‘Supernormal’ phase in hemiblock conduction

Leo Schamroth

From the Department of Medicine, Baragwanath Hospital, and the University of the Witwatersrand, South Africa

A case is described where the basic sinus rhythm is associated with an advanced degree of left anterior hemiblock. The rhythm is complicated by atrial extrasystoles, associated with a lesser degree of left anterior hemiblock. This paradox of ‘supernormality’ is explained on a critical interplay of differential refractoriness within the divisions of the left bundle-branch.

The ‘supernormal’ phase of conduction is a short period of paradoxically improved conduction which may occur during an early phase in the cardiac recovery cycle. The situation is paradoxical, for the apparent ‘improvement’ in conduction occurs during a short critical period early in the cycle, whereas later impulses are blocked or have greater conduction delays. The term is, in a sense, a misnomer, for it does not imply that conduction is better than normal (Lewis and Master, 1924). Rather, it indicates that conduction appears to be momentarily better than that prevailing for the particular case; and the prevailing state of conduction in these cases is always one of depression. Indeed, the ‘supernormal’ phase of conduction does not occur in the normal heart, but only when conductivity is depressed. In other words, the ‘supernormal’ phase is manifested by a temporary improvement of conduction, which is ‘better’ than could be anticipated under the prevailing circumstances. This ‘improvement’ may take the form of an impulse that is conducted when it should be blocked, or an impulse that is conducted with improved conduction when a longer conduction time or delay could reasonably be expected. When considering the property of conductivity, it can generally be assumed that the earlier the impulse within the cycle, the greater its conduction delay. Any unexplained departure from this relation could, in a sense, be regarded as ‘supernormality’.

The mechanism of many forms of so-called ‘supernormal’ conduction has recently become evident through the elegant experiments and analyses of Moe, Childers, and Merideth (1968). They have shown that the ‘supernormal’ conduction does not necessarily imply ‘supernormality’, nor need it, in fact, represent a paradoxical or anomalous situation. The phenomenon can be explained on the basis of well-established physiological and anatomical principles. These are:

1) Differential refractoriness of atrioventricular nodal tissues, which may affect different longitudinal atrioventricular conduction pathways, or different horizontal or sequential atrioventricular junctional layers.

2) The presence of dual atrioventricular conduction systems.

3) Complex interference within the atrioventricular junctional tissues.

4) Fluctuating vagal discharge.

The ‘supernormal’ phase of conduction can theoretically occur at any site where there is an actual or potential conduction delay. Most reported cases have occurred with conduction through the atrioventricular node – the ‘supernormal’ phase of atrioventricular conduction. Cases have also been reported of ‘supernormality’ within a bundle-branch – the ‘supernormal’ phase of intraventricular conduction (Schamroth, 1969). The ‘supernormal’ phase of conduction has even been demonstrated in Bachmann’s bundle of the experimental animal (Childers, Merideth, and Moe, 1968).

The following presentation is an example of the so-called ‘supernormal’ phase of conduction within the divisions of the left bundle-branch, in association with left anterior hemiblock.

Case report

The electrocardiogram (Fig. 1) was recorded from a 34-year-old woman with congestive cardiomyopathy. She was fully digitalized.

The electrocardiogram (Fig. 1) shows a basic sinus rhythm. The P waves are within normal limits. The PP
FIG. 1 Electrocardiogram showing sinus rhythm associated with advanced left anterior hemiblock, and complicated by atrial extrasystoles showing a lesser degree of left anterior hemiblock.

 intervals measure 0.66 s, representing a rate of 91 beats per minute. The PR intervals measure 0.25 s, reflecting first degree atrioventricular block. The rhythm is complicated by atrial extrasystoles. The premature and abnormal P' waves of these atrial extrasystoles can be seen deforming the distal limb of the preceding T waves. This is best seen in the last beat of standard lead III where the distal limb of the preceding T wave is deformed by a distinct terminal negativity.

The atrial extrasystoles are followed by a QRS complex of slightly different configuration than the beats of the basic sinus rhythm. This is considered in greater detail below. The extrasystoles occur as: the last beat in standard lead I and standard lead II, the second, fourth, and last beats in standard lead III, the third and last beats in leads aVR and aVL, the third, sixth, and last beats in lead aVF, and the third and sixth beats in leads V1 and V6.

QRS pattern of sinus beats

The basic QRS pattern of the sinus beats is one of advanced left anterior hemiblock. The QRS duration is slightly prolonged to 0.11 s. The mean manifest frontal plane QRS axis is directed at –60 degrees, reflecting a left axis deviation due to left anterior hemiblock. This results in the following manifestations:

1) There are deep S waves in standard leads II and III and lead aVF. The S wave in standard lead III is 22 mm in depth, i.e. greater than 2 mV.

2) There is a tall R wave in lead aVL.

3) There is a relatively large initial vector directed inferiorly and to the right at about +120 degrees on the frontal plane. This results in prominent initial R waves in standard leads II and III, and lead aVF, and a prominent initial q wave in lead aVL.

The T wave axis is directed inferiorly and to the right at about +110 degrees on the frontal plane. This results in upright T waves in standard leads II and III, and lead aVF, and inverted T waves in standard lead I and lead aVL.

The left anterior hemiblock also results in a prominent terminal S wave in lead V6.

Disuction

The left bundle-branch is a relatively flat band which divides almost immediately after leaving the bundle of His into two major sweeps or radiations: the anterosuperior division and the posteroinferior division (Fig. 2). Activation normally occurs concomitantly through both divisions. Terminal activation through the anterosuperior division is downwards and to the right (illustrated as vector I in diagram A of Fig. 2). Terminal activation through the posteroinferior division is upward and to the left (illustrated as vector 2 in diagram A of Fig. 2).

FIG. 2 Diagrams illustrating the possible mechanism for the paradox of the ‘supernormal’ phase of hemiblock conduction.
Since activation occurs concomitantly through both divisions, the two forces complement each other, resulting in a mean vector which is directed downward and to the left (illustrated as vector M in diagram A of Fig. 2). If conduction is delayed or blocked within the anterosuperior division – a left anterior hemiblock – activation will occur solely or dominantly through the posteroinferior division. This results in a vector which is directed upward and to the left – a left axis deviation (diagram B of Fig. 2).

The conduction defects resulting from the hemiblocks have been highlighted by the elegant studies of Rosenbaum and his associates (Rosenbaum, Elizari, and Lazzari, 1968, 1969a, 1970; Rosenbaum et al., 1969b, c, d). They describe four types of left anterior hemiblock.

**Type I – standard type – left anterior hemiblock**

This is characterized by:

a) A mean frontal plane QRS axis directed between -45 degrees and -60 degrees.

b) There is a normal q wave in standard lead I and lead aVL.

c) The voltage of RI, SII, and SIII is moderate. The S wave in standard lead III has an arbitrary upper limit of 1.5 mV.

d) The left chest leads – V5 and V6 – have relatively deep S waves.

e) The normal initial q waves in the left chest leads tend to disappear.

In the type II left anterior hemiblock, the electrocardiographic features of the standard type are modified by a horizontal heart position. In the type III left anterior hemiblock, the electrocardiographic features of the standard type are modified by a vertical heart position. These types are not germane to this presentation and will not be considered further here.

**Type IV left anterior hemiblock**

This is essentially an augmented or more advanced form of left anterior hemiblock. The voltage criteria described in type I are increased. Standard leads II and III have very deep S waves; the S wave in standard lead III exceeds 1.5 mV. This is shown in this case (Fig. 1).

The atrial extrasystoles are associated with the type I or standard type of left anterior hemiblock. The QRS voltage is moderate and much diminished when compared with the sinus beats. The S wave in standard lead III does not exceed 1.5 mV. The initial r waves are still prominent in standard leads II and III, and in lead aVF. The initial q wave is still prominent in lead aVL. The QRS complexes of the atrial extrasystoles tend to vary somewhat depending on the degree of prematurity. Thus, the first extrasystole (third beat) in lead V6 shows the remnant of a much diminished terminal s wave. The second extrasystole (sixth beat) in this lead has no terminal s wave at all. The QRS deviation is only 0.08 s as compared with the 0.11 s of the sinus beats. The T wave axis has deviated to the sagittal plane.

The principal features distinguishing the higher grade of left anterior hemiblock (type IV) from the standard type (type I) are thus clearly evident in this case. They are:

1) An increase in QRS duration.

2) An increase in QRS magnitude, particularly the S waves in standard leads II and III and lead aVF.

3) The development of a terminal S wave in lead V6.

**Mechanism of ‘supernormality’**

It is evident that the atrial extrasystoles which are premature and hence must encounter a greater degree of refractoriness in the conducting tissues are paradoxically conducted with a lesser degree of left anterior hemiblock. It is tempting to apply the term ‘supernormal phase’ to this paradox, yet consideration of differential refractoriness of the conducting tissues provides a more physiological explanation.

In left anterior hemiblock, conduction is blocked or delayed within the anterosuperior division, so that the activation pathway travels preferentially through the posteroinferior division, and the territory normally activated by the left anterosuperior division is activated dominantly by the activation front passing through the posteroinferior division.

![Diagram](http://heart.bmj.com/)

**Fig. 3** Diagram illustrating the possible mechanism for the paradox of the ‘supernormal’ phase of hemiblock conduction. P = posterior division; A = anterior division; black shading = absolute refractory period; stippled shading = varying degrees of partial refractoriness.
This would occur, for example, if the anterosuperior division has a much longer conduction time. The principle is depicted in diagram B of Fig. 2 and by impulse 1 of Fig. 3. The posteroinferior division has an arbitrary conduction time of X whereas the anterosuperior division has a much longer conduction time of 5X. Conduction, therefore, occurs solely through the posteroinferior division. An atrial extrasystole, because of its prematurity, encounters a greater state of refractoriness within the posteroinferior division. This is arbitrarily depicted as a conduction time of 3X (diagram C of Fig. 2 and impulse 2 of Fig. 3). This permits a greater synchrony in conduction between the 2 divisions. In other words, there is some conduction through the anterosuperior division and hence a lesser degree of left anterior hemiblock. Theoretically, with even greater prematurity (as depicted diagrammatically by impulse 3 of Fig. 3), conduction time would be equal, and thus occur synchronously through both divisions, and the hemiblock would disappear.

References


Requests for reprints to Professor Leo Schamroth, Department of Medicine, Medical School, University of the Witwatersrand, Hospital Street, Johannesburg, South Africa.