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EARLY ONSET AND SIGNIFICANCE OF INCOMPLETE LEFT POSTERIOR HEMIBLOCK IN NIGERIANS

T. O. COLE AND C. O. MBANEFO

Department of Medicine, University College Hospital, Ibadan

Summary

Following detailed clinical and electrocardiographic analysis, twenty-five patients with incomplete left posterior hemiblock without associated complete right or left bundle branch block are presented. First-degree atrioventricular block was relatively common in these patients and associated incomplete right or left bundle branch block was also present in a few. Hypertension was the commonest underlying cardiovascular disease but two of them, without any clinically overt cardiovascular disease, presented with a history of episodic dizziness or syncopal attacks. With the present knowledge of the anatomy and histopathology of intraventricular conduction system, the relevance of tendency for Africans to form excessive fibrous tissue has been emphasized. Considering the unexpectedly low average age of these patients, suggestions have been made with a view to reducing the rate of progression of such atrioventricular conduction defects to the need for implanted cardiac pacemakers.

Résumé

Une analyse clinique et électrocardiographique détaillée permet d'identifier vingt-cinq patients dotés d'un hémiblock posterieur guache incomplet sans association d'un bloc complet de branches du faisceau de His. La presence d'un bloc atrio-ventriculaire du premier degré était assez fréquente chez ces patients ainsi que celle d'un bloc incomplet de branches droite et gauche du faisceau de His, parmi quelques-unz d'entre eux. L'hypertension était la maladie cardiovasculaire potentielle la plus répandue. Deux patients, bien que ne présentant aucune affection cardiovasculaire decelable par examen clinique, se plaignaient de vertiges épisodiques ou de syndopes. A la lumière de nos connaissances actuelles de l'anatomie et de l'hystopathologie des systemès de conduction intraventriculaires, la tendance manifestée par les Africains à une production excessive de tissus fibreux, a été soulignée. Compte tenu de la moyenne d'âge remarquablement basse de ces patients, des propositions furent énoncées an vue de jugular la progression de tels troubles du système de conduction atrio-ventriculaire par l'implantation de stimulateurs cardiaques.

Correspondence: Dr T.O. Cole, Department of Medicine, University College Hospital, Ibadan, Nigeria.

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Introduction

It is now established that the normal human intraventricular conduction network is and operates as trifascicular, the terminal segments of which are the right bundle branch (RBB) and the two divisions, anterior and posterior of the left bundle branch (Rosenbaum, Elizari & Lazzari, 1968; Rosenbaum, 1968). Of late electrocardiographic (ECG) studies in man have shown that complete heart block (CHB) is more often preceded by blocks in these peripheral segments, for instance, right bundle branch block (RBBB) with or without left anterior (LAH) or left posterior hemiblock (Rosenbaum, 1968; Kulbertus & Collignon, 1969). Rosenbaum *et al.* (1969a) described the syndrome of RBBB with intermittent LAH and left posterior hemiblock (LPH) and the consequent complete intraventricular

block. According to the authors, LPH is usually preceded by RBBB and/or left anterior hemiblock. This is understandable as the left posterior segment, apart from the better and different blood supply, is shorter, thicker and haemodynamically less vulnerable compared with the other two fascicles (Rosenbaum *et al.*, 1968; Rosenbaum, 1968). However, several cases of LPH in association with predivisional left bundle branch block (LBBB) rather than RBBB, have been described in Nigerians (Cole & Falase, 1975; Cole & Mbanefo, 1978; Cole, 1979). It is relevant to mention that at no time was RBBB with or without left hemiblock, recorded in any of these patients. In one of them, the associated LPH was initially incomplete (Cole, 1979). The frequent observation of this degree of LPH without associated complete right or predivisional left bundle branch block in this hospital, precipitated a review of this seemingly peculiar conduction defect in Nigerians. The present communication describes the results of the study and discusses the relative implications in the atrioventricular (AV) conduction system in Nigerians.

Materials and methods

This report is based on the study of the first twenty-five indigenous Nigerian patients with isolated incomplete LPH, that is, without associated complete RBBB or predivisional LBBB, recognized in less than a year in 1978, from the Cardiology and Renal Units, Department of Medicine, University College Hospital, Ibadan. The diagnosis of incomplete LPH was made after detailed clinical, radiological and ECG assessment.

Pure LPH produces a non-specific ECG pattern, that is, S_1 Q_3 with a mean QRS electrical axis shift to around +120° (Rosenbaum *et al.*, 1968; Rosenbaum, 1968, 1969). The diagnosis can therefore only be made after excluding other possible causes of such ECG changes. In the previously reported case of predivisional LBBB with the associated LPH initially incomplete (Cole, 1979), the ECG diagnosis of the latter, was based on a mean QRS electrical axis around +75° and the relatively small S_1 Q_3 pattern. This later became more complete, having fulfilled the above criteria laid down for diagnosis. Understandably, the ECG diagnosis of pure incomplete LPH in the present study was arbitrarily based on (i) a mean QRS electrical axis between +80° and +110°. (ii) S_1 Q_3 pattern that is, the terminal and initial QRS forces respectively, such as can occur due to vertical orientation of the heart on deep inspiration and (iii) a slightly, if at all, prolonged QRS duration. These were only accepted after excluding the possibility of right ventricular hypertrophy — acute or established — and a vertical heart due to slender body build or emphysema (Rosenbaum *et al.*, 1968; Rosenbaum 1968, 1969). The ECG diagnosis of incomplete RBBB or LBBB was based on the conventional QRS pattern with the duration between 0.10 and 0.12 sec. In the presence of incomplete bundle branch block, the criteria for incomplete LPH were accepted as remaining unchanged although the terminal forces were a little delayed. Incomplete LBBB was regarded as predividional in view of its association with block in one of the left fascicles. Such a combination is not possible in the divisional type, the ECG pattern of which disappears when left hemiblock becomes manifest (Rosenbaum, 1969). Cases with clinically overt non-cardiac causes of conduction defect (Rosenbaum, 1968, 1969) were not included in the study. PR interval of 0.21 sec or more was accepted as prolonged.

Results

These twenty-five patients, fourteen male and eleven female, were aged 26–71 years (mean 44.2). The peak frequency of age distribution (40%) was in the fifth decade and 20 (80%) of them were aged 50 years and below (Table 1). It is interesting to note the relatively sudden fall in the age distribution as from the sixth decade. Table 2 summarizes the clinical data, degree of incomplete LPH and other associated AV conduction defects in these patients. Hypertension

TABLE 1. Age and sex distribution of twenty-five patients with incomplete left posterior hemiblock

Age group (years)	No. of cases	Male	Female
1 – 10	—	—	—
11 – 20	—	—	—
21 – 30	2	1	1
31 – 40	8	3	5
41 – 50	10	6	4
51 – 60	3	2	1
61 +	2	2	—
Total	25	14	11

TABLE 2. Age, sex, clinical data and degree of conduction defects in the twenty-five patients with incomplete left posterior hemiblock

Case No.	Sex	Age (yr)	Clinical data	Hr/ min	P-R (sec)	QRS Axis	QRS Duration (sec)	Associated conduction defects
1	F	36	Routine	76	0.21	+80°	0.08	1st degree AV block
2	F	26	Routine	88	0.16	+85°	0.08	—
3	M	29	Palpitations	70	0.16	+85°	0.08	—
4	M	46	Hypertension	56	0.18	+80°	0.08	Sinus bradycardia
5	M	42	Hypertension	90	0.18	+80°	0.08	—
6	M	39	Hypertension	84	0.16	+80°	0.08	—
7	M	43	Hypertension	78	0.18	+85°	0.08	—
8	M	44	Hypertension	68	0.18	+90°	0.08	—
9	M	52	Hypertension	72	0.18	+90°	0.08	—
10	M	43	Hypertension	54	0.18	+90°	0.08	Sinus bradycardia
11	M	35	Hypertension	68	0.16	+90°	0.08	—
12	F	37	Hypertension	66	0.20	+90°	0.08	—
13	F	32	Hypertension	95	0.16	+90°	0.10	Incomplete LBBB
14	F	41	Hypertension	86	0.14	+90°	0.08	—
15	M	68	Hypertension	78	0.16	+95°	0.08	—
16	M	38	Hypertension	84	0.22	+100°	0.08	1st degree AV block
17	M	71	Hypertension	82	0.16	+100°	0.08	—
18	F	50	Hypertension	80	0.21	+100°	0.08	1st degree AV block
19	F	54	Hypertension	116	0.14	+100°	0.08	Sinus tachycardia
20	F	38	Hypertension	55	0.20	+105°	0.12	Sinus bradycardia, incomplete RBBB
21	F	46	Thyrotoxicosis	118	0.12	+100°	0.08	Sinus tachycardia
22	M	45	Syncopal attacks	76	0.38	+105°	0.08	1stdegree AV block
23	F	48	Dizzy attacks, mitral incompetence	82	0.21	+85°	0.12	1st degree AV block, incomplete LBBB
24	F	40	Menorrhagia	84	0.14	+90°	0.08	—
25	M	51	Obese, normotensive osteoarthritic	74	0.22	+85°	0.08	1st degree AV block

was the commonest underlying cardivascular disease. One patient had thyrotoxic heart disease and another, mitral incompetence with associated dizzy attacks. The others had no clinically overt cardiovascular disease, the ECG having been requested for on a routine basis, except in one of them with a history of episodic syncope. In none of the patients were there any radiological findings suggestive of right ventricular hypertrophy or vertical heart with or without emphysema.

The range of electrical axis shift was below +100° in 18 and +100° and above in the other seven. All the patients were in sinus rhythm. First-degree AV block was present in six, incomplete predivisional LBBB in two and incomplete RBBB in another one (Figs 1–4). There was no record of Mobitz type I or II or complete AV block in any of the patients.

Discussion

Apart from the change in direction of the early forces, LAH or LPH shifts the mean QRS electrical axis to a pre-defined level, superiorly and to the left or inferiorly and to the right, respectively (Rosenbaum *et al.*, 1968; Rosenbaum, 1968; Rosenbaum *et al.*, 1969a). In some cases of left hemiblock, the mean QRS electrical axis shift may not be up to the level laid down in the criteria for diagnosis. According to Rosenbaum *et al.*, (1969b), it still remains doubtful whether such a situation arises when the left hemiblock is incomplete or whether complete left hemiblock may also produce an incomplete QRS electrical axis shift. However, several cases in favour of the existence of incomplete left hemiblocks have since been described (Rosenbaum *et al.*, 1969a; Rosenbaum, 1969; Cole & Falase, 1975; Cole, 1979). In such cases involving the left posterior segment, the hemiblock was associated with complete RBBB (Rosenbaum *et al.*, 1969a) or predivisional left bundle branch block (Cole, 1979). In the present study other causes of $S_1 Q_3$ pattern with mean QRS forces directed to the right, have been excluded. With the background

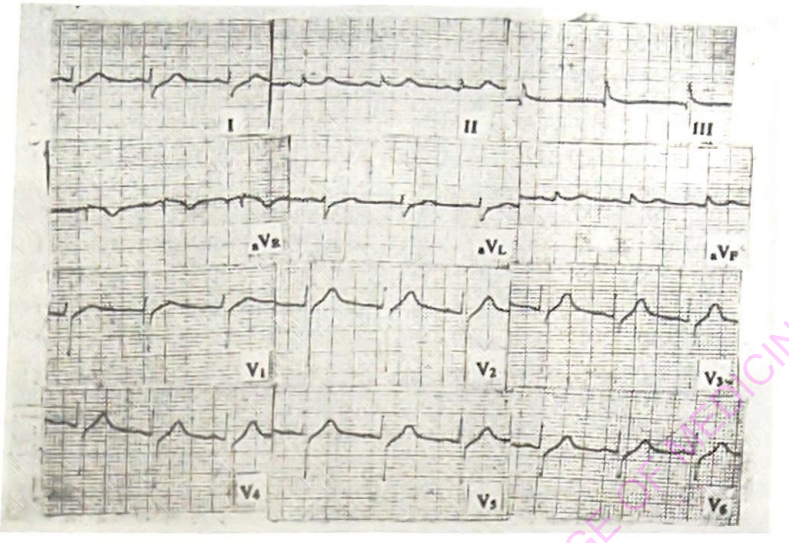

FIG 1. Twelve-lead electrocardiogram with the pattern of isolated incomplete LPH. Heart rate, 82/min and QRS duration, 0.08 sec. Note the mean QRS electrical axis oriented to the right (around +100°) with S in lead I and small Q in lead III (Case No. 17).

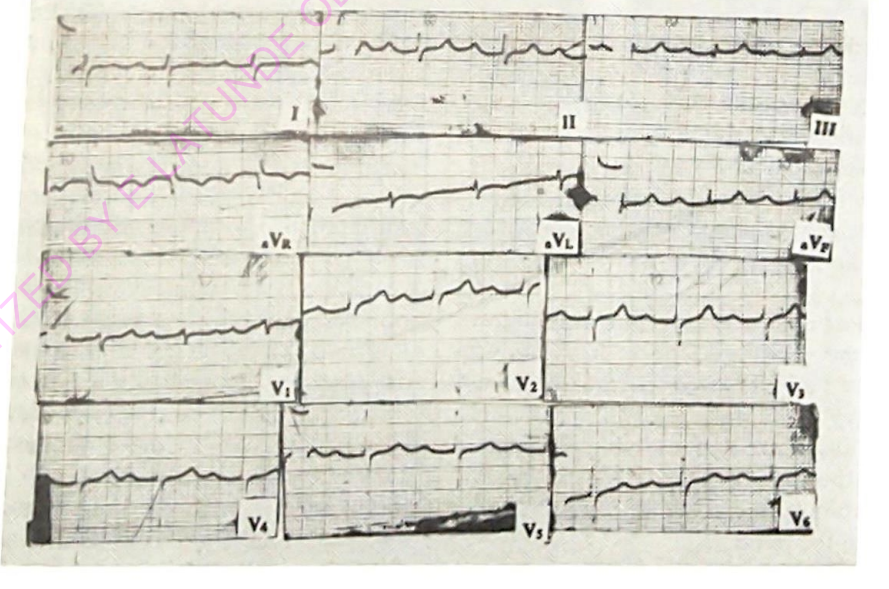

FIG. 2. Twelve-lead electrocardiogram showing incomplete LPH associated wih marked first degree AV block. Heart rate, 76/min, P-R interval, 0.38 sec and QRS duration 0.08 sec. (Case No. 22).

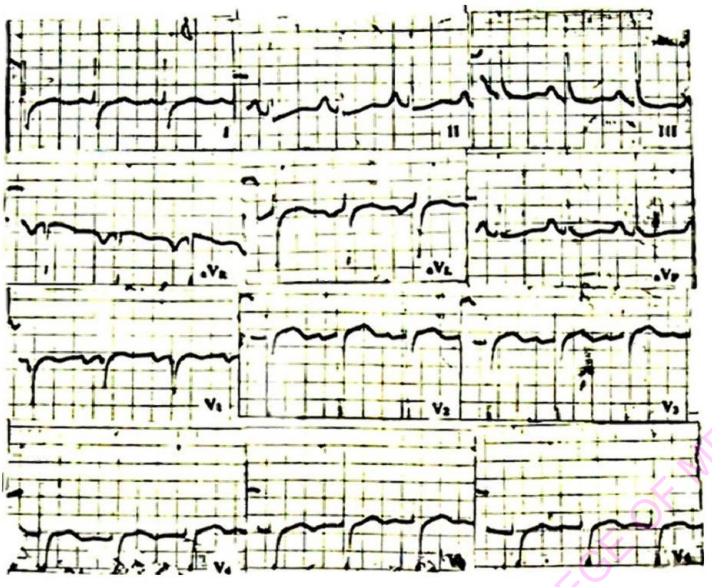

FIG. 3. Twelve-lead electrocardiogram showing incomplete LPH with associated incomplete predivisional LBBB. (Case No. 23). Heart rate, 82/min and QRS duration, 0.12 sec. Note the typical pattern of incomplete LBBB in precordial leads.

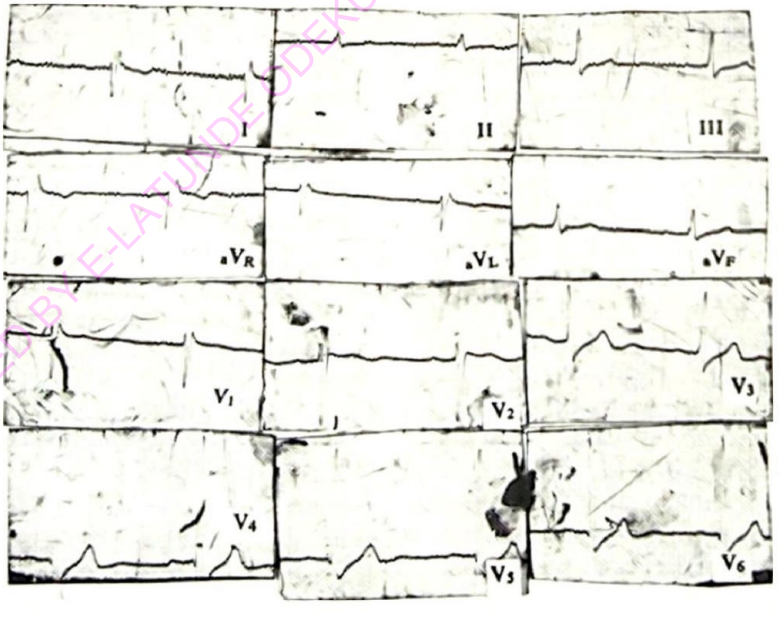

FIG. 4. Twelve-lead electrocardiogram: Incomplete LPH with associated incomplete RBBB (Case No. 20). Heart rate, 55/min and QRS duration, 0.12 sec. The pattern of incomplete RBBB is well represented in right praecordial leads.

of detailed clinical and radiological assessment, the ECG changes observed in these patients are no doubt, compatible with various degrees of incomplete left posterior hemiblock.

Of late, knowledge of the anatomy, histopathology and electro-physiology of normal and abnormal intraventricular conduction system has rapidly advanced (Rosenbaum *et al.*, 1968; Rosenbaum, 1968, 1969). The 'penetrating' portion of the bundle of His, thin and cylindrical, runs through the central fibrous body and is susceptible to pathological changes in the surrounding fibrous structure. The 'branching' portion then starts from where the predivisional LBB with its narrow proximal part, emerges from the bundle of His. Prior to branching, however, the LBB widens as the short and robust left posterior segment begins to detach itself; this becomes complete at the crest of the muscular part of the intraventricular septum. The 'branching' portion then continues until it finally divides into the long, thin and cylindrical RBB and slender left anterior segment. Understandably, predivisional LBB is most vulnerable at its point of origin where it may be exposed to mechanical compression between the membranous septum and the overlying endocardium (Rosenbaum, 1969).

According to Lenegre (1964), most cases of LBBB are mechanical in origin and this has been substantiated by histological studies of the conduction system (Lev, 1961; Lev, Sodi-Pollares & Friedland, 1963). The usual thickening of the endocardium and sclerosis of the membrane in this area, each of which advances with increasing age (Lev, 1961; Rosenbaum, 1969) may be relevant in the cases presented. The close anatomical relationship of the predivisional LBB and early fibres of the left posterior segment has indeed, been emphasized (Rosenbaum *et al.*, 1968; Rosenbaum, 1968). It is therefore possible for such changes to also involve the proximal fibres of the left posterior segment. This will then, to a varying extent, impede impulse transmission through this area of the intraventricular trifascicular system. Considering the relatively early age in the majority of the patients, the liability of Africans to form excessive fibrous tissue (Beet, 1956) may be relevant (Cole, 1979). This is evidenced, for instance, by clinico-pathological observations in Nigerians with established rheumatic valve disease (Cole, 1976). It should perhaps be mentioned that the previously described primary sclerodegeneration (Lenegre, 1964) or fibrosis (Lev, 1964) limited to the conduction system, is not likely to have been the principal cause of any of these patients. Such cases, in the early phase, invariably manifest as RBBB and/or LAH rather than isolated conduction defect in the short and thick left posterior segment (Rosenbaum, 1969; Sowton, Hendrix & Roy, 1974).

As demonstrated by His bundle electrogram (HBE), first-degree AV block usually indicates conduction disturbance in the AV nodal structure (Narula *et al.*, 1971) and very rarely, block in the bundle of His or the unaffected conduction segment (Murphy *et al.*, 1977). It is possible that this particular AV block in the series, developed following extension of the pathological process to the tail end of the AV node. Episodic dizziness or syncope reflects many causes even in patients with intraventricular conduction defects (De Pasquale & Bruno, 1973; Dhingra *et al.*, 1974a). On the basis of available evidence these symptoms should nevertheless, suggest intermittent CHB in such patients (Cosby & Bilitch, 1972; Dhingra *et al.*, 1974b). There was no ECG evidence of CHB in any of our patients with these symptoms. However, the degree of incomplete LPH and other associated AV conduction defects may reflect the severity of the pathological fibrosis involving the proximal portion of the trifascicular system. With the normal anatomical penetration of the bundle of His through the central fibrous body, recurrent dizziness or syncope in these patients probably developed following intermittent CHB originating from this site.

The low incidence of incomplete LPH after the fifth decade is probably a reflection of the population age structure and/or the limited number of patients in the series. In cases of complete AV block with the site below the AV node, the idioventricular focus is usually unreliable (Katz & Pick, 1956). Such patients consequently, have a high risk of episodic syncope following asystole or ventricular dysrhythmias. The prognosis is further influenced by many other factors including in particular, the degree of myocardial efficiency (Crosby & Bilitch, 1972). The majority of our patients in general, present late in hospital and so far, it is not clear how rapidly the pathological changes progress. Although highly speculative, it is possible that some individuals in the elderly age group outside the hospital popu-

lation, subsequently succumb to intermittent CHB, as the onset is usually unpredictable (Dhingra *et al.*, 1974a). This is perhaps, evidenced by the previously described cases of persisent predivisional LBBB with changing left hemiblocks and associated syncopal attacks (Cole, 1979).

The morbidity and mortality of AV conduction defects in the developed countries have been to a great extent, reduced by temporary and/or permanent pacing (Sowton *et al.*, 1974; Dhingra *et al.*, 1974b). Apart from the established indications, evidence is now available that minor degrees of AV conduction defects may be associated wih the risk of rapid progression to CHB and its catastrophic complications (Dhingra *et al.*, 1974b; Young *et al.*, 1977). Evidence is now accumulating in Nigeria that CHB, previously assumed to be rare (Carlisle & Ogunlesi, 1972; Cole & Falase, 1975), is not uncommon in the indigenous population (Cole & Mbanefo, 1978; Cole 1979). It is possible that the progression in some of these cases might have been accelerated by the pathological fibrosis referred to in this communication. So far, it is not clear whether or not and how soon, such cases will develop intermittent or persistent CHB with or without the usual complications. Further, the high cost of cardiac pacing is also relevant considering the present socio-economic and public health standards in developing countries. At the moment, therefore, a useful approach is to rigidly attend to the various relevant factors involved in the progression AV conduction defects in such patients. These include adequate management of the underlying heart diseases, if any, and correction of metabolic disturbances, particularly in those on anti-arrhythmic drugs (Gertenblith *et al.*, 1978). This approach will not only reduce the rate of progression to CHB, but also the morbidity and mortality of this condition in Nigerians.

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