

SYSTEMATIC ANALYSIS OF THE ECG

Rate and regularity

Although the average heart rate in this 10-second ECG recording is 15 beats \times 6 = 90 per minute, it is quite obvious that the rhythm is irregular. The irregularity, however, is not completely irregular and 3 runs are seen with 2 intervening gaps. During the runs, the rate and rhythm is identical, the rate is 100 per minute (300 divided by the R-R interval of exactly 3 "big blocks"). When the R-R intervals are compared to one another, a slight wobble is noted of around 20ms (half a small block). What about the pauses or gaps? Firstly, they are both of equal duration and, secondly, they are essentially equivalent to 2 R-R intervals (perhaps with a few milliseconds difference accounted for by the wobble already noted).

Atrial activity

It appears that there is no normal atrial-to-ventricular (A-V) relationship with regards to the P-waves and normal PR intervals. Certainly, in the gaps in ventricular activity, no discrete P-waves are visible and there is no normal sinus node activity. Therefore, it would be most prudent or time/cost-effective to proceed to next analyse the QRS complexes and then to return to scrutinise the ECG for any atrial activity.

QRS complexes

All the QRS complexes are identical and wide: 140ms. The axis is around minus 75°. The immediate question should be whether these QRS complexes could possibly be conducted. Obviously they are not conducted by the normal ventricular conduction system because they would then be narrow. So could they be conducted by part of the conduction system as in left bundle branch block (LBBB) (with normal right bundle conduction) or vice versa? As the QRS in V1 is not positive, RBBB can be excluded. Further analysis of the QRS to determine whether the morphology is in keeping with LBBB must take into account the fact that in LBBB the right bundle is not affected and there must be evidence of normal right bundle (i.e. fast) conduction. The slow conduction to the left is quite evident, perhaps not so much in V5 or V6 but definitely in the higher "left leads" I and aVL which have wide (slow) positive deflections. But where is the evidence of normal right bundle conduction? The place to look is in the right chest leads V1 and V2. Yes, there are positive deflections; however, especially in V2, this is quite broad (50 - 60ms) and definitely wider than

expected with the normal rapid conduction of the right bundle in typical LBBB. (See Figure 1) Therefore, based on their morphology alone, there is no evidence of the QRSs being the result of conduction even by part of the conduction system.

ST segments and T-waves

Abnormal depolarisation causes abnormal repolarisation. Are the observations in this ECG what can be expected with these abnormally wide QRS complexes? Generally with wide QRSs, the T-waves are opposite polarity to the QRS complexes. Not so in this ECG. The ST segments of wide QRS complexes are often dragged in the direction of the T-waves. If this is either marked or in opposite polarity to the T-waves, there is a primary intrinsic abnormality present and not just one secondary to the abnormal QRSs. The ST segment elevation in the V leads would be considered to be acceptable. In the inferior limb leads (II, III and aVF) the ST segments are very markedly elevated; best seen in III with

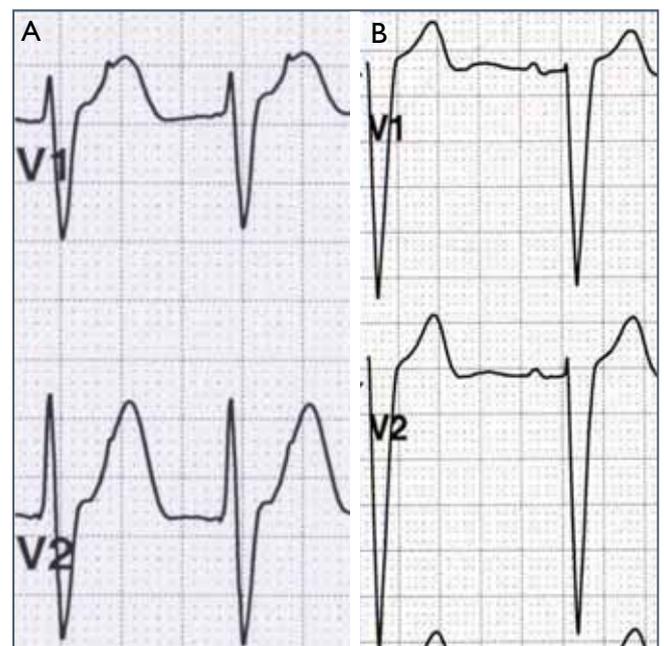


FIGURE 1 A & B: Analysis of the initial R in V1 and V2
I A: V1 and V2 from ECG#28. I B: V1 and V2 from a different patient with typical left bundle branch block (LBBB). The initial R-wave in I A may be misinterpreted as narrow and the QRS complex misinterpreted as LBBB. In comparison, in I B the initial R-wave is extremely sharp as expected with normal rapid conduction down the right bundle in LBBB.

elevation of 5mm. All the usual causes of ST elevation must be considered.

IN SUMMARY

The ECG shows absence of a normal sinus rhythm, wide non-conducted QRS complexes in runs at 100 per minute, 2 pauses which are double the R-R interval and marked ST segment elevation inferiorly.

FURTHER DISCUSSION

The differential diagnosis of wide QRS complexes includes:

- Conducted beats with a conduction system disorder: as in RBBB or LBBB. (Drugs with anti-arrhythmic properties may also cause QRS abnormalities.)
- Conducted atrial rhythm but via an additional or accessory pathway as in Wolff-Parkinson-White syndrome (WPW) or Antidromic Atrioventricular Re-entry Tachycardia (AVRT).
- QRSs arising from ventricular tissue: May be focal or re-entry; it could be slow or fast. If fast (>110/min), this is ventricular tachycardia; if slow (20 - 40/min) this would be a ventricular escape rhythm, which is not ever-evident being suppressed by any faster rhythm such as sinus or nodal rhythm unless these have failed or conduction has failed as in heart block. Intermediate rates, approximately 50 - 100/minute, would be called accelerated ventricular or Idioventricular rhythms.
- Paced beats: slow conduction with wide QRS occurs because pacing leads are implanted in the myocardium and depolarisation proceeds slowly.

These possibilities will be considered in turn.

LBBB with conduction down an intact right bundle was discarded in the analysis above.

Could these QRSs be accounted for by an accessory pathway? The WPW ECG pattern in sinus rhythm is characterised by sinus P-waves, short PR, and widened QRSs with delta waves. In ECG #28, analysis of the first QRS after the first gap shows that there is a P-wave immediately followed by the abnormal QRS. Is this QRS therefore a pre-excited beat of WPW? Although

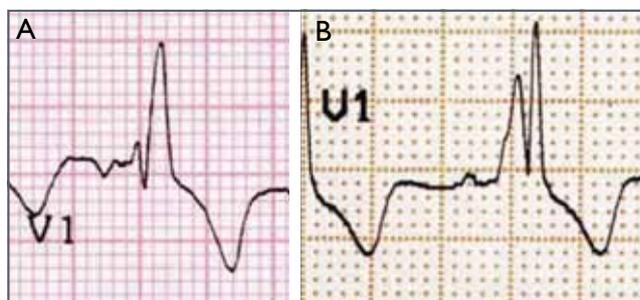


FIGURE 2 A & B: Two different patients with delays late and early in the QRS

In 2 A from a patient with RBBB, the main delay is in the latter part of the QRS with slow conduction and delayed depolarisation of the right ventricle. In 2 B, most of the delay occurs initially due to slow depolarisation via an accessory pathway (causing a delta wave of WPW). The terminal part of 2 B displays rapid conduction via the AV node.

superficially it may appear so in this particular beat analysis of the other complexes rules this out:

- In WPW, the delta wave causes slurred slow conduction at the beginning of the QRS and a normal rapidly conducting terminal part of the QRS. In this ECG the initial part of the QRS is faster than its terminal section. (See Figure 2)
- Additionally, apart from the beat after the gap, none of the others have a P-wave associated with the QRS.

So could this be a short run of self-limiting atrioventricular re-entry (AVRT) or WPW tachycardia that occurs after one sinus beat? No, this is not the case because:

- All the QRS complexes are the same: In AVRT, if orthodromic, the QRS complexes after initiation lose the delta and appear to be normally conducted, and, if antidromic, the QRS would be conducted via the accessory pathway exclusively and become more bizarre after the first sinus conducting beat.
- In AVRT there is an obligatory 1:1 relationship between A and V: i.e. one cannot get another QRS unless the impulse from the ventricles has been up to the atria. Therefore, P-wave identification may be diagnostic. Re-examination of ECG #28 for P-waves reveals that there are P-waves. They are best seen in V1 and the inferior leads: small negative deflections coinciding with the upstroke of the T-wave. How certain is it that

these deflections could just be part of the T-wave? Their absence on the first T-waves after both pauses confirms that the subsequent deflections are indeed P-waves and also reveals that there is not a 1:1 AV relationship, thus excluding AVRT.

Pacing in the conventional right apical situation could account for many of the observations in this ECG: the QRS width, the axis directed superiorly and even the morphology that is reminiscent of LBBB but not quite LBBB. But there are no pacing spikes. Unfortunately not seeing pacing spikes does not mean that they are not there: the ECG recording system may filter them out or they may be visible in only one or a few of the 12 leads. One could be forgiven for interpreting the tiny deflections at QRS onset in V3 as pacing spikes. Is there other evidence that could reject pacing as a possibility? What about the gaps? They are exactly double the R-R intervals: The absent QRS could be because of failed pacemaker capture; this would be confirmed if a pacing spike were visible half way in the gap. However, no spikes not followed by a QRS are seen. An alternative explanation is a pacemaker that has failed to issue a spike. Only the knowledge that the patient does not have a pacemaker, excludes this possibility.

The morphology of the QRS is perfectly compatible with pacing. What if, instead of a pacing spike triggering the QRS, a focus in the ventricle fired: it would look identical. If the focus is firing moderately rapidly and independently, the result would be an accelerated Idioventricular rhythm. To account for the gaps, the focus could be firing intermittently or perhaps it is firing regularly but the impulse is just not getting out past the surrounding tissue into the ventricle. Put simplistically in the heart the fastest rhythm “wins” and takes control. If the sinus rate were faster than 100/minute, it is likely that this ventricular rhythm would not be seen. The gaps give a clue that not all is normal with the sinus node because one should have easily identified a P-wave. The duration between the last P(#4) at the start of the gap and the next P(#5) that almost

merges with the QRS is just longer than 1 second (over, 5 big blocks). This P#5 is different to the other P-waves: it is positive in V1, aVF and negative in aVR, all in keeping with sinus origin but at a slow rate of under 60/minute. (See Figure 3) If this P#5 had come in a little earlier, it is quite probable that it would have conducted. Here, it came late as a result of the sinus bradycardia or slow sinus node recovery time, which means the ventricles fired before the sinus P-wave was given an opportunity to conduct to the ventricles. There is no AV block because nowhere is there a P-wave that should have conducted but failed to do so. After the 2nd gap there is also a sinus P-wave (P#12) which is just visible before it is obscured by the QRS. Apart from the 2 sinus P-waves, other P-waves, as noted previously, happen to coincide with the beginning of T-waves and are negative in the inferior leads; therefore, not due to sinus but retrograde V-A conduction. These retrograde P-waves are not seen after the first QRS following the gaps because the atria have not yet repolarised after the sinus beat. They are seen with each subsequent QRS because, due to the sinus bradycardia, there is delay in atrial depolarisation and the atria are available to the retrograde VA conduction.

The last question is:

Why has this patient got an accelerated Idioventricular rhythm? Three conditions should be considered: acute myocardial infarction; an acute inflammatory cardiac condition; and recent cardiac surgery. The combination of this arrhythmia, its arising from the inferior ventricular wall, the marked ST segment elevation in the inferior leads and the sinus bradycardia are all suggestive of a very recent acute inferior myocardial infarction. Indeed, this was the case. The admission ECG 8 hours earlier, shows acute infero-posterior infarction (Figure 4). What action is needed? No specific treatment is needed of this transient accelerated idioventricular rhythm.

Answer: i

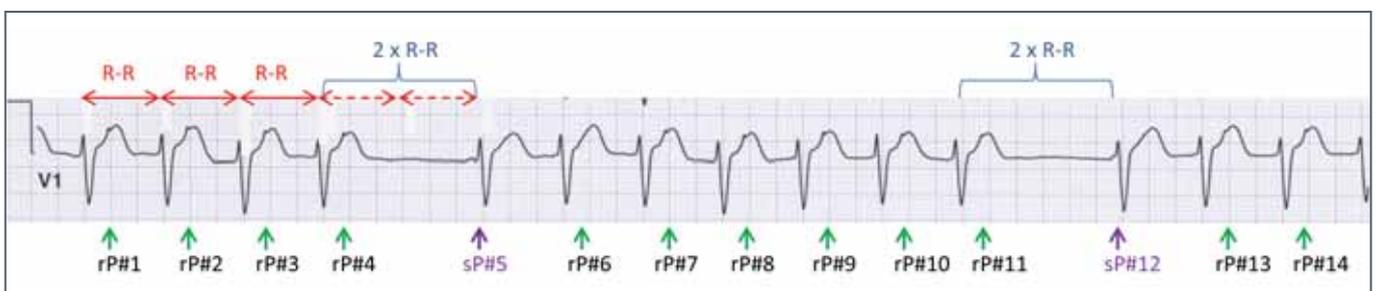


FIGURE 3: The rhythm strip of ECG #28

The RR intervals are essentially regular. The 2 pauses or gaps are equivalent to 2 R-R intervals. Fourteen P-waves are visible. sP#5 and sP#12 are the partly obscured sinus P-waves. All the others are retrograde P-waves (rP) which follow each of the QRSs except those immediately after the gaps.

CONCLUSIONS /LESSONS

- Wide QRS complexes are the result of conduction abnormalities or arise in the ventricles.
- Conduction abnormalities causing wide QRS may be due to conduction system disorders affecting the bundles, accessory pathways or drugs.
- Confirmation of bundle branch block requires evidence of rapid conduction of the unaffected bundle.
- Wide non-conducted QRS complexes are either intrinsic (ventricular escape, accelerated idioventricular rhythm or ventricular tachycardia, as classified by rate) or extrinsic (pacing).
- Accelerated idioventricular rhythm is usually transient and the result of an acute process: infarction, inflammation or injury (surgery).

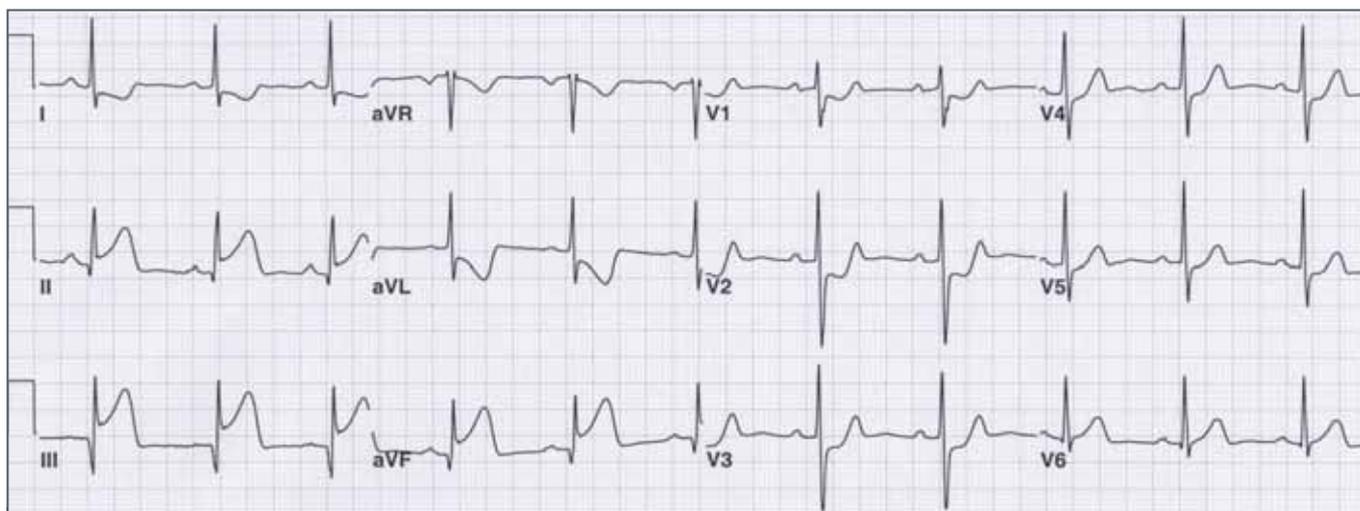


FIGURE 4: The admission ECG

The ECG recorded on admission 8 hours before ECG #28 showing sinus rhythm with acute infero-posterior myocardial infarction.