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REVIEW

The Electrocardiographic Footprints of Atrial Ectopy

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Atrial ectopics, also known as a premature atrial complexes (PAC) or atrial premature depolarisations (APD), are supraventricular beats arising from a focus other than the sinus node. Because the various foci provide an array of electrocardiographic (ECG) appearances, an extensive, but confusing nomenclature has developed. Atrial ectopics are a very common finding on Holter ECG monitoring at all ages, the incidence increasing in frequency with age. In the otherwise normal heart, they are generally infrequent and an innocent finding, but in patients with heart disease, they may be a harbinger to more serious atrial tachyarrhythmias. In this review, the ECG footprints of atrial ectopy will be defined. These footprints include prematurity and P wave morphology. The associated features of variable atrioventricular (AV) conduction, variable post-ectopic pauses and variable QRS morphology due to aberrancy will also be discussed. Each of these features will be explained in detail with ECG examples.

Keywords Atrial ectopic • Premature atrial complex • Atrial premature depolarisation • Electrocardiography • Ventricular aberration

Introduction

The term ectopic means ''out of place'' or ''abnormal position'' and, in relation to the heart, refers to an early heartbeat, whose origin is from a focus other than the sinus node. Atrial ectopics originate from an irritable focus in that chamber and clinically they may be asymptomatic or described by the patient as an ''extra'' beat or irregular pulse. The term ectopy is synonymous with ectopic, but it also helps identify combinations of ectopics, such as an atrial bigeminy or couplets.

Nomenclature

Ectopic beats, anatomically and on electrocardiographic (ECG) recognition, can be divided into supraventricular or ventricular. The term supraventricular ectopic is widely used as it encompasses all ectopic activity above the ventricles. However, the term can be easily confused with ventricular ectopics, both in conversation or reports. The term atrial ectopic is therefore linguistically easier to differentiate from ventricular, but the purist will argue that the junctional ectopic is not truly atrial, despite having all the footprints of atrial ectopy to be described. The terms, atrial extrasystole or premature atrial contraction (PAC), although frequently used in the literature imply a mechanical event, rather than the electrical event seen on the ECG. For this reason, some ECG reporters now refer to a PAC as a premature atrial complex. Both this and atrial premature depolarisation (APD) are cumbersome terms. On the other hand, the term ectopy fails to explicitly convey the early nature of these premature electrical events, although that is generally taken as a given.

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Those supraventricular ectopics that originate from low in the atrium are commonly referred to as low atrial or junctional, although both have distinguishing features to be discussed. Another occasionally used term is nodal ectopic, which limits its origin to the atrioventricular (AV) node and could be confused with the sinus node.

Escape beats should be distinguished from atrial ectopics. When the sinus cycle pauses or slows sufficiently, subsidiary escape pacemaker foci assume control of the cardiac rhythm. The fastest of these are theoretically atrial escape foci, distinguished by arriving later than the basic sinus cycle and manifesting a different P wave morphology to sinus beats. The AV node, being the next fastest subsidiary automatic pacemaker, only controls the heart rate when the sinus cycle length is too slow and no atrial escape rhythm supervenes, particularly in the young overnight or pathologically in patients with sinus node disease. Thus a sinus pause without an atrial escape beat may result in an AV nodal or junctional escape beat, which will also occur later than the timing of the expected sinus cycle and may have a characteristic 'retrograde' P wave morphology (generally narrow due to an origin in the atrial septum, inverted or biphasic in the inferior limb leads, and with a transition to negative in V5 to V6).

The Genesis of Atrial Ectopy

Both focal and re-entrant mechanisms may theoretically be operative in the genesis of atrial ectopy, although in the absence of signicant atrial myopathy, the former is far more common. Focal arrhythmias may be caused by abnormal automaticity, although triggered activity, usually due to delayed afterdepolarisations, is the more common mechanism underlying focal atrial ectopy as well as sustained focal atrial tachycardia.[1] This form of impulse generation is often seen at sites of anatomic heterogeneity, and such sites in the atria represent the common locations that harbour atrial ectopic foci. In the right atrium, these include the crista terminalis, the tricuspid annulus, the right atrial appendage, the parahisian region and the coronary sinus os. In the left atrium, the pulmonary veins, the mitral annulus (especially at the aortomitral continuity) and the left atrial appendage are the commonest sites of origin. In the AV junction, any structure from the compact AV node to the His bundle may be responsible for junctional ectopy or junctional escape rhythms.

The Footprints of Atrial Ectopy

There are two basic footprints used to diagnose an atrial ectopic on the ECG. These have been illustrated in Figure 1 as:

- a premature atrial complex (P wave) with a
- different P wave morphology from the sinus P wave.

Additionally, the post-ectopic behaviour of the conduction system may display:

- a normal, shorter or longer AV conduction (PR interval)
- no AV conduction (known as a non-conducted or blocked atrial ectopic)
- a normal or aberrantly conducted (wide) QRS complex
- a full, more than, or less than, compensatory sinus pause following the ectopic.

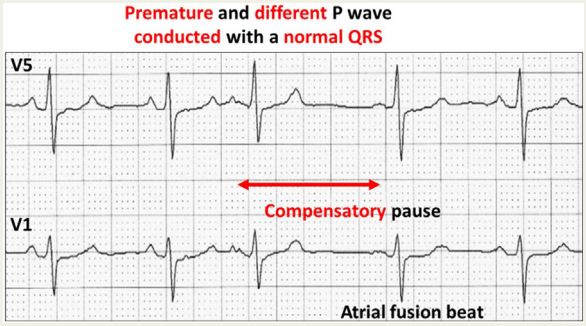

Figure 1 The footprints of an atrial ectopic. Two channel Holter electrocardiograph (ECG). In this example, the ectopic is premature, has a different P wave, is conducted with a normal QRS and followed by a compensatory pause. Following the pause, the next P wave is an atrial fusion beat; an amalgam of a sinus and an escape beat.

There are consequently a wide range of appearances important for recognition of atrial ectopy which will be discussed in detail.

Prematurity

The hallmark of atrial ectopy is prematurity. By definition, an escape beat is not premature. It may occur late and immediately before the next scheduled sinus beat, during the sinus P wave as a fusion beat (Figure 1) or with sinus arrhythmia or sinus pauses, the escape beat may occur after the commencement of the scheduled sinus cycle (Figure 2). Such fusion beats are essentially a competition between two native pacemakers; the sinus node and an ectopic pacemaker focus in either the atrium or AV junction. Such a rhythm disturbance is generally physiological and frequently presents overnight in the young and in particular, the athlete. If persistent, a junctional rhythm will emerge. The question remains that when the complex lies within or follows the sinus P wave, is it actually late or very premature (Figure 2)?

P Wave Morphology

Atrial ectopics may theoretically arise from any part of the atria syncytium, although the vast majority arise from stereotypical sites of anatomic heterogeneity. The P wave morphology usually differs from the sinus P wave and in the absence of structural atrial disease, the surface 12-lead ECG can provide a remarkably accurate guide to determining the site of of origin. This accuracy is predicated, however, on being able to see an entirely unencumbered P wave, where some isoelectric Twave-Pwave segment is visible so that initial forces are not obscured. While this situation can occur spontaneously in the setting of focal atrial tachycardia, it is

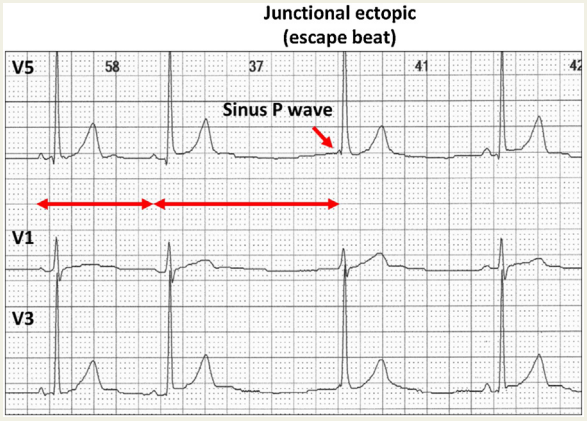

Figure 2 Three channel Holter electrocardiograph (ECG). Sinus arrhythmia (shorter horizontal red arrow) with a junctional escape ectopic following a pause (longer horizontal red arrow). Immediately prior to the ectopic, there is the beginning of a sinus P wave.

less common with isolated atrial ectopics that often occur synchronously with the preceding T wave, rendering their P wave partially or fully hidden. From the experience of mapping focal atrial arrhythmias from various sites, a well-validated algorithm has been developed to accurately predict the site of origin of the ectopic atrial focus.[2]

The most important initial feature of an interpretable P wave to consider is its appearance in lead V1. Left atrial sites of origin have positive P waves in V1 while right atrial sites are usually negative or biphasic. The inferior leads provide information on the location of the focus in the craniocaudal axis. The normal sinus P wave morphology is usually biphasic in V1 with an inferiorly directed axis in the limb leads. This is because the sinus node is located at the top of the fibromuscular ridge in the right atrium called the crista terminalis. Ectopics arising from the crista thus have a similar biphasic appearance in V1 to sinus beats (Figure 3A), although their limb lead appearance is usually sufficiently different (often less positive, isoelectric or even negative in the inferior leads) to distinguish them. Other sites of origin of atrial ectopy have their own characteristic P waves signatures.[2]

If the ectopic focus is close to the AV node, then because of its caudal location, the P wave is inverted in the inferior leads and this is referred to as a low atrial ectopic (Figure 3B). A junctional ectopic in turn, will also have either an inverted P wave if there is retrograde conduction or an absent P wave if there is no retrograde conduction. With atrial pacing, similar findings can be seen with varying positions of the lead in the right atrium.[3]

AV Conduction

Atrial ectopics generally have normal AV conduction, particularly when the ectopic focus is remote from the AV node. The PR interval shortens with low atrial foci, but is usually still >120 ms. With a junctional focus, the PR interval is <<120 ms and may be negative as the P wave in that instance is generated by retrograde conduction to the atrium from the junctional focus. The P wave timing, in relation to the QRS complex, depends on the relative velocity of conduction from the junction to the low atrium versus conduction to the ventricle. It is not surprising, therefore, that the differentiation between low atrial and junctional ectopics may be difficult.

Additionally, atrial ectopics that arise close to the insertion of manifest left lateral bypass tracts in the Wolff-Parkinson-White

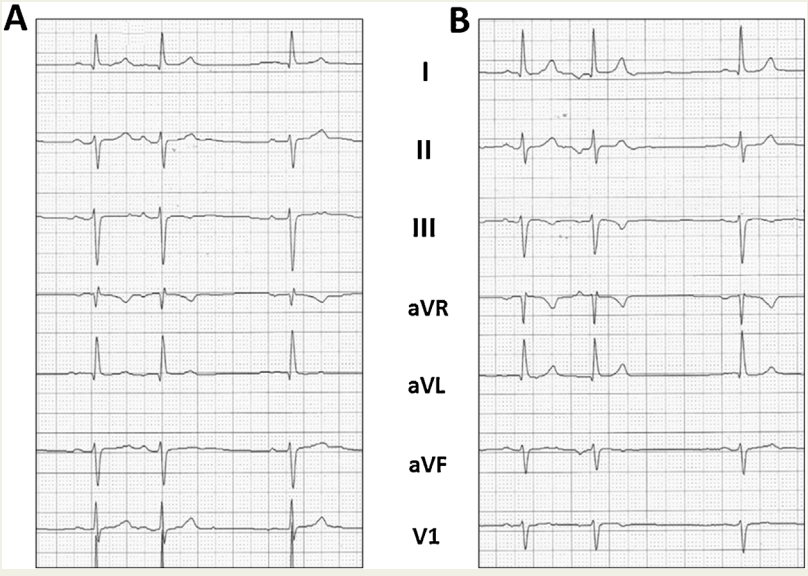

Figure 3 Seven lead ECGs from the same patient. A: Crista terminalis atrial ectopic with a P wave almost identical to the sinus P wave.B: Low atrial P wave. The P wave is inverted in most leads, but not V1.
Abbreviation: ECG, electrocardiograph.

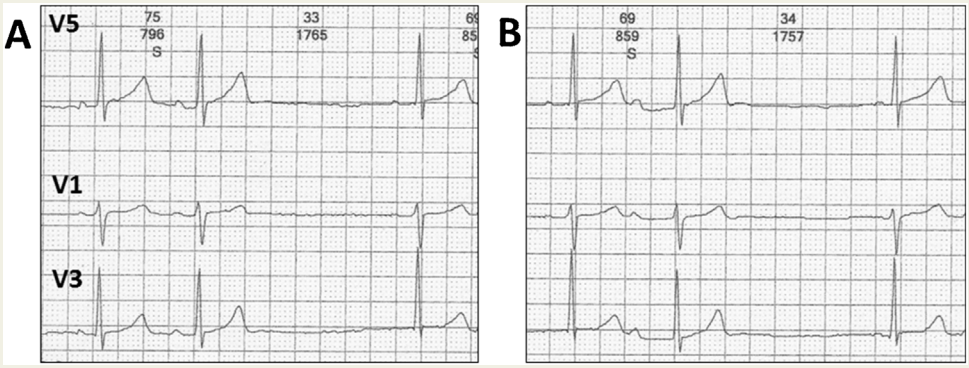

Figure 4 Three channel Holter ECGs from the same patient. A: Ectopic from a high atrial focus. The PR interval is normal. B: An identical atrial ectopic, which is more premature and a prolonged PR interval.
Abbreviation: ECG, electrocardiograph.

syndrome, may significantly shorten the baseline PR and increase the degree of pre-excitation compared to the baseline sinus rhythm as the sinus node is located far away from these bypass tracts.

When the ectopic is very premature, the PR interval may lengthen consequent to the conducting system remaining partially refractory. In this situation, different PR intervals can occur in the same ECG (Figure 4) and once again, this may be physiologic. When PR intervals are markedly different, the presence of dual AV node physiology should also be suspected. This occurs due to functional longitudinal dissociation in the AV node which results in its division into a more rapidly conducting fast pathway and a sluggish conducting slow pathway. The fast pathway has a significantly longer effective refractory period (ERP). In the basal rhythm, AV conduction occurs over the fast AV nodal pathway, however sufficiently premature atrial ectopics may block in the fast pathway if they impinge on its ERP. In this situation, prolonged conduction to the ventricle may occur over the slow pathway, resulting in a much longer PR interval.

If prematurity is sufficient to impinge on the ERP of the AV conduction system, non-conducted atrial ectopy may occur (Figure 5A). These may occur if the atrial ectopic interrupts a Wenckebach sequence, particularly overnight in the young,

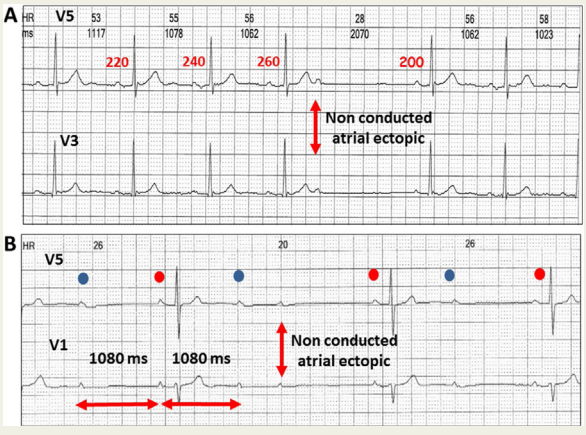

Figure 5 Two channel Holter ECGs. Non-conducted atrial ectopics.
A: A Wenckebach AV sequence is interrupted by a non-conducted atrial ectopic.
B: The sinus cycle is 1,080 ms. A Wenckebach AV sequence is completed with a dropped beat (blue filled circle). A non-conducted atrial ectopic (red vertical arrow), follows, which extends the pause.
Abbreviation: ECG, electrocardiograph; AV, atrioventricular.

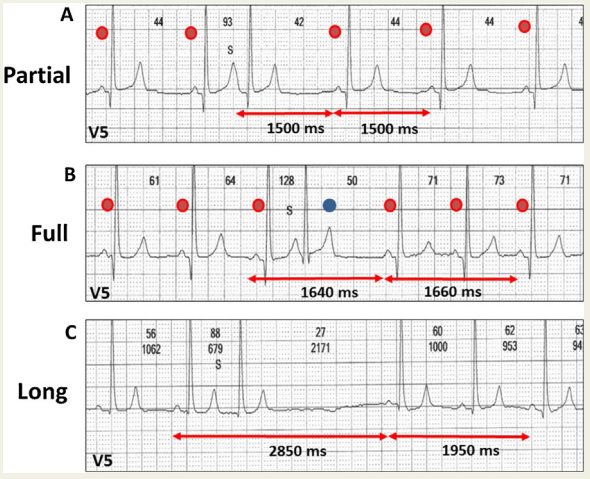

Figure 6 One channel Holter ECGs. Atrial ectopics demonstrating different compensatory pauses. A: The sinus cycle is 1,500 ms. Because of retrograde conduction, a premature atrial ectopic resets the sinus cycle (red filled circle) resulting in a partial compensatory pause. B: The atrial ectopic is more premature and does not reset the sinus node (filled blue circle) resulting in non-conduction and a longer or full compensatory pause.C: The premature atrial ectopic is followed by a long compensatory pause (2,850 ms) due to a prolonged sinus node recovery time (sinus node disease).
Abbreviation: ECG, electrocardiograph.

whereas, if the atrial ectopic occurs after the dropped beat, the Wenckebach pause is extended (Figure 5B).

Post-Ectopic Pause

The premature atrial ectopic will usually be propagated retrograde toward the sinus node. If it invades and depolarises the sinus node, resetting of the sinus cycle will occur. Consequently, the cycle length after a premature atrial ectopic is longer than the basic sinus cycle, creating a partial compensatory pause (Figure 6A). If however, the sinus node is not depolarised, the next sinus cycle results in a concealed sinus P wave and a full compensatory pause (Figure 6B). On occasion, resetting the sinus node results in a prolonged suppression of sinus activity (greater than compensatory pause) or an abnormal sinus node recovery time, indicative of sinus node dysfunction (Figure 6C).

The absence of a compensatory pause is referred to as interpolation. As stated earlier, if the sinus node is not depolarised by an atrial ectopic, then the next sinus cycle occurs on time. In the presence of long sinus cycles such as sinus bradycardia, a premature atrial ectopic may conduct to the ventricle leaving enough time for the AV conduction to at least partially recover before the next sinus beat. This next sinus P wave will conduct to the ventricles, albeit usually with a prolonged PR interval. In Figure 7, three atrial ectopics are shown during sinus bradycardia. In Figure 7A, the ectopic is relatively late and a compensatory pause is present. In Figure 7B and C, the atrial ectopic is more premature allowing the next sinus P wave to conduct with a prolonged PR interval. Consequently, the R to R interval with the embedded atrial ectopic is slightly longer to compensate for the prolonged PR interval. Interpolated atrial ectopics are physiologic and depend on timing intervals and refractory periods of the underlying milieu.

Not all atrial ectopics without a compensatory pause are interpolated. In Figure 8, the atrial ectopic with ventricular aberration (or possibly a ventricular ectopic) once again occurs during sinus bradycardia. However, the R to R interval with the embedded ectopic is shorter than the sinus cycle length. In this example, there is a retrograde P wave from the atrial ectopic which also conducts anterograde via a non-refractory conducting system. This is not a sinus beat, but rather an echo beat and is referred to as reciprocation.

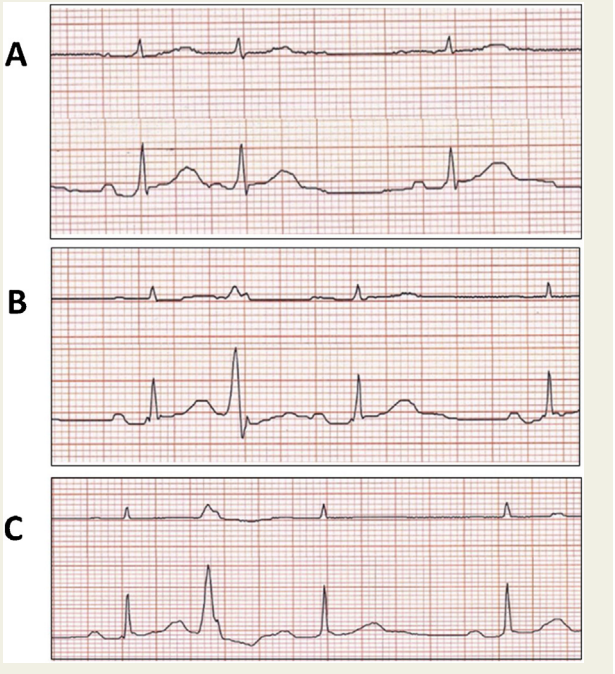

Figure 7 Two channel Holter ECGs, leads unknown, from the same patient. Interpolated atrial ectopics with aberration.
A: The premature, narrow QRS, atrial ectopic results in a compensatory pause. B, C: The atrial ectopics are more premature and the QRSs are conducted with increasing aberration and no compensatory pause. Following the aberrant ectopic, the next sinus beat has a prolonged PR interval.
Abbreviation: ECG, electrocardiograph.

QRS Morphology

The atrial ectopic usually generates a QRS complex similar to the one generated by the sinus node (Figures 1, 3, 4 and 6). However, depending on the prematurity of the atrial ectopic, it may be conducted with a wider QRS referred to as aberration (Figures 7–9). Although first described in atrial fibrillation by Gouaux and Ashman in 1947 [4], the term "aberration" was coined much earlier by Lewis as an abnormal distribution of a supraventricular impulse in the ventricle.[5] Now known as the Ashman phenomenon, the features of aberration as described with atrial fibrillation also apply to atrial ectopics.

On the ECG, QRS widening results from functional right bundle branch block (RBBB) or left bundle branch block (LBBB) configuration caused by physiological aberrancy of

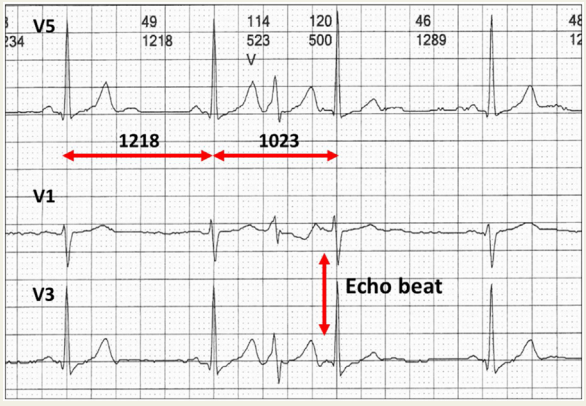

Figure 8 Three channel Holter ECG.
There is an atrial ectopic with aberrant conduction (or possibly a ventricular ectopic), no compensatory pause and the cycle with the embedded ectopic is shorter than normal sinus cycle. Following the ectopic, there is a retrograde P wave (vertical red arrow) which conducts with a normal QRS, suggesting this is an echo beat.
Abbreviation: ECG, electrocardiograph.

conduction to the ventricle. This is due to impingment upon the ERP of the right or left bundle branch by the premature wavefront arising from the ectopic focus. As right bundle branch aberrancy is more common, the usual appearance in lead V1 of the ECG is characteristic with an abnormal premature P wave followed by a modestly widened QRS with an rSR′ or rsR′ appearance, coined ''rabbits ears'' by Marriott.[6] The QRS complexes of the other V leads are often narrower than seen with a left ventricular ectopic, suggesting that the bundle branch block is incomplete and the QRS width dependent on the prematurity or coupling interval of the atrial ectopic (Figure 9B).[7]

The electophysiologic explanation for ventricular aberration is shown in Figure 10. In summary, the action potential duration of the left bundle branch is usually shorter than the right bundle branch. During the latter part of rapid repolarisation or phase 3, there is a relative refractory period, where the bundle branch is able to conduct an atrial ectopic with a bundle branch block pattern. The absolute refractory period also lengthens depending on the preceding long cycle followed by the short cycle of the premature atrial ectopic. There have been a variety of bundle branch block configurations described, even in the same patients, with the most common patterns being either a lone RBBB or a RBBB with an axis shift suggesting coexisting fascicular block.[8] This implies that varying refractory periods occur in different limbs of the conducting system allowing for the range of bundle branch block configurations and QRS widths.

In a bygone era, it was particularly important to differentiate atrial ectopics with aberrant ventricular conduction from ventricular ectopics (Table 1). The ventricular ectopic has more prognostic significance compared to ectopics from supraventricular sites. This was particularly relevant with atrial fibrillation, where aberrant conduction generally reflected an uncontrolled ventricular response requiring more digitalis, whereas, ventricular ectopics suggested digitalis toxicity, a far more sinister scenario. Today, digitalis toxicity is clinically rare.

Atrial Ectopy Combinations

Atrial ectopics frequently occur in multiple combinations, which demonstrate characteristic appearances on the ECG.

Multifocal

Most atrial ectopics are unifocal in that they are generated from a single focus that have identical or near-identical P waves. When atrial ectopics occur with different P wave configurations and varying coupling intervals, they are referred to as being multifocal. In Figure 11A, there are atrial ectopics from both high and low atrial foci. The terms chaotic atrial mechanism or wandering atrial pacemaker are used when there are at least three different P wave foci and an

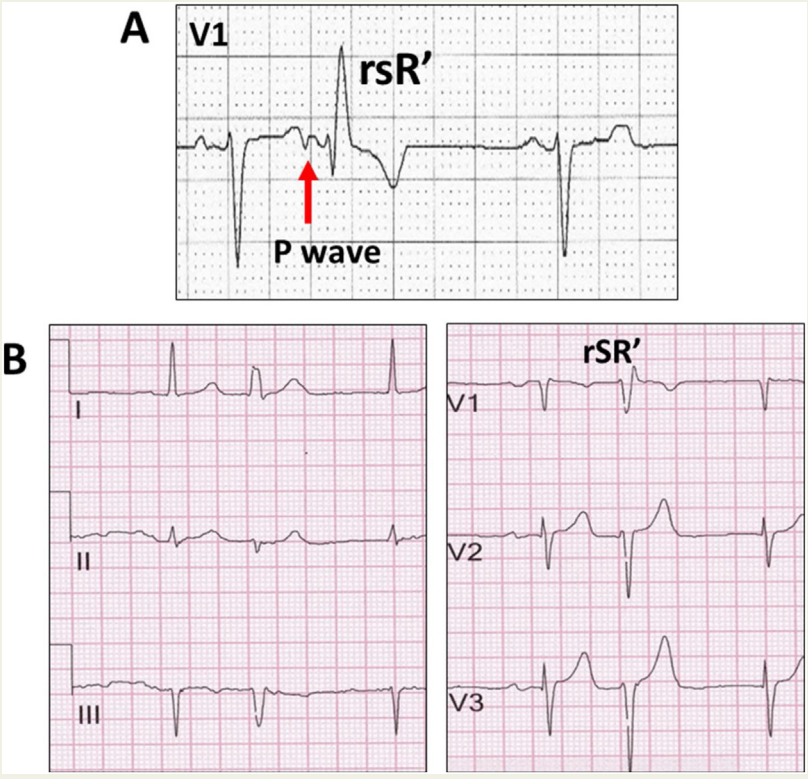

Figure 9 Atrial ectopics conducted with aberration. A: One channel Holter ECG. rsR′ pattern. B. Six lead electrocardiograph (ECG). rSR′ pattern. In the other leads, the ectopic QRS is near normal width, which helps differentiate it from a ventricular ectopic.

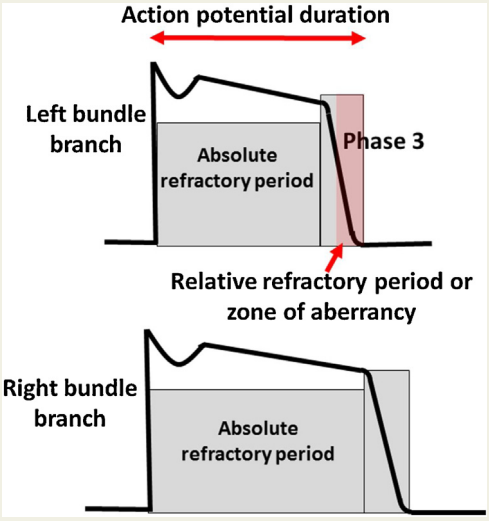

Figure 10 Ventricular aberration.
The action potential duration is longer in the right bundle branch, compared to the left. During the relative refractory period of the left bundle branch, conduction is allowed with a right bundle branch block (RBBB) configuration.

Table 1 ECG differentiation of atrial ectopy with aberration from ventricular ectopy.

Significant:
• P wave present. No P wave, then ectopic may be ventricular, junctional or atrial with P wave buried in preceding T wave
• V1 demonstrates rSR′ or rsR′ configuration if right bundle branch block aberrancy
• If left bundle branch block aberrancy, V1 demonstrate negative wide QRS with sharp intrinisoid deflection with characteristic notched R wave in V6 (with loss of normal septal Q waves)
Weak:
• Long cycle - short cycle (ventricular ectopics also adhere to the rule of bigeminy)

irregular rhythm (Figure 11B). If the heart rate is >100 bpm, then it is referred to as multifocal atrial tachycardia, which not surprisingly is frequently the precursor of atrial fibrillation, particularly in patients with chronic obstructive pulmonary disease. In this situation, there is diffuse atrial disease, which includes the sinus node and the response to therapy is poor and the prognosis guarded.[9]

Bigeminy, Trigeminy and Quadrigeminy

Atrial bigeminy or ''two twins'' refers to a run of at least two atrial ectopics, each one following a sinus beat with a fixed coupling interval exhibiting a long cycle, short cycle sequence (Figure 12). The ectopics can be generated from any atrial site and can demonstrate Wenckebach second degree block sequences, both in the AV node (Figure 12B) and bundle

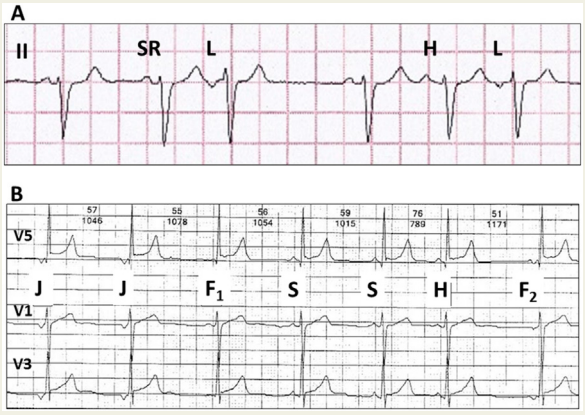

Figure 11 Multifocal atrial ectopics. A: ECG, SR. There are two configurations of atrial ectopics; high (H) and low atrial (L). B: Three channel Holter ECG. Wandering atrial pacemaker. There are sinus beats (S), high atrial ectopics (H), junctional ectopics (J) and different atrial fusion (F_1,F_2) beats.
Abbreviations: ECG, electrocardiograph; SR, sinus rhythm.

branches (Figure 12C). When the atrial ectopics are non-conducted, a bradycardia is present, which may mimic either sinus bradycardia with the non-conducted P wave embedded in the preceding T wave (Figure 13A) or 2:1 AV block, when the ectopic P wave focus lies adjacent to the sinus node and mimics a sinus P wave, albeit premature (Figure 13B).

Atrial trigeminy also refers to a run of atrial ectopics with fixed coupling intervals, but this time every third beat with two sinus beats between. Once again, they can be generated from any supraventricular focus (Figure 14A and B). Atrial quadrigeminy in turn, has three sinus beats followed by an atrial ectopic (Figure 14C). Another repetitive atrial ectopic focus, but without a fixed coupling interval is atrial parasystole.[10] Essentially a dual or biatrial rhythm with the regular slow parasystolic focus competing with the faster sinus mechanism. The ectopic beats can appear anywhere in the non-refractory sinus cycle, but are obviously inhibited during the atrial refractory period. Atrial fusion and ventricular aberration occur. The parasystolic focus cannot be reset by sinus beats, whereas the sinus node can. Being a recognised arrhythmia associated with digitalis toxicity, it is very rarely seen or recognised today.[11]

Couplets, Triplets

When two consecutive atrial ectopics occur, they are referred to as a couplet (Figure 15), whereas three consecutive atrial ectopics are a triplet (Figure 16). Once again they can be generated from any supraventricular site and can demonstrate Wenckebach AV block sequences.

Clinical Features and Aetiology

An irregular pulse due to atrial ectopy is clinically a very common finding and can occur at all ages, although more

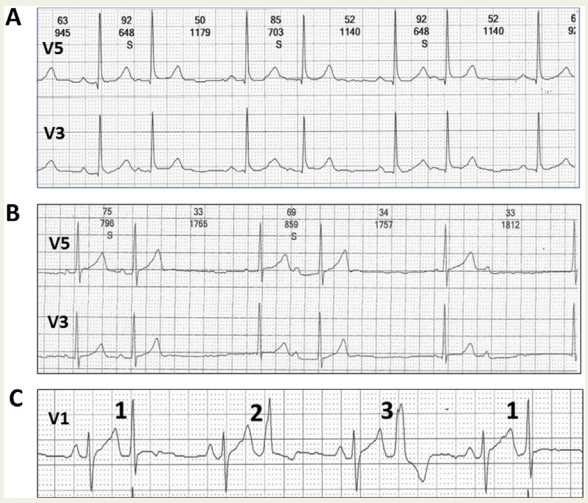

Figure 12 Atrial bigeminy. A: Two channel Holter ECG. Following each sinus beat is an atrial ectopic with a fixed coupling interval. B: Two channel Holter ECG. The atrial ectopics demonstrate a Wenckebach AV sequence with a dropped beat. C: One channel Holter ECG. Atrial bigeminy with a 3:2 Wenckebach sequence in the RBBB. The sequence commences with normal QRS conduction (1), followed by partial right bundle branch block conduction (2) and then a complete right bundle branch block (3). Abbreviations: ECG, electrocardiograph; RBBB, right bundle branch block.

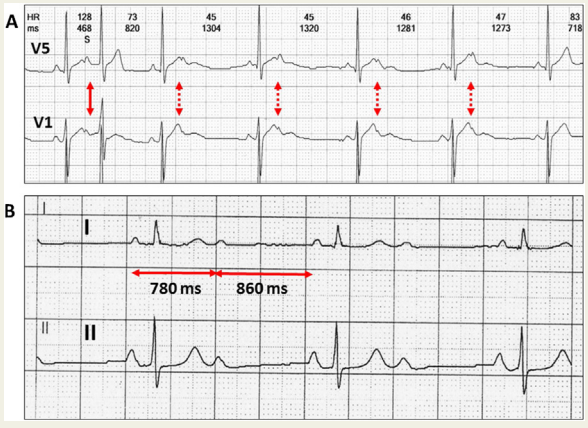

Figure 13 Non-conducted atrial bigeminy. A: Two channel Holter electrocardiograph (ECG). The first atrial ectopic is conducted (solid vertical arrow). Then follows a sequence of non-conducted atrial ectopics, mimicking sinus bradycardia. The non-conducted atrial ectopics are embedded in the T wave of the previous sinus beat (stippled vertical arrows). B: ECG leads I and II. The premature atrial ectopics in bigeminy are late and similar to the sinus P wave configuration, mimicking 2:1 AV block.

commonly in the elderly, and is frequently asymptomatic. In a select young male population, the incidence was 0.4% by ECG diagnosis,[12] whereas with Holter ECG monitoring, the frequency in an asymptomatic volunteer population was

Figure 14 Trigeminy. A: Two channel Holter electrocardiographs (ECGs); leads V5 and V1. A very premature and partially aberrant, high atrial ectopic (solid vertical arrows) follows each second sinus beat. B: One channel Holter ECG; V5. Low atrial ectopics (solid vertical arrows) follow each second sinus beat. C: ECG; lead II. Quadrigeminy. Low atrial ectopics (solid vertical arrows) follow each third sinus beat.

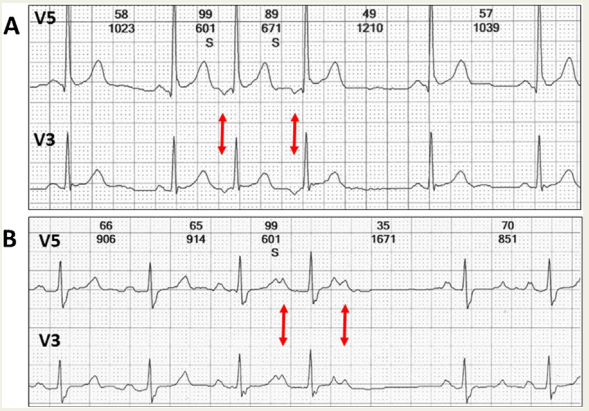

Figure 15 Two channel Holter ECGs. Atrial couplets. A: Two low atrial ectopics (solid vertical arrows) follow the sinus complex. B: Two high atrial ectopics (solid vertical arrows) follow the sinus complex. There is a 2:1 Wenckebach AV sequence in the couplet with initially a prolonged first degree AV block and then a non-conducted ectopic which completed the sequence. Abbreviations: ECGs, electrocardiographs; AV, atrioventricular.

greater than 60%,[11] in a group of normal male medical students 56%[13] and in normal young females 64%.[14] In a population with palpitations, 38% were due to atrial ectopy.[15] More than a 100 atrial ectopics over a 24-hour period was rare in otherwise normal adults.[14,15] Consequently, atrial ectopy in the apparently normal heart is regarded as an innocent finding, particularly if it is infrequent.

Atrial ectopics are more common with increasing age and febrile illnesses, particularly myopericarditis. Other cardio-

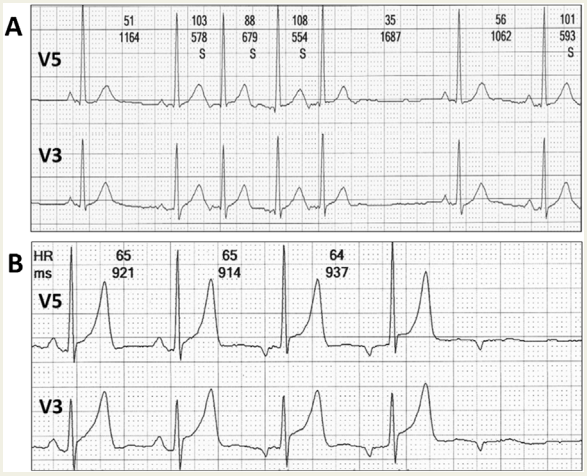

Figure 16 Two channel Holter ECGs. Atrial triplets. A: Three low atrial ectopics follow the sinus complex. B: Three low atrial ectopics follow the sinus complex and demonstrate a 3:2 Wenckebach AV sequence with a non-conducted ectopic completing the sequence.
Abbreviations: ECGs, electrocardiographs; AV, atrioventricular.

pulmonary disorders include mitral valve disease, left and right ventricular failure, post open heart surgery, chronic obstructive pulmonary disease and ischaemic heart disease. Symptomatic atrial ectopy may be due to hyperthyroidism, emotional stress, fatigue, tobacco, alcohol and caffeine use or abuse. Sympathomimetic drugs, illegal stimulants and exercise may also result in frequent symptomatic episodes. It is in these susceptible patients, that atrial ectopics may be a harbinger to supraventricular tachyarrhythmias and in particular atrial fibrillation.

Asymptomatic individuals with atrial ectopics rarely require therapeutic intervention. In symptomatic patients, reassurance is usually the first line of therapy. If a trigger is identified such as tobacco, alcohol or caffeine, then its use should be restricted. Underlying causes such as hyperthyroidism or heart failure should be treated. The most frequently used therapeutic agents are antiarrhythmic drugs, although beta blockers are particularly helpful as an interim measure, whilst treating hyperthyroidism. Digitalis generally is not effective and atrial ectopics may be a sign of toxicity. In patients in whom the ectopics precipitate tachyarrhythmias such as supraventricular tachycardia or atrial fibrillation, therapy is likely to be required. This may be with beta blockers or other antiarrhythmic agents although catheter ablation may be indicated depending on the precise arrhythmia and the patient's wishes.

Conclusions

Atrial ectopics can be generated from irritable foci anywhere in the atria, although most arise from characteristic sites of anatomic heterogeneity. They increase in frequency with age and are generally innocent, unless there is an underlying cause. The ECG footprints of atrial ectopy are prematurity and a non-sinus P wave morphology. Additionally, variable AV conduction, post-ectopic compensatory (or otherwise) pauses and QRS widening due to aberrant conduction may all be observed and all should be considered, when interpreting this common electrophysiology phenomenon.

Disclosures

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