REVIEWS

Vagaries of acceleration dependent aberration

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The earliest electrocardiogram illustrating bizarre intraventricular conduction of supraventricular impulses was published by Sir Thomas Lewis in 1910.¹ In a subsequent communication Lewis labelled this form of abnormal conduction "aberration" and the ventricular complexes as "aberrant beats."² In that same paper he also proposed a mechanism for the aberration, namely "...disturbances of conduction in the smaller branches of this system and it is held that definite branches are affected in this manner, though these branches cannot be identified at the present time". The underlying milieu for aberration proposed by Lewis stood the tests of further observation and experimentation. Altered, asynchronous conduction has been demonstrated for all segments of the His-Purkinje system as well as the Purkinje-myocardial junctional areas and, rarely, as a result of preferential atrioventricular and, perhaps, His bundle conduction. The mechanisms responsible for initiating the conduction alterations are, however, even more complex as attested to by the vast number of published

reports, both basic and clinical, on aberrancy that has been generated since Lewis' initial observations.

In 1983, on the occasion of the 75th anniversary of Sir Thomas Lewis' description of aberration, Dr Dennis Krikler, Editor of the *British Heart Journal*, invited an editorial on that subject.³ As our contribution to this Festschrift issue of the *British Heart Journal* honouring Dr Krikler, we have elected to continue that discussion and focus on the vagaries of acceleration dependent aberration. The subject is particularly well suited to Dr Krikler's long time interest in the history of cardiology and, in particular, in tracing the development of concepts in electrophysiology.

We have collated scattered reports on eccentric forms of acceleration dependent aberration and on mechanisms that could explain the vagaries proposed in the hope that further interest in the mechanisms underlying aberration will be stimulated. The material comes from our files and our earlier publications.

Background and observations

In 1913, Lewis published two electrocardiograms illustrating the disappearance of bundle branch block with slowing of the heart rate (fig 1).⁴ Although the tracings were recorded on different days, they probably are the earliest example of acceleration dependent aberration.

The classic manifestation of acceleration dependent aberration is the appearance of aberration at a critical cycle length reached in the course of an increase in heart rate, and normalisation of conduction with slowing of the heart rate to the point that the RR cycle is again longer than that at which aberration was initiated (fig 2).⁵

While the predictability of the relation between rate and aberrancy is characteristic for acceleration dependent aberration, there are exceptions to this heart rate dependency that we will refer to as vagaries or eccentric forms of acceleration dependent aberration, an example of which also was first reported by Lewis.⁶ The vagaries will be divided into two groups: *Group 1*—unexpected normalisation of conduction when aberration would be expected to continue—and *Group 2*—unexpected appearance or persistence of aberration under conditions in which it would not be expected. Examples under each group are presented and discussed below.

Fig 18.

Fig. 18. Curves taken from the three leads in a case of aortic disease during a febrile attack. They show defective conduction along the right division of the auriculo-ventricular bundle (see Fig. 16).

Fig. 19.

Fig. 19. Curves from the same patient, taken a day later and during the subsidence of the fever. The ventricular portions of the curves have changed profoundly; there is now no evidence of bundle defect, but of preponderance of the left ventricle.

Figure 1 Earliest example of acceleration dependent aberration. (Reproduced from Lewis T. *The mechanism and graphic registration of the heart beat*. London: Shaw and Sons, 1920:125.)

Figure 2 Acceleration dependent aberration with unexpected (1) persistent aberration at cycles longer than the critical cycle due to transseptal concealed conduction shortening the bundle branch to bundle branch interval to a critical cycle of 700 ms and (2) absence of shortening of the critical cycle when compared with the immediately preceding cycle. (Reproduced from Fisch C, Zipes DP, McHenry PL. Rate dependent aberrancy. Circulation 1972;48:714 by permission of the American Heart Association, Inc.)

Figure 3 Relation of the coupling interval of the normal and aberrant QRS (abscissa) to the preceding RR cycles (ordinate). The presence of paradoxically normal QRS at coupling intervals of 0.40 to 0.50 s is dependent on the preceding RR measuring approximately 0.50 to 0.80 s. This dependency on duration of the preceding cycle suggests a shift of the supernormal period to the right with the longer preceding RR intervals and the location of the supernormal period at 0.40 to 0.50 s after the QRS. (Reproduced with permission from Fisch C. Electrocardiography of arrhythmias. Philadelphia: Lea and Febiger. 1990:243,244.)

GROUP 1
Normalisation with abrupt shortening of the cycle length

One of the most common vagaries of acceleration dependent aberration is normalisation of intraventricular conduction at cycles shorter than those of the aberrantly conducted complexes. This occurs with an abrupt shortening of the RR interval such as that which may be seen with atrial fibrillation⁷ (figs 3 and 4), single atrial premature complexes (fig 5), runs of atrial premature complexes, or 3:2 atrioventricular block.

The most likely mechanism responsible for unexpected normalisation of conduction with abrupt shortening of RR interval is conduction during the supernormal period of His-Purkinje recovery.^{7,8} This is supported by the fact that there is a constant, or rarely a shorter, H-V interval,⁹ and that the coupling of the normally conducted impulses, in most cases, varies directionally with the preceding bundle branch to bundle branch interval the duration of which may be dependent on the presence or absence of concealed transseptal conduction from the contralateral bundle (figs 3 and 5).^{8,10-12} In a unique example in which the His bundle electrogram was recorded, the H-V interval of the supernormally conducted impulse remained normal, or was shorter than the H-V of the aberrantly conducted impulses (fig 4).⁹

Equal delay of conduction in the bundle branches has been suggested as an alternative to supernormality as an explanation for unexpected normalisation of the QRS at short cycles. The finding that would support this mechanism for paradoxical normalisation would be prolongation of the H-V interval recorded simultaneously with normalisation of the intraventricular conduction. Such observations have, as yet, not been reported. Prolongation of the PR interval commonly accompanies the normal QRS, but this may be an expression of atrioventricular nodal rather than bundle branch prolongation.

Normalisation during an accelerating tachycardia

Occasionally intraventricular conduction becomes normal after several cycles of acceleration dependent aberration.

While the initial aberration in fig 6 represents acceleration dependent aberration due to bundle branch refractoriness, normalisation of conduction is most likely due to a gradual shortening of the bundle branch refractory period over several cycles. This may be a normal adaptation of action potential to rate or possibly a result of an increase in the concentration of circulating catecholamines. Increased speed of conduction in the contralateral bundle branch or an equal delay in both bundles also are possible, but, as discussed above, these are unlikely mechanisms for normalisation of conduction.

On rare occasions, aberration disappears in the course of a gradual acceleration of heart rate, a phenomenon that has been observed during stress tests (fig 7). In such instances a 2:1

Figure 4 Normalisation of QRS at paradoxically short cycles with a slight shortening of the H-V interval supporting supernormal His-Purkinje conduction as the cause of the normalisation. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias*. Philadelphia: Lea and Febiger, 1990:241.)

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Figure 5 Atrial premature complex with a paradoxically normal QRS is followed by a normally conducted sinus impulse whereas the atrial premature complex with aberration is followed by an aberrant QRS. The latter is most likely due to concealed transseptal conduction of the atrial premature complex shortening the bundle branch to bundle branch interval resulting in acceleration dependent aberration. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias*. Philadelphia: Lea and Febiger, 1990:233.)

bundle branch block may precede or follow normalisation of conduction.

To the best of our knowledge the record reproduced in fig 8 is the only known example of paradoxical normalisation of conduction after a long preceding cycle when, according to the Ashman phenomenon, aberrant conduction would be expected^{13 14} rather than the normal conduction noted in this tracing. It is most likely that prolongation of the transmembrane action potential after the long pause fortuitously allows for conduction during the supernormal period of recovery.

Intermittent normalisation and aberration

Bundle branch block and normal conduction may be recorded intermittently during a regular rhythm. The ratio of bundle branch block to normal conduction may vary from a sporadic bundle branch block to 2:1 or higher ratios of bundle branch block to the normally conducted QRS complexes (figs 9 and 10).

The mechanisms proposed to explain the intermittent bundle branch block include: (a) small changes in cycle length not recognisable in the electrocardiogram, (b) block of

the impulse above the bundle branch lesion responsible for the bundle branch block allowing for a prolonged recovery time,¹⁵ (c) Wenckebach conduction in the bundle branch,¹⁶ (d) deceleration and acceleration dependent bundle branch block,¹⁷ and (e) supernormal conduction.⁹ The two latter mechanisms may be facilitated by concealed transseptal conduction^{11 18} (fig 10) that alters the duration of the bundle branch to bundle branch interval, the duration of the action potential, and thus the relative position of the supernormal period of recovery.

Phase 4 depolarisation has been suggested as a mechanism for 2:1 bundle branch block. With phase 4 depolarisation, it has been postulated that after a normal QRS the left bundle branch to left bundle branch interval is relatively "long" and thus results in bundle branch block. In the presence of the left bundle branch block, an impulse conducts along the right bundle branch, traverses the septum, and reaches the left bundle branch after some delay. The result is a "short" left bundle branch to left bundle branch interval, dissolution of phase 4 depolarisation, and as a result normal conduction.¹⁷ Although electrocardiographic observations tend to support phase 4 depolarisation as a possible mechanism of deceleration dependent aberration,^{19 20} some believe, based on cellular observations, that deceleration dependent aberration is the result of "complex oscillatory changes in membrane properties of depressed bundle branch Purkinje fibres during diastole."²¹

Another mechanism that could explain 2:1 bundle branch block is that block above an area of injury occurs so that the left bundle branch to left bundle branch interval encompassing the normal QRS is equal to two sinus cycles, allowing for bundle branch recovery and normal conduction, or, alternatively, it is also possible that delay of conduction above an area of injury could allow for recovery of excitability of an injured area and/or other electrophysiological parameters necessary for normal conduction. This mechanism resembles the gap phenomenon. For these two latter mechanisms to be possible, the bundle branch must not be activated retrogradely. Should retrograde conduction take place, prolonged recovery is no longer possible.

GROUP 2

Onset of aberrant conduction without a recognisable shortening of RR interval

This is a common phenomenon (fig 2). While

Figure 6 Normalisation of acceleration dependent aberration without any demonstrable change in heart rate. The most likely mechanism is shortening of the refractory period in response to the tachycardia. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias*. Philadelphia: Lea and Febiger, 1990:69.)

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Figure 7 Paradoxical normalisation of the QRS in course of acceleration of the heart rate during a stress test. Mechanisms suggested as an explanation for the normalisation include increase of sympathetic activity, catecholamine release, supernormal conduction, and physiological shortening of the refractory period.⁹

there is no doubt that the aberration is a function of acceleration of the heart rate, this may become obvious only when long records are available to allow the gradual shortening of the cycle length to be recognised. Failure to appreciate shortening to the “critical” aberrancy-inducing RR interval reflects a limitation of electrocardiographic technology; aberration may be a function of small changes of the cycle, often measured in milliseconds, and such changes may not be recognisable in the surface electrocardiogram. It is also possible under certain conditions that with gradual acceleration of the heart rate the refractory period inappropriately lengthens, resulting in conduction delay or block without the need for shortening of the “critical” cycle.

Figure 9 Intermittent normalisation of conduction during a regular rhythm with acceleration dependent aberration. The fact that the aberration represents acceleration dependent aberration is proved by normalisation of conduction with an 80 ms prolongation of the cycle in lead V4.

Figure 8 Paradox of normal conduction after a long RR cycle when according to the Ashman phenomenon aberration would be expected. The most likely explanation is supernormal conduction made possible by the prolonged recovery period with a shift of the supernormal period to the right. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias*. Philadelphia: Lea and Febiger, 1990:236.)

Persistence of aberration at a cycle longer than the “critical” cycle

This phenomenon also is a frequent finding in clinical electrocardiograms (fig 2). The most likely mechanism is concealed transseptal conduction that shortens the bundle branch to bundle branch interval. As a result of transseptal conduction the actual bundle branch to bundle branch interval is shorter than the manifest QRS to QRS interval (fig 2). Not only is the bundle branch to bundle branch interval altered but the duration of the subsequent refractory period also is changed. Transseptal conduction has been shown experimentally in the dog^{10 11} and in humans.^{12 22 23} Whereas concealed transseptal conduction alone may shorten the bundle branch to bundle branch cycle sufficiently to result in acceleration dependent aberration and its persistence, often it combines with other mechanisms to propagate aberrancy at cycles longer than the “critical” cycle. In some patients the discrepancy between the “critical” cycle and cycle at which normal conduction resumes may be on the order of 210 ms, an interval longer than could be explained simply on basis of the transseptal conduction.²⁴

Alternate mechanisms for persistence of aberration may be “fatigue” and overdrive suppression, both of which are a function of increased rates. “Fatigue” is a concept proposed by Shearn and Rytdand in 1953 to explain the unexpected persistence of aberration with slowing of the rate⁵ and is a descriptive phenomenon, the cellular basis for which is unclear. It is an attractive and logical concept that suggests that a persistently accelerated rate, sufficiently rapid to induce acceleration dependent aberration, alters the electrolyte milieu and that such alterations may persist over several cardiac cycles, some longer than the “critical” cycles at which aberrancy was initiated.

Overdrive suppression of conduction, first demonstrated by Scherf in the dog,²⁵ is manifest by the appearance of bundle branch block upon cessation of a rapid heart rate or, on occasion, after a single early excitation or stimulus (fig 11). Overdrive suppression of bundle branch conduction is in many ways

Figure 10 2:1 Left bundle branch block followed by persistent aberration, the result of a slight acceleration of the heart rate. TC, transseptal concealment.

similar to overdrive suppression of atrio-ventricular conduction. The duration of suppression depends on the rate of the overdriving impulse: the higher the rate the longer the recovery time. Overdrive suppression after ventricular tachycardia is manifest by a gradual normalisation of bundle branch conduction in the face of acceleration of the sinus rate (fig 11).²⁶ It is a phenomenon which, unlike “fatigue”, could explain aberration after a single early excitation or stimulus. The exact electrophysiological basis responsible for overdrive suppression is uncertain.

Unexpected persistence of aberration induced by the Ashman phenomenon

A relatively frequent observation first recorded by Lewis⁶ is the persistence of aberration once initiated by sudden prolongation of the refractory period in response to a long cycle—the Ashman phenomenon (figs 12 and 13). The aberration persists at cycle lengths identical to those manifesting normal conduction.

Whereas the Ashman phenomenon is responsible for aberration of the first complex,^{13 14} persistence of the aberration may be due to delayed concealed transseptal conduction from the left to the right bundle branch resulting in shortening the right bundle branch to right bundle branch interval and thus initiation of acceleration dependent aberration. A constant heart rate and a fixed relation of transseptal

conduction to anterograde conduction in the contralateral bundle branch are critical for perpetuation of the aberration.

Appearance of aberration during a regular heart rate

Acceleration dependent aberration may appear after several normally conducting impulses during a regular heart rate (fig 14). The appearance of aberration without a demonstrable change in the heart rate is most likely due to gradual prolongation of either voltage or the time dependent refractoriness (fig 15). Inappropriate restitution of ionic concentrations may contribute to the time dependent refractoriness and may to some extent also explain “fatigue” and overdrive suppression (see above).

Aberration secondary to gradual delay of bundle branch conduction

Gradual delay of bundle branch conduction (fig 16), evidenced by a gradual widening of the QRS, has been equated with type I, Wenckebach, second degree bundle branch block.^{16 27} Similarly, intermittent normalisation of bundle branch conduction may represent concealed Wenckebach conduction (fig 9). Once the delay of bundle branch conduction reaches a critical point and a complete bundle branch block is inscribed, any further delay of bundle

Figure 11 Overdrive suppression of bundle branch conduction by ventricular tachycardia. (Reproduced from Fisch C. Bundle branch block after ventricular tachycardia: a manifestation of “fatigue” or “overdrive” suppression. *J Am Coll Cardiol* 1984;3:1562 by permission of the American College of Cardiology.)

Figure 12 Aberration initiated by the Ashman phenomenon and perpetuated by concealed transseptal conduction that shortens the bundle branch to bundle branch interval thus initiating acceleration dependent aberration. Solid bar denotes right bundle branch refractory period, and broken and dotted lines show the right and left bundle branch block respectively with transseptal conduction of the latter. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias: from deductive analysis to laboratory confirmation—twenty five years of progress*. J Am Coll Cardiol 1983;1:206.)

V3

Figure 13 Right bundle branch initiated by the Ashman phenomenon and perpetuated by acceleration dependent aberration with the obligatory shorter bundle branch to bundle branch interval being the result of concealed transseptal conduction. The paradox of normal conduction of the atrial premature complex is probably due to conduction during the supernormal period (Reproduced with permission from Knoebel SB, Fisch C. *Concealed conduction*. In: Fisch C, ed. *Complex electrocardiography I*. Philadelphia: FA Davis, 1973.)

branch conduction cannot alter the QRS duration and thus will go unrecognised.

Aberration and the “crossover” phenomenon

The paradox of left bundle branch block at shorter coupling intervals and of right bundle branch block at longer coupling intervals (fig 17) has been recognised for years.²⁸ It is often referred to as the “crossover” phenomenon and reflects the paradox of a longer refractory period of the left bundle branch at short coupling intervals and a longer refractory period of the right bundle branch at longer coupling intervals. The mechanism responsible for this phenomenon is unclear but is probably secondary to injury.²⁹

Aberrant conduction after premature complexes or pauses

Aberrant conduction may terminate a compensatory pause after an atrial premature complex while, paradoxically, normal conduction may follow a shorter compensatory pause induced

Figure 14 Appearance of a gradually increasing aberration without any demonstrable change in heart rate is recorded in the top trace. The Ashman phenomenon and acceleration dependent aberration with a progressive widening of the QRS is illustrated in the bottom trace. The most likely mechanism of the acceleration dependent aberration is an inappropriate, gradual prolongation of the refractory period. (Reproduced from Fisch C, Zipes DP, McHenry PL. *Rate dependent aberrancy*. Circulation 1973;48:714 by permission of the American Heart Association, Inc.)

Figure 15 Time dependent refractoriness is illustrated by failure of the stimulus to elicit a response after full recovery of transmembrane action potential. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias*. Philadelphia: Lea and Febinger, 1990:60.)

by a ventricular premature complex (fig 18). The most likely mechanism for aberration after the atrial premature complex is concealed conduction of the atrial premature complex into the bundle branch thus shortening the bundle branch interval as discussed above. A possible but unlikely mechanism for aberration after an atrial premature complex is diastolic depolarisation. Phase 4 depolarisation is, however, incompatible with bundle branch block during sinus rhythm at considerably shorter cycles, unless one accepts the presence of acceleration and deceleration aberration.

In the presence of multiform ventricular premature complexes a compensatory pause following one pattern of ventricular premature complex may be terminated with aberration while the compensatory pause of a different form of ventricular premature complex may terminate with a normally conducting QRS (fig 19). The most probable explanation for such observations is that concealed conduction into the bundle branch with shortening the bundle branch interval occurs with one type of ventricular premature complex and not the other.

Rarely, intermittent supraventricular tachycardia is interrupted by pauses that are followed by aberrant conduction at a rate identical with that of the normally conducted complexes (fig 20). The mechanism for the aberration in such cases is unclear. One can postulate that the first aberrant QRS after the pause is due to diastolic depolarisation and that propagation of aberration is due to concealed transseptal conduction from the contralateral bundle branch thus shortening the bundle branch to bundle branch interval.

Figure 16 Progressive delay of left bundle branch conduction suggesting type I, Wenckebach, bundle branch conduction.

Figure 17 This figure shows (1) right bundle branch acceleration dependent aberration (2) paradox of normal conduction of atrial premature complexes, and (3) APC with left bundle branch block conduction reflecting the crossover of the refractory periods of the right and left bundle branch.

Acceleration dependent aberration appearing at slow heart rates

One of the most intriguing vagaries of acceleration dependent aberration is its appearance at extremely slow heart rates. While the vast majority of patients manifest acceleration dependent aberration at cycles longer than 750 ms, aberration at intervals as long as 1000 ms and occasionally as long as 1800 and 2000 ms have been recorded (fig 21). Although significant prolongation of the action potential duration has been recorded in abnormally functioning Purkinje fibres,³⁰⁻³² aberration at such strikingly slow rates cannot be readily explained by simple prolongation of recovery of transmembrane potential (voltage dependent refractoriness). Time dependent refractoriness can be postulated (fig 15). It is also possible that a delay or block of conduction in

Figure 19 Concealed conduction of ventricular premature complex 2 into left bundle branch shortens the bundle branch to bundle branch interval and consequently the compensatory pause is terminated by a sinus impulse with left bundle branch block. Ventricular premature complex 1 does not conceal into the left bundle branch, is followed by a left bundle branch to left bundle branch interval that is longer than that following ventricular premature complex 2, and is therefore terminated with a normal QRS. (Reproduced from Fisch C, Zipes DP, McHenry PL. Rate dependent aberrancy. *Circulation* 1973;48:714 by permission of the American Heart Association, Inc.)

Figure 18 Concealed conduction of the atrial premature complex into the left bundle branch with shortening of left bundle branch to left bundle branch interval is responsible for the paradox of left bundle branch block after pauses longer than the pauses that follow ventricular premature complexes and which are terminated by a normal QRS. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias*. Philadelphia: Lea and Febiger, 1990:81.)

the His-Purkinje system can result because of an abnormal state of the His-Purkinje system, with a reduction of diastolic potential,³³ coupled with gradual diastolic depolarisation and slowing of the upstroke velocity and a shift in threshold toward zero. Injury of the His-Purkinje system may cause such changes.²⁹

Another mechanism for aberration at unexpectedly long cycles may be concealed conduction into a bundle branch block. This has been observed with atrial and ventricular premature complexes. Similarly, during atrial fibrillation, persistence of aberration at long cycles may be due to concealed conduction of fibrillatory waves into a bundle branch as mentioned above.

Discussion

While a strong case can be made in favour of one or another mechanism for aberration in any given setting and some have been documented experimentally, the fact remains that most explanations still are based in deductive reasoning and extrapolations from the behaviour of the myocardium as reflected in the electrocardiogram by the P and QRS and, occasionally, the His-bundle electrocardiogram. It is almost certain that some of the conclusions so arrived at are in error. Many of the basic, cellular processes responsible for aberration remain to be elucidated. More sophisticated techniques for recording activation sequences and local action potential characteristics in the human will be required to allow more precise assessment of the mechanism for aberrancy under physiological and pathological conditions. However, for the purpose of giving structure to the observations presented above, it may be helpful to review some concepts about aberrancy that have developed since Lewis's original observations.

There is strong evidence indicating that acceleration dependent aberration is a marker of heart disease. While the heart disease may not be clinically evident initially, clinical signs

Figure 20 Aberration follows paradoxically longer cycles and continues at a rate identical to that of the normally conducted complexes.

of heart disease usually appear with time. Evidence supporting the association between acceleration dependent aberration and heart disease includes: (a) the high prevalence of heart disease in association with the acceleration dependent aberrancy, (b) the appearance of aberrancy at surprisingly slow rates, (c) the predominance of left bundle branch block aberrancy, (d) the frequent coexistence of acceleration with deceleration dependent aberration, the latter being a sign of heart disease, (e) the occasional appearance of acceleration dependent aberration after several equal RR cycles indicating inappropriate refractory period stability and/or abnormal electrophysiological properties, (f) independence from the duration of the preceding cycle (fig 22).

Acceleration dependent aberration differs from physiological or “expected” forms of aberration as follows: “expected” aberration is seen in normal hearts; “normal” aberration is elicited by a premature impulse or stimulus and rarely, if ever, by a gradual acceleration of the rate because atrioventricular refractoriness will usually exceed His-Purkinje refractoriness thus precluding aberration; physiological aberration is nearly always of right bundle

Figure 21 Aberration at cycle lengths varying from 920 to 1250 ms. The fact that aberration is acceleration dependent is documented by normalisation of conduction after the long compensatory pause induced by the blocked atrial premature complex. Aberration at such long cycles may be due to time dependent refractoriness, to injury,²⁹ or a combination of the two. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias*. Philadelphia: Lea and Febiger, 1990:62.)

Figure 22 Relation of the coupling interval (abscissa) to preceding cycle length (ordinate) of the right and left bundle branch block aberration. Whereas left bundle branch block is independent of the duration of preceding cycle, right bundle branch is dependent on the duration of the preceding cycle. The right bundle branch block follows long preceding cycles in keeping with the Ashman phenomenon. It is possible that the considerable shorter coupling interval of the left bundle branch as compared with that of the right bundle branch reflect the crossover phenomenon of the refractory periods of the bundle branch. (Reproduced with permission from Fisch C. *Electrocardiography of arrhythmias*. Philadelphia: Lea and Febiger, 1990:66.)

branch block pattern; coupling of a stimulus that elicits physiological aberration is quite short so that the stimulus occurs during the recovery of the action potential and the aberration can be readily explained by voltage dependent refractoriness; and the presence or absence of aberration is dependent on the duration of the preceding cycle length (fig 22). Because of the differences between aberration that may be present in the normal heart and acceleration dependent aberration which is nearly always an expression of an abnormal state, the two forms should be considered to be different phenomena if interpretation of the data dealing with aberration is to be meaningful.

Proposed mechanisms for acceleration dependent aberration as presented above include: (a) prolongation or failure of the action potential to shorten appropriately in response to an increase in heart rate, creating the milieu for voltage dependent refractoriness,³⁴ (b) refractoriness persisting after completion of repolarisation (time dependent refractoriness) or, (c) myocardial injury involving one of the bundle branches or fascicles resulting in partial depolarisation of cells within the affected bundle creating conduction delay or block that is independent of refractoriness.²⁹ Local action potential alterations also may electrotonically affect cells more proximal in the conduction system creating slowed conduction or block at that level.

While it is possible that in pathological states voltage or time dependent refractoriness may be altered sufficiently to be responsible for acceleration dependent aberration, and, while functional conduction disturbances have been

shown to be favoured at low basic heart rates,³⁵ it is unlikely that either could account for aberration at cycle lengths of 1000 ms or longer, a not infrequent finding in acceleration dependent aberration. In such instances, disturbances of conduction are more likely to be secondary to alterations in action potential characteristics such as phase 4 depolarisation, slowing of the upstroke of phase 0, or reduction of threshold potential due to electropathology. A unifying concept could, therefore, be that injury to the conduction tissue, at diverse levels, is the basic defect that underlies acceleration dependent aberration.

When the vagaries of acceleration dependent aberration are under consideration still other mechanisms or combinations of mechanisms need to be invoked. Examples of these are presented above and include: (a) shortening of the bundle branch to bundle branch cycle without change in the manifest QRS to QRS cycle secondary to concealed transseptal conduction^{12 17 23} or concealment of atrial and ventricular premature complexes into the bundle branches, (b) local injury enhanced by acceleration of the heart rate and a resultant depression or block of conduction manifesting as fatigue,⁵ or overdrive suppression,^{25 26} (c) conduction during the supernormal period of recovery,⁹ (d) block above the area of injury allowing for bundle branch recovery, (e) "crossover" of the refractory period duration of right and left bundles,^{28 36} (f) equal prolongation of bundle branch conduction, (g) diastolic (phase 4) depolarisation,^{17 33} or, (h) Wenckebach block in a bundle branch.^{16 27}

Aberration under normal physiological conditions, acceleration dependent aberration and its vagaries, and the similarities and differences between the various "normal" and "abnormal" forms of aberrancy provide a model for understanding electrophysiological functioning that is unequalled by any other concept. Even if basic cellular and intracellular mechanisms for electrophysiological alteration are eventually totally understood there will continue to be the need for an integrated approach to the conducting system and its functional vagaries. Knowledge of ion channel functioning under a wide variety of intervention and injury, action potential characteristics in response to such changes, the effects on refractoriness, cell-to-cell communications, and the effects of all of these on conduction patterns is essential if we are to be able to delineate mechanisms for aberrancy with more precision that has been possible to date. There is a large body of experimental data about the cellular electrophysiological properties underlying aberration. A systems approach is needed to make these observations applicable to the human.

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