Second Degree Block Within the Bundle Branches

N. Cristal M.D., and W. Ho M.D.

We present the ECG tracings of three patients in whom second degree block type 1 (Wenckebach) or type 2 (Mobitz) within the bundle branches was inferred from the surface ECG and the criteria for the electrocardiographic diagnosis is reviewed. This entity probably occurs more often than previously suspected and only awareness of its occurrence and accurate diagnostic criteria will reveal its true frequency and prognostic implications.

Experimental1-2 and clinical3 studies have clearly demonstrated that the conduction of an impulse can be progressively or suddenly blocked at any level within the conductive system. However, the standard electrocardiogram (ECG) has been considered of little value in locating precisely the zone of impaired conduction, especially when the block is distal to the atrioventricular junction.3

A better understanding of the electrophysiologic properties of the conductive tissue has drawn attention to the fact that, under appropriate circumstances, a second degree block within the bundles can be visualized in the surface ECG.4,5 Several reports were published recently6-8 and criteria for diagnoses determined.8

In the present paper, we report two additional cases of second degree heart block type 1 (Wenckebach) and one case of heart block type 2 (Mobitz) within the bundle branches, and the criteria for ECG diagnoses are reviewed.

CASE REPORTS

CASE 1

The electrocardiogram in Figure 1 was that of a 64-year-old white woman, suffering from arteriosclerotic heart disease and hypertension. The 12-lead ECG showed mild left ventricular hypertrophy. Frequent atrial premature beats were present, most of them nonconducted. During periods of regular sinus rhythm (P-P interval 0.80 sec) (Fig 1), successive complexes showed an increasing degree of left bundle branch block (LBBB), while the P-R interval remained constant (0.20 sec). The QRS interval was 0.08 sec in the nonconducted beat, 0.10 sec in the second beat and 0.13 sec in the third beat. The cycle is closed by a premature (nonconducted) atrial contraction. Premature atrial contractions (nonconducted) follow the first and last sinus beat in the tracing. This tracing represents a cycle of increasing block, namely second degree heart block type 1 (Wenckebach), "manifest" or "directly visualized" within the left bundle.

CASE 2

The ECG in Figure 2 was recorded from a 75-year-old white man, admitted with palpitations and dyspnea of three days duration. The ECG tracing showed a regular supraventricular rhythm of 150 beats/min (Fig 2). An intra-atrial electrocardiogram (not included) revealed atrial flutter, 500 beats/min. Two types of QRS complexes were noticed: normally conducted QRS and aberrant (LBBB) QRS. The two forms of complexes occurred in periods of 1 normal and 1 LBBB (2:1 block), 1 normal and 2 LBBB (3:2 block) or 1 normal and 3 LBBB (4:3 block). After DC cardioversion the patient reverted to sinus rhythm, with normal conduction. This tracing shows some interesting features which deserve further comment. In the upper panel, a 2:1 block is predominant. As pointed out by Langendorf,9 in the presence of 2:1 block the distinction between type 1 and type 2 block becomes difficult. Usually, prolonged observation of the rhythm may help in the differentiation by revealing spontaneous changes in the conduction ratio and/or in the conduction delay. The most important aids in the differential diagnosis are the variations in conduction delay, which are characteristic of type 1 block. These variations are more easily visualized when the block is located in the A-V node than within the bundles. In the latter case, if the variations in the conduction time remain above the limit of 0.04-0.06 sec (which is the accepted critical time interval necessary for the impulse to arrive from the contralateral ventricle and to produce a complete BBB pattern) the Wenckebach period will remain "concealed" within the bundles and its presence may be inferred from the sequence of beats in which all but the first show a complete BBB pattern. Only if the opening beat (the first beat followed the normal conducted beat) has a conduction delay between 0.01 and 0.04 sec will variations in the QRS complex occur, progressing from a lesser to a higher degree of BBB.4,8

Fortunately, changes in the conduction ratio and conduction time occurred in this tracing, resulting in a change in the conduction ratio from 2:1 block (upper panel) to 3:2 block (middle panel) and 4:3 block (lower panel). During the transition from 4:3 block to 3:2 block (beats 5,8,7-lower panel) the conduction delay changed and a "manifest" Wenckebach period was recorded, with progressive increase in the QRS duration to 0.09 and 0.11 sec. This change, which is equivalent to the increase in the P-R interval in A-V block, confirms the presence of second degree block type 1 (Wenckebach) within the left bundle.
Comments

The tracings of these two patients show a regular sinus or supraventricular rhythm, normal and aberrantly conducted QRS complexes appearing periodically with a constant P-R interval for every type of beat. In case 1, progressively increasing LBBB is present and in case 2, sequences in which all except the first beats show a predominantly complete block pattern. Thus, both these cases fulfill the criteria of Friedberg and Schamroth for the diagnosis of Wenckebach phenomena in the bundle branch.

Case 3

The ECG tracing showed in Figure 3 was that of a 65-year-old white man with an acute anteroseptal myocardial infarction, in cardiogenic shock. The ECG tracing (Fig 3) showed a normal sinus rhythm (100/min), with a constant P-R interval (0.18 sec). In lead 1, beats 3,6,8 and 11 showed a RBBB pattern. In a later V1-like monitor tracing the RBBB pattern appeared sporadically or in groups, until finally permanent RBBB was established. The beats following those with RBBB differ from the sinus beat, possibly representing incomplete right bundle branch block.

Comments

- This patient was severely ill, with myocardial infarction and grave hemodynamic derangement. Before death, he developed complete A-V block. The ECG tracings showed the appearance without any warning of aberrantly conducted beats suggesting the presence within the bundle branches of a mechanism similar to the type 2 (Mobitz) second degree heart block.

Discussion

Electrograms recording the potentials of specific components of the conductive system in situ have confirmed the hypothesis suggested by experimental studies that most of the electrophysiologic properties of the system are common to all its components. Another point clarified by these specific recordings is that the localization of the zone of impaired conduction largely determines the prognostic significance of the block.

The development of cardiac pacemakers, as well as the introduction of new antiarrhythmic drugs, oblige the clinician to define more accurately the localization of a conduction defect. Unfortunately, the intracardiac recording of specific potentials such as the His bundle recording is still far from being a routine widespread procedure and clinical electrocardiographic decisions remain based on the standard surface ECG.

The recognition of type 1 block within one of the bundles depends on a fortuitous timing of the impulse delay. This is particularly true in regard to the "manifest" type and the excellent explanation of the "delay-pattern" relation by Rosenbaum together with the systematization of the diagnostic ECG criteria by Friedberg and Schamroth bring this into sharp focus. The first and second cases reported here demonstrate clearly how these criteria should facili-
tate the diagnosis of this type of conduction defect and may also offer an explanation for many cases of “nonrate-dependent bundle branch block.”

Second degree block type 2 (Mobitz) is specifically an entity of the His-Purkinje system. Thus, from a theoretical point of view, we can hypothesize that if the block develops suddenly in one of the fascicles of the system while the others are previously blocked and unable to transmit impulses, the consequence will be a complete block, namely, a dropped beat. However, if at the moment the block develops in one of the fascicles, the other fascicles are able to transmit the impulse, the result will be a QRS complex displaying the pattern of bundle-branch block.

This hypothesis is confirmed by the case reported by Maramba, and by case 3 of the present report. It is significant that in the case reported here, not only was the ECG pattern of type 2 (Mobitz) block present within one bundle, but also the “clinical” pattern of this type of block developed: the patient later showed widespread damage to the trifascicular system, manifested by the appearance of complete A-V block. Furthermore, the isolated QRS complexes showing a RBBB pattern herald the appearance of a stable RBBB, in the same way that the blocked beat in Mobitz type 2 block frequently heralds the appearance of complete A-V block.

In our opinion, the criteria for ECG diagnosis put forward by Friedberg and Schamroth are valid, but in order to encompass all the possible instances of block within the bundles, we suggest two additions:

1) The A-V conduction time (P-R interval) must be strictly regular in every type of beat, i.e., a constant P-R interval for all beats showing a BBB pattern even if this interval is different from the P-R interval of normally conducted beats. It should be noted that the P-R interval may vary if more than one bundle is affected asynchronously. This addition takes into account the fact that the development of block within a bundle “uncovers” the conduction time through the “unblocked” bundle and this time may remain constant (with a fixed P-R interval), although it may be prolonged or even shortened (super normal conduction).

2) If the complexes with a BBB pattern occur sporadically, in only single beats, type 2 (Mobitz) block within the bundle can be inferred. The presence of this type of block will be strongly supported if the sporadically blocked beats precede the development of a stable pattern of BBB. However, as in second degree A-V block, sometimes the differentiation between type 1 and type 2 block may be difficult.

Although cases in which second degree heart block confined to the bundle branches with a diagnosis made by the standard ECG are scattered, the increasing number of reports dealing with this problem probably indicate that this entity is more frequent than previously suspected, being hitherto overlooked in most cases. Only awareness of its occurrence and application of accurate diagnostic criteria will reveal its true frequency and by this means enable us to derive more precise conclusions about its prognostic significance and its possible therapeutic implications.

References


Human Pulmonary Speleology

In the tome edited by Nagaishi, C (The Caverno-
tomy, Tokyo, Igaku Shoin, 1968) surgical opening of
lung cavities is interchangeably referred to as caverno-
tomy and speleotomy. The latter term is of Greek deriva-
tion, *spelaion* meaning cave. To me it remains an un-
forgettable revelation when in 1950 I viewed through a
scope the inner surface of a lung cavity. This maneuver
was designated by the then pioneer Maurer of Davos,
Switzerland, as speleoscopy. Prior to the era of specific
therapy, tuberculosis was considered the most common
cause of pulmonary cavity formation. Since then more
and more awareness prevails relative to a long list of
diseases which may be associated with destructive pul-
monary changes of this type. It is well to consider
pertinent causal factors as well as the mode of develop-
ment of cavities. Among the causal factors of prime
importance are: microbial agents, parasitic infestation,
sequestration of circumscribed lung areas, enzymatic
influences ("autophagism"), foreign bodies, and trauma.
The mode of development may be attributable to inhala-
tion (pathogens, chemicals), aspiration of water, partic-
ularly sea water, of material from the oropharynx or
stomach, bronchial occlusion with consequent atelec-
tasis, trauma (penetrating and nonpenetrating), spread
of infection from adjacent organs and structures, hem-
ogenous transmission of infection, entry of parasitic ova
through the digestive tract. Millions of persons in the
United States are estimated to be infected with certain
fungi, particularly with *Coccidioides immitis* and *His-
toplasma capsulatum*. Occurrence of cavities in pul-
monary mycoses has been recognized for years. In coc-
cidioidomycosis thin-walled, cyst-like cavity may be de-
tected, usually in the upper lung fields, as solitary or
multiple lesions. When bronchopneumonia is present, it
is likely to contribute to the thickness of the cavity wall
as seen in the x-ray. In others, adjacent pulmonary
infiltration may be absent. In a large group of patients
with coccidiodial cavity-abscess in the lung, Winn, W
A (Chest 54:268, 1968) noted primary cavity, abscess-
ing nodules, abscesses and residual cavities. In histo-
plasmosis the usual site of cavity is the apical and
subapical region. Greer, A E (Fungus Diseases of the
Lung, Springfield, C C Thomas, 1962) observed solitary
or multiple cavities in 36.6 percent in aspergillosis, in 22
percent in mucormycosis, in 21.4 percent in coc-
cidioidomycosis, in 11 percent in candidiasis and in 10
percent in North American blastomycosis. Of the para-
sitic diseases that caused by Echinococcus, and En-
tameba histolytica may present difficult diagnostic prob-
lems. An even greater challenge may have to be faced in
instances of primary bronchogenic carcinoma with cavi-
ty. The incidence of the latter varies from 3 to 15
percent. The cavity in these cases may be paraneoplastic
(in juxtaposition, usually distal to the tumor) or intra-
neoplastic. Paraneoplastic lung abscess is attributed to
bronchial obstruction by the tumor, with consequent
atelectasis and superimposed infection. In some cases
carcinoma may arise in the wall of a lung cyst or it may
invade a bulla located peripherally. Cavity within the
tumor is thought to develop because of insufficient blood
supply. Zorini, A O (Riforma Med 78:533, 1964) ad-
vanced the concept that enzymes produced by tumor
cells bring about tissue necrosis and liquefaction. Cavity
in metastatic carcinoma varies from 1 to 7.5 cm in
diameter. Its wall thickness measures from 0.3 to 2.5 cm.
The inner surface is likely to be scalloped. Rotte, K H
(Arch Geschwulstforsch 33:275, 1969) observed cavities
in 1.6 percent of patients with metastatic lung can-
cer. In sarcoidosis cavitation may result from aseptic
necrosis within the lesion; also, in instances of pro-
nounced fibrosis one may find thin-walled cysts similar
to emphysematous bullae. These cysts, like cavities of
other origin, may become infected with Aspergillus, with
consequent "fungus ball." Some of the other conditions in
which pulmonary cavity may be part of the clinical
picture are: a variety of severe lung infections, neo-
plasms other than carcinoma, Caplan's syndrome, rheu-
matoid disease without exposure to dust of coal or silica,
histiocytosis-X, septic embolism, rarely pulmonary
infection, foreign body in the lung, penetrating and non-
penetrating trauma. Bronchial brush biopsy is a welcome
addition to our pertinent diagnostic armamentarium.
Forrest, J V (Radiology 106:69, 1973) reports that
"bronchial brush biopsy is a highly effective method of
diagnosing cavitary lung disease, especially if the cavity
can be entered. Bronchial brush biopsy was performed
26 times in 25 patients with a wide variety of cavitary
lung diseases which could not be diagnosed by other
methods short of surgical exploration. Fifteen of 26
brush biopsies provided diagnostic material (including
12 of the 16 cases in which the brush entered the
cavity)."

Andrew L. Banyai, M.D.

60 CRISTAL, HO

CHEST, 66: 1, JULY, 1974

---

8 Friedberg HD, Schamroth L: Concealed Wenckebach
phenomenon in the left bundle branch. Br Heart J 34:370-
373, 1972
9 Langendorf R, Cohen H, Gozo EG: Observations on
second degree atrioventricular block, including new cri-
tera for the differential diagnosis between type I and
rhythm II block. Am J Cardiol 29:111-11, 1972
10 Cristal N, Gueron M, Hoffman R: 'V,-like' and 'aVF-like'
leads for continuous electrocardiographic monitoring. Br
Heart J 34:696-698, 1972