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Electrocardiographic Correlation of Anatomical and Haemodynamic Data in Ostium Primum Atrial Septal Defects

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The electrocardiographic characteristics of ostium primum atrial septal defects were first recognized in 1952 (Zuckermann *et al.*, 1952) and have subsequently been well documented (Rogers and Rudolph, 1953; Blount, Balchum, and Gensini, 1956; Toscano-Barbosa, Brandeburg, and Burchell, 1956; Giraud *et al.*, 1957; Pryor, Woodward, and Blount, 1959; Burchell, Du Shane, and Brandeburg, 1960).

The salient electrocardiographic features are the association of a right bundle-branch conduction defect with left axis deviation of the mid vector (30–60 msec.; superior to $-30°$ and of counterclockwise inscription in the frontal plane).

An altered sequence of ventricular excitation is now the generally accepted cause for the electrocardiographic pattern, though the left ventricular hypertrophy frequently seen in this condition was thought in some early papers to contribute to the left axis deviation (Blount *et al.*, 1956).

The anatomical findings of the specialized ventricular conduction system (Morison, 1913; Lev, 1958; Neufeld *et al.*, 1961; Visioli, Baragan, and Lenègre, 1962; Verduyn Lunel, 1964; Feldt, Du Shane, and Titus, 1966), as well as recent electrophysiological data concerning the sequence of epicardial depolarization (Durrer, Roos, and Van Dam, 1966), corroborate the theory of the altered sequence of activation.

The QRS complex in ostium primum atrial septal defect lends itself particularly well to trivector analysis. The first vector represents the activation of the interventricular septum and apical septal masses (20 ± 10 msec.), the second vector the excitation of the left ventricle (30–60 msec.), and the third vector the excitation of the right ventricle (>60 msec.).

The purpose of this paper is to apply trivectorial analysis to the electrocardiogram and to correlate the axis orientation and voltage deflection of each individual vector with the haemodynamic, surgical, and anatomical findings in ostium primum atrial septal defect.

Patients

This series consists of 22 patients who were investigated at the Royal Postgraduate Medical School between 1959 and 1967, diagnosed as having ostium primum defects, and subsequently operated upon. There were 12 male and 10 female patients ranging from 2 to 28 years of age.

Operative Findings. A complete common atrioventricular canal was noted in 6 patients and a partial form of the defect in 16: in one of these there was also a communication in the region of the membranous septum between the left ventricle and the right atrium (Gerbode *et al.*, 1961).

A cleft of the anterior leaflet of the mitral valve was observed in 15 patients. In addition to the ostium primum defect, the following conditions were noted: patent foramen ovale (2 patients), atrial septal defects of the secundum type (7 patients), and pulmonary valve stenosis (3 patients).

Mitral regurgitation was observed at operation in 13 of the 15 patients with a cleft valve. The mitral insufficiency was graded by the surgeon as slight in 1, moderate in 7, and severe in 5.

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Although left ventricular angiocardiograms were important in assessing mitral incompetence pre-operatively, only the surgical evaluation of the degree of insufficiency has been used for purposes of correlation.

Criteria of Electrocardiographic Analysis. The P–R interval was corrected for heart rate and age of patient according to Alimurung and Massel (1956). Axis determination was made from the hexaxial reference system with accuracy to 15°. Individual vectors were separated as stated in the introduction, and left axis deviation was diagnosed only when the second vector was −30° or superior.

Criteria for right atrial hypertrophy were: (1) P wave voltage in lead II higher than 2·5 mm. and duration longer than 0·08 sec.; (2) peaked P wave in leads II and III; (3) tall, early peaking of the P wave in the right praecordial leads (Nadas, 1963). Left atrial hypertrophy was assessed by a broad (0·11 sec.) notched P wave in leads I and II and a late negative deflection of the P wave in V1 wider than 0·04 sec. and deeper than 1 mm.

According to the classical concept, complete right bundle-branch block was present when the QRS complex lasted 0·12 sec. or more and when the intrinsicoid deflection was of 0·09 sec. or more in the right praecordial leads.

In the presence of delay in right bundle-branch conduction, assessment of right ventricular hypertrophy is difficult and will be discussed separately. Criteria for left ventricular hypertrophy were: (1) the sum of the voltage of RI and SIII greater than 25 mm. in adults and 30 mm. in the under 18-year group; (2) an R wave in aVL higher than 11 mm. in adults and 15 mm. in the under 18-year group; (3) a delayed onset of intrinsicoid deflection in the praecordial leads of left ventricular qRS morphology (≥0·045 sec. in both groups) (Gubner and Ungerleider, 1943; Nadas, 1963; Lipman and Massie, 1965).

Results

The rhythm was sinus in all cases except one in which a junctional rhythm had been observed for three years. First-degree atrioventricular block was present in 9 cases (41%). The mean P wave axis was +40° and was not influenced by mitral insufficiency.

Left atrial enlargement alone was present in 4 cases (2 with significant mitral incompetence, 2 without mitral incompetence). Right atrial enlargement was observed in 6 instances (4 without mitral incompetence, 2 with mitral incompetence). There were 4 cases with biatrial enlargement, all assessed as having moderate to severe mitral incompetence. A direct correlation ($p > 0{\cdot}001$) was noted between the height of the P wave in lead II and the right ventricular systolic pressure (Fig. 1). A conduction delay of the right bundle branch was noted in all cases and a complete right bundle-branch block was present in 7 patients.

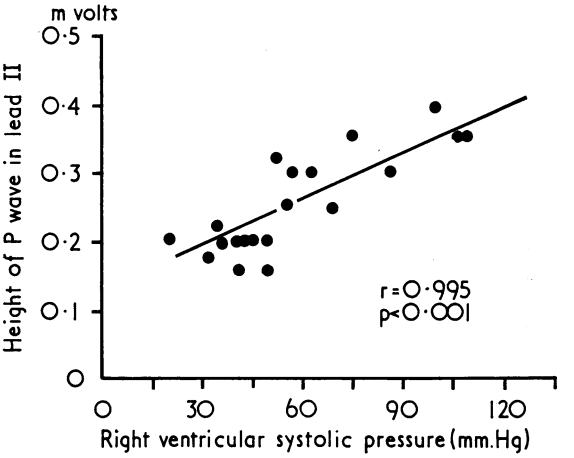

FIG. 1.—Correlation between the height of the P wave in lead II and the right ventricular systolic pressure.

The mean QRS axis was usually in the left superior quadrant (Fig. 2). However, in the cases with severe right systolic ventricular hypertension (>70 mm. Hg), the mean axis shifted to the right or inferior quadrants in all but one patient who had associated left ventricular hypertrophy from severe mitral insufficiency.

It was found that the electrocardiogram could always be specifically analysed according to the three vectors which compose the triphasic QRS complex in the limb leads (Fig. 3). The results of this analysis are set out in Fig. 4. The first vector was surprisingly localized with a mean of +95° and was not influenced by mitral regurgitation or right ventricular hypertension. The second vector in the cases with mitral incompetence (mean: −67°) appears slightly more to the left than the comparable vector in the group with competent mitral valves (mean: −45°), but this variation is not statistically significant ($p > 0{\cdot}1$). The third vector, more widely distributed, was always in the right superior or inferior quadrants with a mean of +200° and was not influenced by right ventricular systolic pressure.

In the 12 patients with moderate-to-severe mitral incompetence, two criteria for left ventricular hypertrophy were present in 100 per cent of the cases and all three criteria in 75 per cent (8 cases). Not more than one criterion was present in the patients without mitral incompetence.

The haemodynamic effects on the right ventricle were difficult to assess by electrocardiographic analysis. The criteria most frequently used in the correlation between the electrocardiogram and the

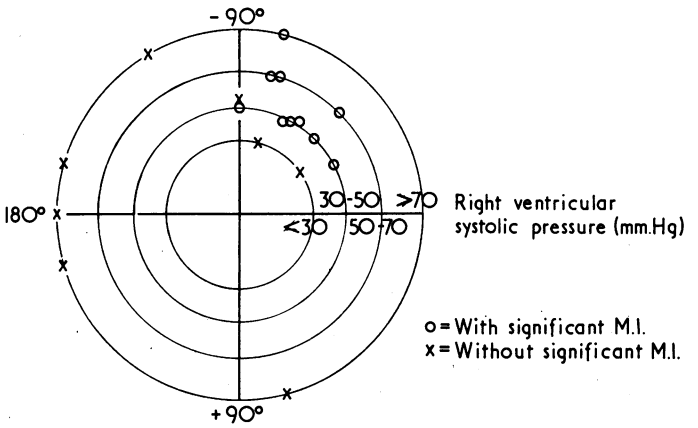

FIG. 2.—Distribution of the mean QRS axis in relation to the right ventricular systolic pressure and analysis of the influence of significant mitral incompetence (M.I. = mitral regurgitation).

right ventricular systolic pressures are the height of R or R′ in V1, R/S ratio in V1, and R/S ratio in V6. In cases associated with a conduction disturbance of the right bundle branch and right ventricular overload, the analysis of the scalar electrocardiogram is frequently complicated by the presence of a terminal vector to the right, superiorly and anteriorly or posteriorly (Lipman and Massie, 1965). This vector is related to the activation of the basal right septal mass, the basal portion of the right ventricular free wall and crista supraventricularis (Durrer, Roos, and Büller, 1965). It may be recorded either positively, isoelectrically, or negatively in V1. Therefore, the R′ wave in V1 does not represent all the forces of the right ventricle when the terminal vector in the horizontal plane is to the right and posterior. We found no correlation between the height of the R or R′ wave in V1 and right ventricular systolic pressure. The R/S ratio in V6 is more valuable, as, in this lead, the R wave is an accurate specific reference of the left ventricle and the S wave represents the right ventricular forces whether directed to the right, to the right and anterior, or to the right and posterior. Fig. 5 depicts the significant relationship of the R/S ratio in V6 to the right ventricular systolic pressures. In the cases when this pressure is greater than 50 mm. Hg, the R/S ratio is less than 1. This correlation is valid except in patients with left ventricular hypertrophy where the high voltage R wave is no longer a reference wave.

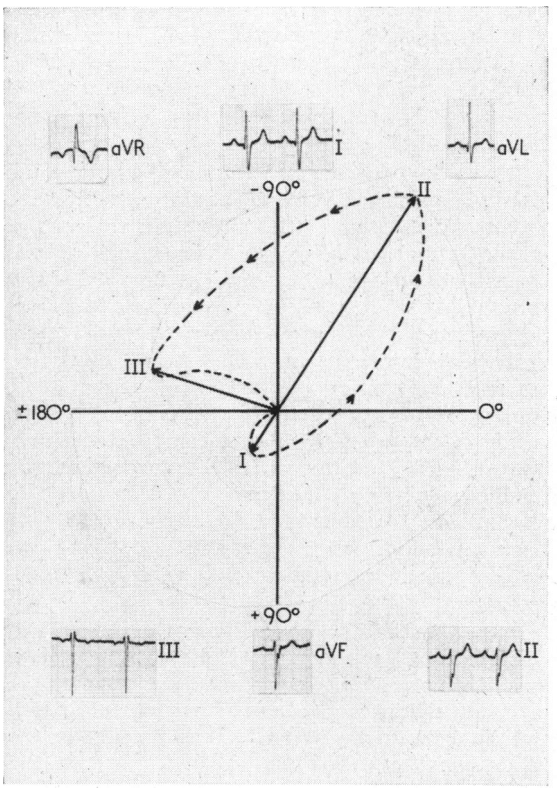

FIG. 3.—Schematic representation of a frontal plane loop corresponding to the scalar limb leads. The three vectors are drawn within the loop and are particularly well separated in leads I, III, and aVL.

The morphology of the QRS complexes in V1 varied considerably (rSR′ in 10 cases; rsR′s′ in 5; Rs in 3; R in 2; r, notched s and qR in 1). In the cases with R, Rs, and qR pattern in V1, regardless of the height of the predominant wave, severe right ventricular hypertension was usually present (systolic pressure greater than 85 mm. Hg in 5 cases and 50 mm. Hg in the sixth instance).

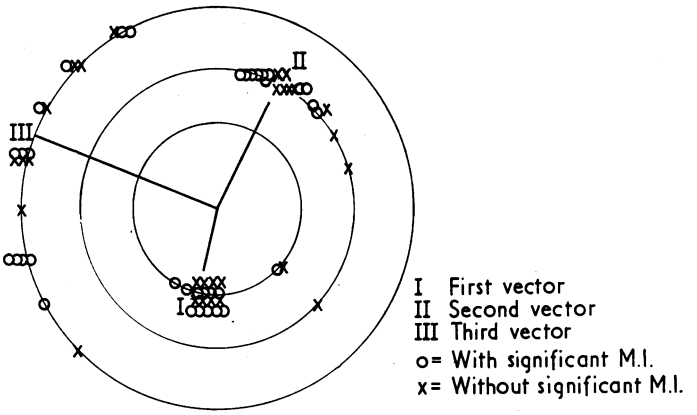

FIG. 4.—Individual vector orientation in a 360° reference system related to the dynamic influence of mitral insufficiency. The highly localized distribution of the first vector, the left axis deviation of the second vector, and the wide and rightward distribution of the third vector are well shown.

DISCUSSION

Sinus rhythm was found in all but one case, which is in accordance with most series. A junctional rhythm was the single exception: this did not coincide with single atrium as sometimes reported (Blondeau, Maurice, and Lenègre, 1966). No instance of atrial fibrillation was observed, which is probably due to the young age-group (mean: 11 years) of this surgical series (Wood, 1956; Somerville, 1965).

First degree atrioventricular block was present in 41 per cent of our cases, and this is in agreement with other series (Burch and DePasquale, 1959: 40 per cent; Beregovich *et al.*, 1960: 61 per cent; Blondeau *et al.*, 1966: 80 per cent). We were unable to support either De Oliveira and Zimmerman's (1958) findings that a long P–R interval is frequently associated with considerable left-to-right shunt or Fernandez-Caamano, Bouché-Fernandez, and Heller's (1966) assumption that a short P–R interval with a P wave axis superior to 15° signifies a very large atrial septal defect.

Left atrial enlargement did not indicate mitral insufficiency (Al Omeri *et al.*, 1965; Blondeau *et al.*, 1966). Biatrial enlargement, however, was only seen in cases with moderate to severe mitral incompetence. The finding of Ferrer *et al.* (1961) of a direct correlation between the height of the P wave in lead II and right ventricular systolic pressure was confirmed.

Left axis deviation is unrelated to mitral incompetence and is thought to be a manifestation of abnormal excitation, as proposed by Toscano-Barbosa *et al.* (1956), and, more recently, supported by Durrer *et al.* (1966).

The anatomy of the conduction system has been described in a limited number of cases of ostium primum atrial septal defects and may explain the altered sequence of excitation (Morison, 1913; Lev, 1958; Neufeld *et al.*, 1961; Visioli *et al.*, 1962; Verduyn Lunel, 1964).

The atrioventricular node is displaced posteriorly and the common bundle which skirts the lower edge of the defect is sometimes found to be tortuous and elongated (Lev, 1958; Neufeld *et al.*, 1961).

Stretching of the distorted bundle by cardiac dynamics may decrease the resting potential of the enhanced diastolic depolarization produced by the stretch (Singer, Straus, and Hoffman, 1967). This loss of resting potential may slow conduction in the bundle, and may be an additional electrophysiological cause of a prolonged P–R interval in an anatomically abnormal conduction system.

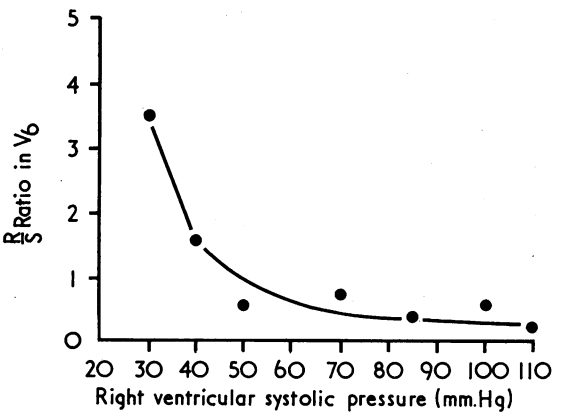

FIG. 5.—Relation of R/S ratio in V6 to the right ventricular systolic pressure.

The left bundle bifurcates very soon after its origin into two fasciculi (superior and inferior). The histological findings concerning the superior division are controversial (Visioli *et al.*, 1962). The inferior division has been found to descend abruptly to the apex and give off small branches to the postero-basal area of the septum (Verduyn Lunel, 1964). The excitation is thought to spread down the inferior fibres, reach the postero-basal part of the left ventricle earlier than usual and finally spread superiorly (Durrer *et al.*, 1966). Therefore, the direction and inscription of the vector loop is superior and counterclockwise, resulting in left axis deviation of the second vector.

The first vector shows an unusually localized distribution on the hexaxial reference system. This well-defined first vector probably results from unopposed left-to-right activation of the mid part of the interventricular septum from the inferior division of the left bundle. There is no near simultaneous activation of the septum from right to left because of the conduction delay of the right bundle-branch. Furthermore, the influence on the initial vector of the early excitatory forces present in the postero-basal region of the left ventricular wall cannot be excluded.

The third vector was always to the right and either superior or inferior. This vector is related to the right bundle-branch delay which has been clearly demonstrated by epicardial recordings (Durrer *et al.*, 1966) and is obvious electrocardiographically.

In the presence of these conduction disturbances, left ventricular hypertrophy, which alters the magnitude of the second vector, is assessed by the criteria of left ventricular hypertrophy usually applied in the presence of left axis deviation (Cabrera, 1959). From our results, mitral insufficiency, as a cause of left ventricular hypertrophy, may be suspected when two or three of the following are met: (1) RI + SIII $\geqslant$ 25 mm. in adults or $\geqslant$ 30 mm. under 18 years of age; (2) R in aVL $\geqslant$ 11 mm. in adults or $\geqslant$ 15 mm. under 18 years of age; (3) delayed onset of intrinsicoid deflection in the praecordial leads of left ventricular qRS morphology ($\geqslant$ 0·045 sec. in both groups).

It has been previously assumed (Al Omeri *et al.*, 1965; Fernandez-Caamano *et al.*, 1966) that a high qR complex in the left praecordial leads was a good guide to the presence of mitral regurgitation. We have found this criterion very useful but not completely reliable. It was present in 7 of our 12 cases with moderate to severe mitral insufficiency and also in 2 cases without regurgitation.

It must be noted that left ventricular hypertrophy from other associated causes such as left ventricular-right atrial shunt, large ventricular septal defects, etc., can satisfy the same criteria. Some of these conditions were encountered in our series but occurred in very young patients and were not accompanied by electrocardiographic evidence of left ventricular hypertrophy.

In the absence of left ventricular hypertrophy, considerable right ventricular hypertension may be suspected when the mean QRS axis is shifted to the right or inferior quadrants (Al Omeri *et al.*, 1965), or when the R/S ratio in V6 is less than 1.

We confirmed the finding of Fernandez-Caamano *et al.* (1966) that an R′ wave higher than 10 mm. in V1 is always associated with a right ventricular systolic pressure over 30 mm. Hg, but could find, on the other hand, four instances of right ventricular systolic pressure higher than 40 mm. Hg, with an R′ wave smaller than 10 mm.

Although the height of R or R′ wave in V1 appeared to be an indefinite criterion, the morphologies, R, qR, or Rs in V1 were more reliable indicators of considerable right ventricular hypertension (Blondeau *et al.*, 1966).

Summary

The electrocardiographic findings in 22 cases of ostium primum atrial septal defects were correlated with haemodynamic, surgical, and anatomical data. Except in one instance sinus rhythm was present, and first-degree atrioventricular block was present in 41 per cent of the cases.

The QRS complex was analysed by a trivectorial method. The second vector was found to be responsible for the left axis deviation and to result from conduction abnormalities of the left bundle-branch. Haemodynamic correlations revealed no significant axis shift of the individual vectors but only changes in magnitude. Significant mitral regurgitation may be suspected from criteria of left ventricular hypertrophy and biatrial enlargement. Considerable right ventricular hypertension is associated with tall P waves in lead II; ventricular complex of R, qR, or Rs morphology in V1, and, if no left ventricular hypertrophy is present, with mean QRS axis shifted to the right or inferior quadrants and R/S ratio in V6 smaller than 1.

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