Aberrant Ventricular Conduction of Escaped Beats

Preferential and Accessory Pathways in the A-V Junction

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Escape beats recorded in clinical electrocardiograms sometimes differ in contour and QRS duration from conducted beats, and this in spite of their having a relatively short cycle length. This suggests a location of the subsidiary pacemaker above the bifurcation of the common A-V bundle. While this, unlike other types of aberrant ventricular conduction, cannot be explained on a functional basis, recent investigations on the structure of the normal A-V junction suggest that "para-specific" A-V connections may act as preferential pathways to the ventricles in some A-V nodal escape beats. Difficulties and guides in differentiating A-V nodal from ventricular escapes under such circumstances are pointed out. On the basis of clinical, electrocardiographic and anatomic facts such a normal preferential A-V conduction must be distinguished from an abnormal accessory A-V conduction causing the pre-excitation (Wolff-Parkinson-White) syndrome. However, the two may occur in association.

When consecutive atrial impulses fail to stimulate the ventricles at an appropriate rate, subsidiary, ordinarily subdued, cardiac pacemakers will come into operation. First to escape under such circumstances are centers in the A-V junctional tissues because this area has the ability to create impulses at a rate faster than pacemakers in the ventricles. Centers below the bifurcation of the common bundle will come into action when formation of impulses in the A-V junction is depressed, or conduction to the ventricles of such impulses is prevented by interference or block. It is generally accepted that the distinction between the two types of ectopic beats can be made in the electrocardiogram on the basis of the length of their cycle and the contour of the QRST. Impulses arising in junctional tissues above the bifurcation, in a supraventricular pacemaker, are expected to take over ventricular activation promptly, at rates between 40 and 50, and to produce ventricular complexes resembling beats of atrial origin in all regards. Impulses originating below the bifurcation, in a ventricular pacemaker, appear tardily, at rates of 30 or less, and give rise to aberrant ventricular complexes with prolonged QRS and abnormal ST-T contours.

In the following report several clinical records are presented showing escaped beats during various abnormal rhythms in which the escape origin cannot be easily established on the basis of the above criteria. These are examples selected from many other instances encountered in the course of the past years in routine tracings as well as during the study of simple and complex forms of disturbances of rhythm. Yet, despite this common occurrence, hardly any mention of the problem can be found in the literature. It is the intent of this report: (1) to point out the varieties of contour deviations occurring in escapes of apparently supraventricular origin; (2) to call attention to difficulties and possibilities in determining, under such circumstances, the site of the ectopic subsidiary pacemaker; (3) to attempt an explanation of ventricular aberration in supraventricular escapes by a mechanism which is comparable to, yet different from, that assumed to be in operation in the pre-excitation syndrome.

Material and Methods

The material comprises selected electrocardiograms of 11 different cases in whom escaped beats were recorded as a consequence of various types of
Fig. 1. Typical nodal escapes occurring during sinus bradyarrhythmia, and subsequent to a nodal premature systole with retrograde A-V and aberrant ventricular conduction.

In the first half of the record broad and diphasic P waves are seen indicating irregular and slow (38-46) activity of the sinus node and an abnormal spread of its impulse through the atria. These impulses are conducted to the ventricles at a P-R of 0.20 second, except the second which interferes with one from the A-V node escaping at a cycle length of 1.44 seconds (corresponding to a rate of 42). This nodal beat, apart from the short P-R distance, resembles precisely the conducted sinus beats in contour and QRS duration. The fifth beat of the record is a premature systole of bizarre contour. It is followed by a pause during which a P wave fails to appear at the anticipated time; instead a much earlier, inverted P is seen within the S-T of the premature systole, obviously caused by retrograde spread of the ectopic impulse to the atria. The last two ventricular beats are nodal escapes corresponding in timing and shape to the second beat of the record, and during this time no sign of atrial activity is seen. The sinus node appears to be in a depressed state subsequent to its premature discharge by the retrograde ventricular impulse, but it is possible that retrograde P waves are hidden within the QRS of the two nodal beats.

At first glance, the contour of the premature systole suggests its origin in the ventricles. However, one would expect the subsequent nodal escape interval to be prolonged, as compared to the other escapes, by the retrograde conduction time of the ventricular impulse to the subsidiary A-V nodal pacemaker. Since such prolongation does not occur, it must be concluded that the premature systole takes its origin within, or close to, the point of origin of the escaped beats, that is within the A-V node, and its bizarre contour is caused by aberrant ventricular conduction due to its early appearance in the cycle, when some part of the ventricles is still in a refractory state. The close similarity of sinus and escaping nodal beats on the other hand indicates that both types of impulses use the same pathways in activating the ventricles—as is ordinarily the case.

disturbances of impulse formation or impulse conduction. These records are grouped in figures 1 through 5 according to the type and/or duration of QRS of the ectopic beats. A detailed analysis and interpretation of each tracing is presented in the respective legends and the reasons are there outlined why the origin of the escapes was ascribed to areas above, or below, the bifurcation of the common A-V bundle, or could not be determined. Thus, figure 1 illustrates typical nodal escapes and their close similarity to conducted sinus beats in contrast to aberrant ventricular conduction of a nodal beat occurring prematurely. In figure 2 four cases are reproduced in which a supraventricular origin of the escaped beats was postulated on the basis of their normal QRS duration, in spite of slight or marked differences in contour from that of the conducted supraventricular beats. Figure 3 exemplifies the problem in determining the origin of escaped beats when promptness of their appearance does not conform with a marked contour aberration and a prolonged QRS duration. Identification of a ventricular escape on the basis of the occurrence of ventricular fusion beats is demonstrated in figure 4 in two instances, one of which shows evidence of parasystolic action of the ectopic pacemaker. Finally, in figure 5, a case of ventricular pre-excitation, escaped beats are seen which differ in shape from both the normal and anomalous ventricular complexes and suggest that various pathways to the ventricles may be available to impulses crossing the A-V junctional tissues from above and those arising within them. The physiologic background and new anatomic data to be considered in the explanation of aberrant ventricular conduction of escape beats are pointed out below.

Comment

Ordinarily, aberration in the contour of QRST complexes is the electrocardiographic manifestation of the failure of supraventricular impulses, traversing or initiated in the A-V junction, to complete ventricular activation via ordinary conduction pathways because part of these pathways is in a state of partial or complete refractoriness at the time of arrival of the impulse. With normal or slow heart rates this has to be ascribed to an abnormal prolongation of the refractory period, a block, somewhere in the system of the two bundle branches. When aberration of the QRST complexes is limited to beats with a short cycle it may be due entirely to the operation of the normal refractory period in some parts of the conduction system. In about 85 per cent of the beats affected by the
Fig. 2. Escapes with aberrant contour but without QRS prolongation. The four strips are records from different patients.

In A, atrial fibrillation is present with a slow ventricular rate averaging 55. There are two types of ventricular complexes. The first three and the last, occurring at varying R-R intervals, represent conducted atrial impulses. The fourth to sixth beat, with a prominent S wave and a slightly higher T wave have the same R-R interval of 1.24 seconds (corresponding to a rate of 48), the longest of the record. These evidently are escaped beats. Their normal QRS of 0.08 second, equal to that of the conducted beats, indicates that they arise above the bifurcation of the common A-V bundle, presumably in the A-V node. Their different contour reveals an anomalous spread of the nodal impulses through the ventricles.

In B, similar conditions are present during sinus rhythm (rate 86) with a second degree A-V block, as in A. Again two types of ventricular complexes are seen. One type (the first, fourth and last beats) has a constant relationship to P waves (P-R 0.28 second) and represents conducted sinus impulses; the other type differs from the conducted beats in having no S wave, a depressed S-T and a lower T wave. The P-R distance of these beats is shorter and variable in contrast to that of the conducted beats but the R-R interval is constant (1.34 seconds) corresponding to a rate of 45. These are escaped beats. Their supraventricular (nodal) origin is evidenced by the normal (0.08 second) QRS duration; however, an anomalous ventricular spread is indicated by the difference in contour as compared with beats of sinus origin.

In C, a case with a recent posterior wall infarction, the regular sequence of sinus beats (rate 68, P-R 0.18 second) is disturbed by a ventricular premature systole (the fourth beat). The ensuing ventricular pause is less than compensatory and is terminated by a beat (the fifth) occurring shortly (0.08 second) after a P and differing in contour from the conducted beats by a smaller Q, a larger R, and a less elevated S-T and a more inverted T—an escape beat. The escape, therefore, originates above the bifurcation of the common bundle, but its impulse spread through the ventricles is altered.

In D, a case of intraventricular block (QRS 0.14 second), an escaped beat (the fourth QRS) follows a pause engendered by a nonconducted atrial premature systole; the premature (inverted) P wave is superimposed on the end of T of the third sinus beat. The ectopic atrial impulse entered and transiently depressed the sinus node as evident from the prolongation of the first sinus cycle following the pause. The escaped beat which interferes with this sinus impulse differs from the conducted beats in having a normal (0.08 second) QRS duration. Its origin cannot be definitely established and two interpretations are possible. It may originate in the A-V node and its normal QRS duration could be attributed to recovery of intraventricular conduction during the ventricular pause. Or it may originate in the ventricular septum below a blocking lesion of one of the bundle branches; under these circumstances its impulse could reach both bundle branches in about the same time and thus cause shortening of QRS in contrast to the supraventricular sinus beats. The first interpretation appears to be the more likely one.
Fig. 3. Escapes with aberrant contour and prolonged QRS complexes. Records obtained on three different patients.

Record A starts and ends with a series of regular sinus beats occurring at a rate of 94 and a P-R of 0.24 second. In the middle of the record the P waves disappear and three ventricular beats with prolonged QRS (0.12 second) and an abnormal ST-T contour occur at regular R-R intervals of 1.08 seconds (corresponding to a rate of 56). The last coincides with a sinus P wave reappearing after 3.44 seconds, an interval approaching the duration of five sinus cycles. This suggests the presence of an intermittent S-A block. The three aberrant ventricular beats evidently are caused by impulses of a subsidiary pacemaker escaping during the atrial pause.

In B, during atrial fibrillation, with a ventricular rate averaging 70, in each of the two leads one beat is seen differing from the others in size, shape and QRS duration (0.16 second). Both these beats occur at the same R-R interval of 1.28 seconds, which is the longest of the record. This indicates their ectopic origin in an escaping subsidiary pacemaker.

In C, a case of second degree A-V block, regular P waves can be spaced throughout at a rate of 100. Of three successive P waves, the first is followed by a QRS at a P-R of 0.30 second, the second is blocked and the third just precedes a QRS at a P-R of 0.08 second. Thus, except at the end of the strip, long ventricular cycles (1.08 seconds) alternate with shorter ones (0.80 second)—a ventricular bigeminy. The ventricular beats are of two types. The one with the shorter R-R (and long P-R) has a slender QRS of 0.06 second duration; the other with the longer R-R (and short P-R) consists of a smaller R wave of 0.10 second duration which is more or less slurred in its upstroke. In both the T wave is upright and of the same size and contour. Superficially, intermittent occurrence of an initially slurred R wave in association with an abnormally short P-R could suggest ventricular pre-excitation (the Wolff-Parkinson-White syndrome), anomalous A-V conduction alternating with A-V conduction over normal pathways at a persistent 3:2 A-V ratio. If this were true, however, one could expect that: (1) variations in the degree of slurring of R in the anomalous beats would be associated with variations in their P-R interval and (2) secondary T-wave changes would result from the operation of the ventricular pre-excitation. Since both these anticipated alterations are absent it appears more likely that the anomalous beats represent escaped beats of a subsidiary pacemaker interfering with the conduction of every third sinus impulse with a resulting 3:1 A-V conduction ratio.

In all three records, QRS prolongation of the escaped beats suggests their origin below the bifurcation of the common A-V bundle. On the other hand the promptness of their appearance, at a rate around 50, is unusual for a true ventricular pacemaker but is common for escapes arising in the A-V junction. In these three instances a distinction between nodal escapes with aberrant conduction and ventricular escapes is not possible (fig. 4).
ABERRANT VENTRICULAR CONDUCTION OF ESCAPED BEATS

In A, during atrial fibrillation, three types of ventricular beats are seen. The first type, with a strain pattern and QRS of 0.08 second duration (the second to fourth and the last beat), occurs at irregular R-R intervals at an average rate of 70; these are conducted atrial impulses. The second type, similar in contour to the first but with QRS prolonged to 0.14 second and the S-T more inverted (the second and third last beat), occurs at a cycle of 1.56 seconds, corresponding to a regular rate of 38; these are escaped beats of a subsidiary pacemaker. The third type, intermediate between the first two in QRS duration (0.10 second) and ST-T contour (the first and fifth beat), has the same R-R interval (1.56 seconds) as the second type. These are ventricular fusion beats resulting from competition of a conducted atrial and an escaping ectopic impulse in activating the ventricles. The interval separating the two fusion beats is not a multiple of the cycle length of the ectopic pacemaker, ruling out a parasystolic action of the latter.

In B, at a regular sinus rate of 95, the fundamental disturbance of rhythm is a 2:1 A-V block. Again three types of ventricular complexes are seen. The first type with a P-R of 0.16 second and a pattern of right bundle branch system block (QRS 0.12 second) (the fifth and sixth beat) represents conducted sinus impulses. The second type, with a pattern of left bundle branch system block and a QRS prolonged to 0.12 second (the first beat) occurs at a foreshortened P-R and represents an escape of a subsidiary pacemaker operating at a cycle length of 1.26 seconds, that is at a rate slightly slower than half the sinus rate. The other beats (the second, third and last) resemble in contour one or the other of the two principal types but their QRS and/or T wave is smaller and their QRS duration shorter. These are ventricular fusion beats resulting from simultaneous invasion of the ventricles by the two types of impulses. Their variable contour reflects the relative amount of ventricular myocardium activated by each impulse and depends on the time relation of the ectopic impulse to a P wave. Thus, with a P-R distance of less than 0.16 second, ventricular activation is dominated by the ectopic impulse; with a P-R of 0.16 second the sinus impulse succeeds in "capturing" the ventricles to a greater part (or entirely). The interval between the third and the last beat of the record (both fusion beats) is 5.02 seconds, 0.02 second shorter than four times 1.26 seconds, the cycle length of the ectopic pacemaker. Hence, continuous parasystolic operation of the subsidiary pacemaker is suggested (which could actually be proven in long strips).

In these two cases, the occurrence of ventricular fusion beats is the evidence for the location of the escaping ectopic pacemaker below the bifurcation of the common bundle. No competition for activation of the ventricles is possible when sinus and ectopic impulses share a common pathway through the A-V junction. In both these instances the aberrant contour of the escaped beats can therefore be ascribed to an origin of the subsidiary impulses within the ventricles.

latter mechanism, a contour characteristic for a right-sided conduction defect develops.4

Aberration as encountered in escaped beats and illustrated in figures 2 through 5 does not comply with these criteria. Deviations in their QRS contour cannot be attributed to the action of the normal refractory phase of the conduction system considering the long cycles of these beats. Nor, by the same token, is it possible to invoke a latent abnormal intraventricular conduction defect in all such cases. The operation of the normal or abnormal refractory phase should become manifest in beats of the dominant rhythm rather than in the slow escape beats. Only in figure 2D is such an interpretation feasible though another possibility is indicated in the legend. Furthermore, the type of aberrant ventricular complexes in escape beats does not correspond to that encountered in ordinary instances of aberrant ventricular
Fig. 5. Escapes with aberrant ventricular conduction in ventricular pre-excitation (the Wolff-Parkinson-White syndrome). The four strips (all lead I) were recorded on different occasions in the same patient. The time lines indicate 0.05 second.

In A, at a sinus rate of 51 and a P-R of 0.20 second, ventricular activation and deactivation takes place in a normal fashion as indicated by the slender QRS of 0.08 second duration and the upright T wave. In B, at a sinus rate of 60, ventricular pre-excitation is evident from the shortening of P-R (0.12 second) in association with the typical change in the QRST contour; a pronounced delta wave prolongs the QRS duration to 0.14 second and causes a secondary S-T depression and partial T inversion. In C, the sinus rate varies from 48 to 67 and the last three beats show ventricular pre-excitation as in B. The first two differ from both the pre-excitation beats and the normal beats seen in A. There is a small Q wave, a notch near the top of R, and QRS measures 0.10 second while the ST-T contour is normal. Their R-R interval (1.27 seconds) is 0.05 second longer than the corresponding P-P interval and hence, their P-R distance differs by the same amount. These are escaped beats originating in the A-V node. Their impulses prevent transmission of sinus impulses over normal as well as anomalous A-V pathways with intermittent A-V dissociation resulting. In D, A-V dissociation was induced by carotid sinus pressure (indicated by arrows). The record starts with a pre-excitation beat. As the sinus rate progressively slows to 48, the A-V node escapes at a regular rate of 53 to interfere with five successive sinus impulses. A-V conduction with ventricular pre-excitation is resumed in the last beat following restoration of the sinus rate to 58.

The supraventricular (A-V nodal) origin of the escaped beats is indicated by their normal QRS duration and by the presence of a small "septal" Q wave, like that seen in the normally conducted sinus beats. To account for the difference in contour of these two types of beats some anomalous spread of the nodal impulse in the ventricles must be postulated but differing from that occurring in the pre-excitation beats. It would, therefore, appear that in this case ventricular activation can take place in three different ways: (1) entirely over ordinary A-V conduction pathways, resulting in beats with a normal P-R interval and QRST contour; (2) over an accessory A-V path, resulting in beats with shortened P-R and pre-excitation contour and (3) over some preferential path, limited to impulses originating in the A-V node and resulting in beats with slight QRS aberration.

Conduction. When the QRS of escaped beats is abnormally prolonged and associated with secondary ST-T alterations, the assumption of a supraventricular origin rests on the promptness of their appearance (fig. 3A and B). However, when both the QRS duration and ST-T contour are normal, as in figure 2, their origin above the bifurcation of the common bundle is almost certain.

There are other possibilities to be considered in an attempt to account for an aberrant contour of escaped beats. Thus, alterations in the shape of supraventricular beats are known to occur subsequent to longer ventricular pauses engendered by premature systoles, by a second degree A-V block, or occurring in atrial fibrillation and flutter. However, this type of alteration, the cause of which is obscure, involves mainly the contour of the T wave and usually is restricted to the first beat after the pause. Where the phenomenon continues for several cycles an alternation of the T wave contour may be seen before it resumes its original appearance. Contrariwise, in escaped beats the aberration involves primarily the QRS contour while the T wave may remain completely unaltered, and this is maintained in consecutive escapes. Normalization of the ven-
tricular complex takes place abruptly, with the reappearance of the dominant rhythm. Another rare mechanism could be the operation of a supernormal phase of intraventricular conduction. This may perhaps apply to some specific cases but cannot generally account for a phenomenon occurring so commonly.

While, therefore, in the light of known normal and abnormal cardiac physiology, it seems impossible to explain an aberrant contour of supraventricular escapes on a functional basis, anatomic background for a feasible interpretation has been provided by recent investigations on the structure of the specific conduction system in the mammalian and human heart. The stimulus for the resumption of such studies were the doubts expressed by some workers concerning the existence of a specific conducting system on the one hand, and the unsolved problem of ventricular pre-excitation on the other. The latest authoritative review of the subject by Lev, and his own careful investigations, leave no doubt about the validity of our present concepts concerning a specific muscle system joining atria and ventricles. But his studies also led to the recognition in normal fetal, newborn and young adult human hearts of irregular communications of the A-V node, the common A-V bundle and the beginning of the left bundle branch with ordinary ventricular myocardium, a confirmation of "paraspecific fibers" described previously by Ma-haim and by others. The demonstration of these additional muscle bridges in the normal heart may in the future necessitate some revision of present views concerning the order of ventricular activation provided it could be proved that these fibers have a function of conductivity, a very difficult task.

But supposing that paraspecific fibers, like ordinary specific fibers, do have the potential property of transmitting impulses, their topical relation to nodal tissue could serve very well as a clue for certain hitherto unexplained observations in clinical electrocardiograms and, in particular, the phenomenon discussed in this report, namely, aberration of QRST limited to impulses initiated by nodal or infranodal structures. Confronting electrocardiographic facts, as illustrated in figures 1, 2, 3 and 5, with these recently established histologic data, a hypothesis can be developed to account for the occurrence of normal intraventricular conduction of A-V nodal impulses in some, and more or less aberrant conduction in other instances. All that is required is the assumption that impulses arising in peripheral portions of A-V junctional tissue, in the vicinity of the point of origin of paraspecific fibers, reach part of the ventricular myocardium over preferential (paraspecific) pathways in contrast to impulses arising more centrally in the A-V junction and traveling to the ventricles over ordinary paths, like supranodal impulses (fig. 1). Depending on the length, the course, and the point of insertion of such preferential pathways, and especially their nearness to the ordinary A-V junction, the contour aberration of the resulting ectopic ventricular complex would be more or less pronounced and would vary from case to case (figs. 2, 3 and 5).

Conceivably such preferential pathways could operate even in the absence of paraspecific fibers if one assumes that the arrangement of conducting fibers in the lower A-V node and common A-V bundle is such as to determine impulse distribution to specific limited parts of the ventricular myocardium—an arrangement comparable to that of the internal capsule of the brain. Prinzmetal and associates have postulated this to support their concept of "accelerated A-V conduction." Implication of the latter in the case of nodal escapes is unnecessary, since impulses initiated in distal parts of the A-V junction and proceeding over preferential pathways at a normal speed could reach one ventricle earlier than the other and thus cause aberrant ventricular complexes, in contrast to impulses arising higher up and distributed simultaneously to both ventricles.

Evidently, if this concept is correct, present criteria for location of the origin of escape beats, whether above or below the bifurcation of the common A-V bundle, would require amendments and amplifications. Difficulties will arise in particular when escapes are encountered conforming to idioventricular beats in QRST contour but not in timing (fig. 3). In such cases a retrograde P wave preceding the beats under question would favor their nodal origin. But
this is seen only rarely in escaped beats\(^6\) and cannot be expected in atrial fibrillation or in the presence of A-V block, the two conditions in which escape of subsidiary centers is encountered most commonly. As another point of distinction, foreshortening of the first ectopic R-R cycle may be used—since this occurs at the onset of nodal rhythms in contrast to idioventricular rhythms. But this criterion becomes applicable only if escapes are repetitive (revealing the true nodal interval) and is not observed invariably in nodal rhythms; in fact depression of nodal activity by preceding (conducted) impulses\(^7\) may counteract the apparent abbreviation of, and even prolong, the first R-R in nodal rhythm. The most reliable criterion of an idioventricular origin of escapes is the finding of ventricular fusion beats (fig. 4), a principle also valuable in the identification of ventricular paroxysmal tachycardia.\(^8\) Obviously, when two simultaneous heterogenetic impulses share the activation of the ventricles at least one must have been initiated beyond the bifurcation of the common A-V bundle.

If the concept of functioning short-cuts between A-V junctional structures and the ventricular myocardium should turn out to be valid, then such normal preferential pathways operating in nodal escape beats must be distinguished from abnormal accessory pathways operating in the pre-excitation (Wolff-Parkinson-White) syndrome. Data have been presented in a recent report from this department\(^9\) which strongly favor the latter mechanism against all other invoked in the search for the cause of ventricular pre-excitation. There are several reasons to distinguish the two conditions. Aberrant conduction of escaped beats is common in clinical electrocardiography and bears no relation to the incidence of paroxysmal supraventricular tachycardia in contrast to the pre-excitation syndrome. Furthermore, pre-excitation beats typically reveal a distinct "anomalous component," the delta wave, and secondary ST-T alterations, in contrast to aberrant supraventricular escapes. This is well demonstrated in figure 5, where both types of beats occur side by side. In most instances, the electrocardiographic pattern will permit the differential diagnosis but difficulties may arise when the type of QRS aberration in escape beats imitates the delta wave of ventricular pre-excitation (fig. 3C).

There is also reasonable anatomic background for a hypothesis ascribing the two types of aberration of ventricular complexes to two different types of anomalous A-V conduction pathways. Paraspecific fibers have been described as originating in the A-V node and proximal parts of the specific conduction system, and as maintaining a course close to ordinary conduction pathways. Accessory muscular A-V bridges, on the other hand, demonstrated in a few completely and carefully examined cases with ventricular pre-excitation, were found remote from the A-V junction, bypassing the A-V node. Supposing that both types of fibers have the function of conductivity, one might expect that nodal impulses entering preferential, and perhaps soon rejoining ordinary pathways in the ventricular septum, should produce comparatively little alterations of the ventricular complex in the electrocardiogram compared with pre-excitation beats. In the latter, supraventricular impulses traveling over abnormal accessory A-V connections would directly reach ventricular myocardium of the free walls and hence cause profound alterations in the sequence of ventricular activation and deactivation. Conceivably normal preferential and abnormal accessory pathways could be in action in the same case as is suggested by the observation reproduced in figure 5.

**Summary and Conclusions**

1. An aberrant QRST contour of escape beats is commonly encountered in clinical electrocardiography. Selected examples are presented, illustrating the problems of locating the subsidiary pacemaker above or below the bifurcation of the common bundle under such circumstances.

2. Present criteria based on QRS duration and time of appearance of the escapes may be unreliable in the differential diagnosis of supraventricular and ventricular subsidiary pacemakers. The latter can be diagnosed with certainty when ventricular fusion beats are recorded.
(3) Aberrant conduction of supraventricular (nodal) escapes cannot be accounted for on a functional basis. However, a background for a feasible interpretation is provided by recent histologic studies of the A-V junction in the human heart. Paraspecific A-V connections have been demonstrated which conceivably could represent preferential pathways for impulses initiated in proximal portions of the specific system, in particular in the A-V node.

(4) On the basis of clinical, electrocardiographic and anatomic facts, aberrant intraventricular conduction, supposedly effected by the operation of such normal preferential A-V conduction pathways, must be distinguished from the mechanism of ventricular pre-excitation. The latter is to be ascribed to abnormal accessory A-V bridges bypassing the A-V node. However, the two mechanisms need not be mutually exclusive.

(5) The presented concept of preferential A-V conduction is hypothetical and needs experimental or other confirmation.

**Summario in Interlingua**

(1) Un aberrante contorno QRST de pulsos esclappate es de occurrence commune in electrocardiographia clinic. Es presentate exemplo seligite. Istos illustra le problemes del localisation de centros subsidiari supra o infra le bifurcation del fasce commun.

(2) Le disponibile criterios, que es basate super le duration de QR S e le tempore quando le pulsos esclappate se manifesta, non es semper digne de confidentia in le diagnose differential de centros subsidiari supraventricular e ventricular. In casos del registration de pulsos fusional ventricular, le diagnose de centros subsidiari ventricular pote esser diagnosticate con securitate.

(3) Le conduction aberrante de supraventricular (nodal) pulsos esclappate non es explicabile super un base functional. Tamen, le base de un plausible interpretation es providite per recente studios histologie del junction atrio-ventricular in le corde human. Para-specific connexiones atrio-ventricular esseva demonstrate le quales poteva representar vias preferential pro impulsos initiate in portiones proximal del sistema specific, particularmente in le nodo atrio-ventricular.

(4) Super le base de factos clinic, electrocardiographic, e anatomic, le aberrante conduction intraventricular (supponitemente afficite per le functionamento de tal normal vias preferential de conduction atrio-ventricular) debe esser distinguite ab le mechanismo de pre-excitation ventricular. Iste ultime mechanismo es a ascriber a anormal pontes accessori atrio-ventricular que non passa per le nodo atrio-ventricular. Sed le duo mechanismos non se exclude mutualmente.

(5) Le hic-presentate concepto del conducction atrio-ventricular preferential es hypothetic e require confirmationes experimental o altere.

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