Left Axis Deviation

An Electrocardiographic Study with Post-mortem Correlation

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It is now reasonably well established that the presence of left axis deviation in the electrocardiogram (mean QRS axis between -30° and -90° in the frontal plane) is, with rare exceptions, an indication of cardiac abnormality (Grant, 1956; Curd, Hicks, and Gyorkey, 1961; Libanoff, 1964). It has been postulated that the lesion responsible for the production of such an axis in most cases involves the anterior superior division of the left bundle. This fact seems to have been borne out both in the animal experiments (Watt, Murao, and Pruitt, 1965) and in clinical practice (Samson and Bruce, 1962).

When a lesion involves the anterior superior fibres of the left bundle it alters the sequence of depolarization of the left ventricle. The wave of excitation spreads via the posterior inferior division of the left bundle, and the anterior superior surface of the left ventricle is the last to be depolarized. This results in the left axis deviation of the mean QRS vector in the frontal plane. However, it is not the purpose of this paper to evaluate the above explanation of left axis deviation but rather to describe the pathological findings in patients with left axis deviation.

SUBJECTS AND METHODS

The electrocardiographic records of 353 patients were selected from the files of the Heart Station and Cardiovascular Laboratory of Barnes Hospital. All these records were taken during a period of about 12 years (from 1955 to 1966). The only criteria for selection were the presence of left axis deviation and the availability of necropsy reports. This series is selective in that only the patients with necropsy have been studied, and it deals mainly with the adult age population.

Twelve-lead electrocardiograms were available in all the patients. The QRS axis in all these patients, as

determined by plotting the mean QRS axis in the frontal plane of the hexaxial reference system, was between -30° and -90° (Massie and Walsh, 1960).

The necropsy reports of these patients were scrutinized to determine the cardiac pathology presumably responsible for left axis deviation. Significant coronary heart disease was considered to be present when evidence was found of either (a) a definite myocardial infarction, or (b) moderate to marked arteriosclerosis of the coronary arteries with myocardial fibrosis and/or cardiomegaly.

The criteria for diagnosis of pulmonary emphysema were, however, not so definite, and therefore clinical and/or necropsy evidence of moderate to marked emphysema with or without evidence of right ventricular hypertrophy had to be present on.

Cardiomegaly was said to be present when the heart weighed more than 400 g. in an average man and 350 g. in a woman. Left ventricular hypertrophy was diagnosed when the thickness of the left ventricular wall exceeded 15 mm., and right ventricular hypertrophy was diagnosed when the right ventricular wall measured more than 4 mm.

The diagnosis of cardiomyopathy and idiopathic myocarditis rested on the presence of cardiomegaly and myocardial fibrosis in the absence of hypertension, significant coronary artery disease, and primary valvular disease, such as rheumatic heart disease, congenital heart disease, and syphilitic involvement as seen in the necropsy specimen.

When no definite cardiac pathology was described at necropsy the patient was included in the miscellaneous group. None of the patients in this group had more than minimal arteriosclerosis of the coronary arteries, and there was neither myocardial fibrosis nor evidence of cardiomegaly.

RESULTS

The average age in this series was 67.1 years, with a range of 19 to 94 years. The maximum incidence was between the ages of 50 and 90 years. There were, however, 13 patients below the age of

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TABLE I
PATHOLOGICAL AETIOLOGY OF LEFT AXIS
DEVIATION IN 353 PATIENTS

	No. of patients
Coronary artery disease	299
with infarct	137
without infarct	162
Pulmonary emphysema	12
with right ventricular hypertrophy	4
without right ventricular hypertrophy	8
Rheumatic heart disease	9
Amyloidosis	5
Congenital heart disease	3
Cardiomyopathy	4
Collagen diseases	3
Miscellaneous	18
Total	353

40 years. The sex ratio was 1.85:1 with men predominating.

Table I shows the distribution of the patients according to the necropsy diagnosis considered to be responsible for the production of left axis deviation.

Coronary artery disease comprised the largest number. There were 299 patients in this group, giving an incidence of 85 per cent of the total. Of these, 137 had had a myocardial infarction. Their distribution according to location is shown in Table II. The remaining 162 showed evidence of moderate to marked arteriosclerosis of the coronary arteries, with myocardial fibrosis and/or cardiomegaly.

There were 57 patients in whom a diagnosis of *pulmonary emphysema* was made at necropsy. Of these, however, 45 also had evidence of significant coronary artery disease, either infarction or coronary arteriosclerosis. In these 45 patients it was felt that coronary artery disease, rather than pulmonary emphysema, was the cause of left axis deviation, and as such they were included in the Coronary Artery Disease Group. The remaining 12 patients showed clinical, radiological, and electrocardiographic evidence of pulmonary emphysema. At

TABLE II
INCIDENCE OF MYOCARDIAL INFARCTION
ACCORDING TO ITS LOCATION IN
137 PATIENTS

Site	No. of patients
Antero-septal	39
Massive anterior	21
Diaphragmatic	20
Antero-lateral	8
Postero-lateral	7
Apical	9
Septal	7
More than one site	26
Total	137

necropsy all of them were described as having moderate to marked bilateral pulmonary emphysema. In addition, 4 of them had evidence of right ventricular hypertrophy. None had significant coronary artery disease. Therefore, in at least 12 patients pulmonary emphysema appeared to be responsible for the left axis deviation (Fig. 1).

FIG. 1.—Electrocardiogram of a 70-year-old man, showing P pulmonale, left axis deviation, and prominent S waves in left precordial leads. At necropsy advanced bullous emphysema of both lungs was found.

Rheumatic heart disease was present in 14 patients. Five of them also had a myocardial infarction, and they were included in the infarction group. Out of the remaining 9, 3 had pure aortic valvular disease and the other 6 had combined aortic and mitral valvular disease. No difference was found between aortic stenosis and aortic insufficiency as a cause of left axis deviation. All of these 9 cases had marked left ventricular hypertrophy, and 6 had microscopical evidence of myocardial fibrosis.

This report deals selectively with patients of adult age-group, and hence there were only 3 patients with *congenital heart disease*: 1 with ventricular septal defect, 1 with coarctation of the aorta, and 1 with tetralogy of Fallot, with Potts's anastomosis.

Under the heading of *miscellaneous*, were grouped 18 patients, the only common finding in this group being the fact that the left axis deviation was difficult to explain on the basis of available necropsy data. No significant myocardial pathology was described in these instances. However, in 2 patients with hypertension and left ventricular hypertrophy, and in 1 with aortic valve disease

(syphilitic aortitis), it is conceivable that a lesion responsible for left axis deviation might have been missed, as the necropsy study was not specially undertaken from this point of view. There were 2 patients with renal failure and hyperkalaemia; the electrocardiogram in them showed other manifestations of hyperkalaemia in addition to left axis deviation, and the development of left axis deviation coincided well with the onset of hyperkalaemia (Fig. 2). The evolution of left axis deviation with the onset of hyperkalaemia prompts one to incriminate hyperkalaemia as the causative factor. In the remaining 13 patients in this group no significant pathology was found. This series also included 5 patients with amyloidosis, 4 with cardiomyopathy, and 3 with collagen disease (2 with scleroderma and 1 with dermatomyositis).

The predominant electrocardiographic abnormalities in the entire series included myocardial infarction, left ventricular hypertrophy, incomplete and complete left bundle-branch block, right bundle-branch block, myocardial ischaemia, and non-specific abnormality of the S-T segment and T wave. There were, however, 63 records in which left axis deviation was the only electrocardiographic abnormality. We were interested to find the cause of left axis deviation in these 63 patients. Table III shows an analysis of these patients.

DISCUSSION

It is now strongly suspected that left axis deviation of -30° or more in the frontal plane is an abnormal electrocardiographic finding. In our series there were 353 cases with left axis deviation of this degree, and only 13 of them failed to show any significant abnormality at necropsy. Thus, left axis deviation was a pathological finding in about 90 per cent of the patients. These figures generally agree with the previously published data from various other centres (Curd *et al.*, 1961; Corne *et al.*, 1965).

Coronary Artery Disease. The incidence of coronary artery disease was 85 per cent and that of myocardial infarction 39 per cent. In our series antero-septal, massive anterior, and diaphragmatic myocardial infarcts were the most common of the diagnoses among the infarction group. Considering the emphasis placed by some authors on anterior superior subdivisive block, the incidence of antero-lateral infarction in this series was unexpectedly low. Involvement of some part of the anterior wall of the left ventricle, however, was present in at least 96 of 137 patients with myocardial infarction. In these 96 patients it is possible to visualize

FIG. 2.—Two electrocardiograms of a 42-year-old man with glomerulonephritis. Record A, taken on October 10, 1958 shows an inverted T wave in V3 and flat or low T waves in the rest of the record. The patient developed oliguria on October 16, followed by progressive hyperkalaemia; he died 5 days later. Record B, taken on October 18, shows development of left axis deviation and changes in the T waves which are now upright and peaked in the precordial leads. Necropsy confirmed the diagnosis of glomerulonephritis.

the involvement of the anterior superior fibres of the left bundle by infarction. In the remaining 41 the anatomical location of the infarction was such that it could not have involved these conduction fibres. However, as all these patients had

TABLE III

63 PATIENTS WITH LEFT AXIS DEVIATION AS AN ISOLATED ELECTROCARDIOGRAPHIC FINDING

	No. of patients
Coronary artery disease	48
with infarct	4
without infarct	44
Pulmonary emphysema	2
Hyperkalaemia	1
Scleroderma	1
Ventricular septal defect	1
Cardiomyopathy	1
No cause	9
Total	63

FIG. 3.—Electrocardiogram of a 72-year-old woman with a 10-year history of hypertension. Record A shows evidence of left ventricular hypertrophy. Record B, taken after an episode of sudden weakness and hypotension, displays evidence of acute diaphragmatic infarction and left axis deviation. At necropsy recent diaphragmatic infarction was confirmed.

marked coronary artery disease, it is conceivable that an ischaemic process other than infarction (such as myocardial fibrosis or ischaemia) might have been responsible for the involvement of the anterior superior division of the left bundle, with production of left axis deviation. Whether such a mechanism can be invoked in all these 41 patients is difficult to say with certainty, as we do not have a detailed histopathological study of the conduction fibres of the bundle of His. This difficulty becomes even more marked when one has to explain the development of left axis deviation on the basis of a presumed altered path of conduction in cases of diaphragmatic infarction. There were 20 patients in this series where the infarction was localized to the diaphragmatic surface. In 18 of

them, development of left axis deviation could not be definitely correlated with the occurrence of diaphragmatic infarction, either because of lack of a control tracing before the infarction or because of the presence of left axis deviation in control tracings. There were, however, 2 patients among these 20 in whom left axis deviation developed acutely after the occurrence of a diaphragmatic infarction. The pre-infarction tracing did not show left axis deviation as is seen in Fig. 3. The mechanism responsible for abnormal left axis deviation in these patients would be different from peri-infarction block. Here the infarction leads to loss of inferiorly directed electrical forces, leaving the superiorly directed forces unopposed. The mean QRS axis is thus deviated superiorly and to the left, producing an abnormal left axis deviation (Kohn and Harris, 1965).

Pulmonary Emphysema. It has long been debated whether or not pulmonary emphysema *per se* can give rise to left axis deviation. Most authors believe that it is rarely, if ever, a cause of abnormal left axis deviation, and that when left axis deviation is seen in cases of pulmonary emphysema it is due to associated coronary artery disease (Curd *et al.*, 1961; Davies and Evans, 1960; Banta, Greenfield, and Estes, 1964). This seems to be true for the majority of the cases, but there is a small minority where it seems that pulmonary emphysema may have been responsible for left axis deviation (Castle and Keane, 1965). In our series there were at least 12 patients in whom pulmonary emphysema was diagnosed clinically and confirmed at necropsy, and who showed no significant coronary artery disease. Four of them also had right ventricular hypertrophy. The electrocardiogram in all the 12 subjects showed features suggestive of pulmonary emphysema in addition to the marked left axis deviation, so that we have little doubt that pulmonary emphysema was the cause of left axis deviation. The mechanism of left axis deviation and its significance in pulmonary emphysema remain controversial. Most authorities believe that such an axis, in fact, represents marked right axis deviation, and have referred to it as "axis illusion" or "pseudo left axis deviation" (Walsh, 1967; Pryor and Blount, 1966). The QRS loop in these instances is located posteriorly and much superiorly, and shows counterclockwise inscription in the frontal plane. It is not certain how such a change in QRS axis is brought about in emphysema. Originally it was thought that it was due to a shift in the anatomical position of the heart brought about by the overdistended lungs. Later studies, however, did not confirm such a rotational change.

Moreover, it was conclusively shown that the electrical axis of the heart was not necessarily dependent on the anatomical position of the heart. Grant (1956) gave a different explanation for this type of axis in emphysema. He postulated that abnormal conduction in the emphysematous lung tissue surrounding the heart was the causative factor, and most investigators tend to agree with this explanation (Libanoff, 1964; Walsh, 1967; Pryor and Blount, 1966).

Rheumatic Heart Disease. All our patients with rheumatic heart disease and left axis deviation had involvement of the aortic valve. Aortic stenosis has been found to be the most common lesion in other reported series (Curd *et al.*, 1961; Corne *et al.*, 1965). We did not, however, find any significant difference in the incidence of aortic stenosis and aortic insufficiency, though the number of patients is too small to be statistically significant.

Left ventricular hypertrophy and focal myocardial fibrosis or infarction were present in almost all the patients in this group. We feel, though it cannot be proved, that the underlying pathology in these patients is perhaps involvement of the anterior superior fibres of the left bundle by fibrosis, caused either by rheumatic process or relative coronary insufficiency. The universal presence of aortic valve involvement and the proximity of the conduction fibres to the aortic ring lend support to this view. There was only one patient with pure mitral valve disease, but this was associated with myocardial infarction.

Congenital Heart Disease. According to Ongley and Dushane (1965), left axis deviation is seen in about 5–10 per cent of the cases of uncomplicated ventricular septal defect, and in nearly all those with the atrioventricular canal type of ventricular septal defect.

The patient with coarctation of the aorta in the series showed left ventricular hypertrophy and focal myocardial fibrosis. These findings are sufficient to account for the left axis deviation.

We have difficulty in explaining the cause of left axis deviation in the case of tetralogy of Fallot. Gasul, Richmond, and Krakower (1949) described a case of cyanotic congenital heart disease with left axis deviation, which was thought to be a case of tricuspid atresia. The necropsy showed that this patient had tetralogy of Fallot with a patent foramen ovale. In our patient the pre-operative electrocardiograms are not available, but when he was first seen in Barnes Hospital in 1961 he had evidence of biventricular hypertrophy and left axis deviation (Fig. 4).

Cardiomyopathies. Our series also included 5 patients with cardiac amyloidosis, 4 with cardiomyopathy, 2 with scleroderma, and 1 with dermatomyositis. The cardiac abnormalities and left axis deviation in these entities have been well described (Farrokh, Walsh, and Massie, 1964; Schamroth and Blumsohn, 1965; Sackner, Heinz, and Steinberg, 1966).

There were 2 patients in whom it was felt that hyperkalaemia might have been responsible for the production of left axis deviation (Fig. 2). Pryor and Blount (1966) reported that it might rarely cause left axis deviation.

CONCLUSION

Analyses have been made of 353 patients with left axis deviation on the basis of necropsy diagnosis. Coronary artery disease was found to be the most common lesion, accounting for 85 per cent of the total series. Pulmonary emphysema was thought to be responsible for left axis deviation in 12 patients and hyperkalaemia in 2 patients. The incidence of other abnormalities and their significance in relation to the development of left axis deviation has been discussed. There were 13 patients in this series in whom no definite cause was found for the left axis deviation.

Of the 63 patients who had left axis deviation as the only abnormality in the electrocardiogram, 48 showed evidence of significant coronary artery disease at necropsy.

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FIG. 4.—Electrocardiogram and vectorcardiogram (Frank system) of a 19-year-old youth with tetralogy of Fallot. These records were taken 12 years after a Potts's anastomosis was performed. The electrocardiogram and vectorcardiogram show evidence of biventricular hypertrophy and left axis deviation.

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