate and had to be interrupted several times during operation and 1 patient in whom anomalies of the right and left circumflex coronary arteries were shown by preoperative coronary angiography. The remaining 24 patients showed no change in the postoperative cardiogram.

In the remaining patient, the aorta was clamped at 29°C for 45 minutes without any form of coronary perfusion but this patient showed no postoperative electrocardiographic changes.

(3) Calcification of aortic valve Heavy calcification of the aortic valve with subvalvar extension of calcium was described by the surgeon in 9 patients; 2 already had pre-existing preoperative conduction defects, 4 developed postoperative conduction defects, 1 developed postoperative Q waves, and in the remaining 2 patients there was no change in the electrocardiogram after operation.

Discussion
The incidence of postoperative electrocardiographic changes was high after aortic valve replacement. The abnormalities observed suggest damage to the conducting system and myocardium since the QRS complex, in contrast to the phase of repolarization, is not usually influenced by digitalis, antiarrhythmic agents, electrolyte changes, and pericardotomy.

Of the patients with intraventricular conduction defects, involvement of the anterior division of the left bundle was present in all, a feature that is not surprising when the close relation of this structure to the aortic valve is considered (Lev, 1964). The shift of the mean QRS axis and the change in initial ventricular activation in 3 patients were interpreted as indicating partial interruption of the left anterior fibres, that is, incomplete left anterior hemiblock. The existence of incomplete left anterior hemiblock, however, is still uncertain (Rosenbaum et al., 1969c). Right bundle-branch block was found in 4 patients, and a transient conduction defect in the left posterior bundle, never occurring as an isolated lesion, was also noted in 4 patients.

The pathogenesis of the conduction defects

FIG. 3 'Trifascicular' block. Normal preoperative intraventricular conduction (A) changing to complete AV block immediately after operation (B) and later to left bundle-branch block (C) and left anterior hemiblock (D).
is probably due to involvement of the conducting system during removal of the abnormal valve. Haemorrhagic lesions and necrosis of the atrioventricular conducting system have been observed not infrequently after aortic valve replacement (Hudson, 1967; Niles and Sandilands, 1969). Whether conduction defects are more likely to develop after Starr-Edwards valve insertion than after homograft valve replacement is uncertain, since the two procedures are not equally represented in this series.

The second prominent finding was the development of abnormal Q waves suggesting inferior infarction in 8 patients, and anterior wall infarction in 1 patient. Attention has been drawn to the association between these changes and the difficulties of coronary artery perfusion. An alternative explanation, however, which might account for the appearance of Q waves in the inferior leads is a conduction disturbance in the left posterior bundle. Experimental dissection of the left posterior bundle in primates produces an electrocardiographic pattern resembling inferior wall infarction (Watt and Pruitt, 1969). Left posterior hemiblock has also been shown clinically to cause a shift of the initial QRS vector superiorly and therefore Q waves in leads II, III, and aVF (Rosenbaum et al., 1969b). Recently the spontaneous development of wide slurred Q waves in the inferior leads was described in patients with aortic valve disease without any evidence of coronary artery disease (Warembourg et al., 1970). These authors suggested that the regurgitant jet of aortic incompetence was responsible for the abnormal Q waves by causing endocardial fibrosis in the area of the left posterior bundle. One of the accepted criteria of left posterior hemiblock is, however, a rightward shift of the mean frontal plane QRS axis, which is not a feature in our patients with inferior Q waves.

In conclusion, the frequency of intraventricular conduction defects after aortic valve replacement emphasizes the important anatomical relation between the conducting system and the aortic valve cusps. In addition, the development and variability in the types of intraventricular blocks lend further support to the concept of trifascicular intra-
FIG. 5 Postoperative 'bifascicular' block. (A) Preoperative tracing. Transient postoperative right bundle-branch block with left anterior hemiblock (B), reverting to left anterior hemiblock (C).

FIG. 6 Left anterior hemiblock. (A) Preoperative electrocardiogram, (B) left anterior hemiblock characterized by inferiorly directed initial forces (II, III, and aVF), left axis deviation (-30°), and prolonged QRS (0.10 sec).
FIG. 7 ‘Incomplete’ left anterior hemiblock. (A) Preoperative tracing. (B) Incomplete left anterior hemiblock showing a change of initial ventricular activation (disappearance of Q in II, III, aVF) and left axis shift from +45° to −15°.

FIG. 8 Postoperative development of abnormal Q waves in the inferior leads. (A) Preoperative, (B) postoperative tracing.
ventricular conduction. The clinical importance of these conduction defects is not clear. With the exception of the patient with transient third degree AV block, no haemodynamic complications were seen and the postoperative course was no different from those without electrocardiographic changes. Nevertheless, it is possible that there is an increased risk of developing complete AV block in later life (Lasser, Haft, and Friedberg, 1968).

At present, the differentiation between inferior wall infarction and left posterior hemiblock awaits further study. In view of the difficulties of coronary artery perfusion in our patients, myocardial necrosis seems the most likely explanation for the appearance of Q waves in the inferior leads.

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References

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