

### SYSTEMATIC ANALYSIS OF THE ECG

1. **Rate and regularity:** a ventricular rate of almost 156bpm ( $=6 \times 26$  beats in this 10 second recording, the first and the last beats partly chopped off), absolutely regular throughout.
2. **QRS complexes** are wide, duration 200ms, with an axis of minus  $75^\circ$ .
3. **Atrial activity:** It is difficult to identify any P-waves. Be cautious not to call the initial deflection of the QRS a P-wave. Most of these are components of the QRS (as in VI, II and V4-V6). In VI there is an easily seen negative deflection in the early part of the ST segment. There appears to be a one-to-one relationship between these deflections and the QRS. These deflections could be the evidence of atrial activity or P-waves. (Also in Lead I there is a similar subtle bump just after the big wide positive QRS.)
4. **ST and T-waves** appear abnormal with marked ST elevation merging into peaked T-waves in the inferior limb and anterior chest leads. With such abnormal QRS complexes, these repolarisation changes are expected, with the ST/T opposite to the QRS whether the latter are negative or positive.
5. **Other:** Pacing spikes are present and coincide with the beginning of the QRSs. Note that there is variation in the size of the spike and there are even QRS complexes that are not preceded by a spike (8th QRS from the end.)

The differential diagnosis of an observation of a regular, wide QRS-complex tachycardia includes:

- ventricular tachycardia (VT),
- ventricular pacing,
- abnormal activation of the ventricles, either
  - via an antegradely conducting accessory pathway as in antidromic AV re-entry tachycardia, or
  - via an only partially functioning existing conduction system (i.e. bundle branch block, BBB) in supraventricular tachycardia.

The default diagnosis should always be ventricular tachycardia unless there is other evidence. (Of note, the P-waves, whether they are present or not, do not play an important role in the diagnosis at this stage.) Here there is other evidence: spikes. The question then arises if the QRSs are the result of the spikes or whether the spikes are just superimposed on the QRSs. The variation of the recorded spike size and even absence of it should

not be taken as any evidence of true pacemaker output variability or malfunction. This variability is due to the limitations and resolution of the standard digital ECG recording system. It is remarkable that we ever see any spikes considering that their usual width is 0.5ms which is 1/80 of a small block of 40ms on the ECG paper! Therefore, the assessment of correct pacing or malfunction cannot be dependent on spikes but other characteristics. Is the QRS morphology typical of bundle branch aberration? No. Although in this patient with negative QRS in VI, left bundle branch block (LBBB) should be considered, true conducted LBBB as in an SVT will have evidence of normal rapid right bundle conduction especially in the right chest leads. No such observation is made here.

### IS THIS A PACED RHYTHM?

Three important points need evaluation:

1. The QRS axis,  $-75^\circ$  is typical of pacing from a ventricular lead in the RV apex, the standard pacing site.
2. The "LBBB-like" QRS is expected for pacing from RV apex. Bear in mind that this is not an atrial rhythm conducted with true LBBB because that demands normal R bundle conduction of which there is none. It looks similar to LBBB by virtue of the fact that in both LBBB and RV pacing, the right side activates first and the left last.
3. The rate: is it compatible with a pacemaker or with the programmed parameters/settings. A rate of 156bpm is rather fast but it is possible and perhaps this is what had been programmed in this relatively young man. The maximum rate of most permanent pacemakers is 170 to 180 but almost never set that fast; furthermore, pacemakers cannot go above their programmed upper rates.

### IN SUMMARY

The ECG shows a fast ventricular rhythm that is not intrinsic as in VT but paced.

### FURTHER DISCUSSION

#### Why is the pacemaker pacing so fast?

A number of scenarios need to be considered.

- Pacemaker malfunction, the so-called "run-away pacemaker": this is extremely rare with modern pacemakers. Here the

pacemaker would break its cardinal rule and go faster than the programmed rate. Essentially this almost never happens.

- Rate-responsive pacemaker, i.e. a setting programmed that causes increased pacing rate on exercise: as this patient is at rest with the observed heart rate, this can be excluded. (See Figure 1.1)

- A dual chamber pacemaker with atrial (A) sensing and ventricular (V) pacing: if the atrial lead senses an SVT, it will trigger V-pacing but limited to a maximum rate according to the programmed upper tracking rate. So one needs to consider the causes of SVT: the rate excludes sinus tachycardia at rest (Figure 1.2); the regularity excludes atrial fibrillation. An atrial

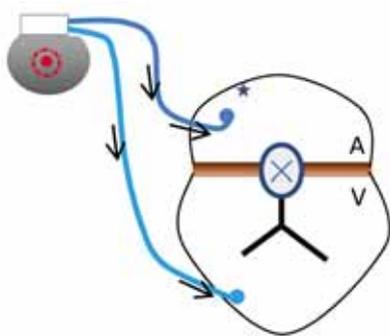


FIGURE 1.1: The normally functioning rate-responsive dual chamber pacemaker (DDD) paces both atria and ventricles in response to activity.

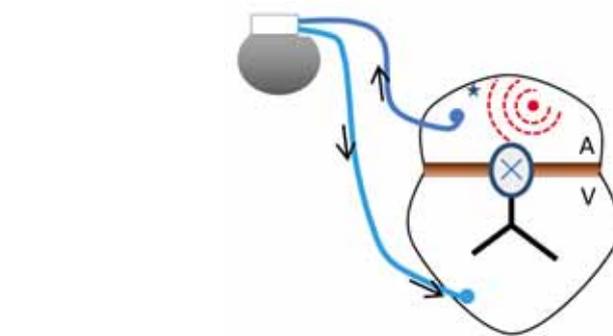


FIGURE 1.3: In supraventricular tachycardias (SVT) that are sensed by the atrial lead, the response of the DDD pacemaker is dependent on the programmed upper rate. An absolute cardinal rule of pacing is that the pacemaker can never pace faster than the programmed upper rate. Therefore, at SVTs faster than the upper rate, the pacemaker, in order to abide by this rule, cannot “sense A and pace V” at 1:1 but with pacemaker Wenckebach or 2:1.

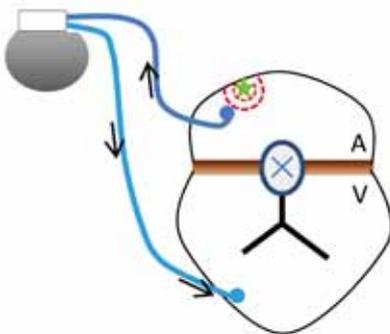


FIGURE 1.2: In sinus tachycardia, the DDD pacemaker senses the atrial activity, tracks it and triggers ventricular pacing.

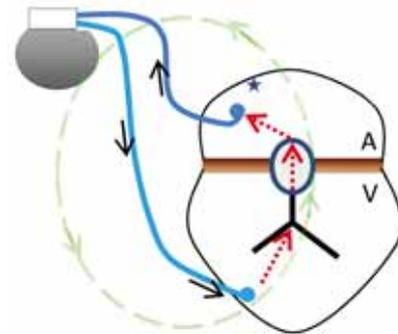


FIGURE 1.4: Pacemaker mediated tachycardia (PMT) due to retrograde V to A conduction: A recurring re-entry circuit occurs if after V-pacing, retrograde conduction occurs and the A lead is allowed to sense this impulse which then triggers a V paced event which again is sensed and the re-entry is perpetuated. The circuit uses both heart with its AV node and the pacemaker for re-entry to occur. The re-entry can be interrupted at any interruptible point in the circuit, i.e. in the pacemaker with a magnet or in the AV node.

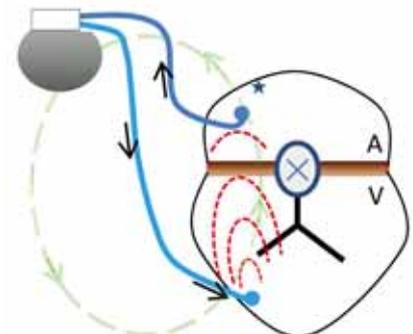


FIGURE 1.5: Pacemaker mediated tachycardia (PMT) due to atrial lead farfield V-sensing. A re-entry circuit comprising heart and pacemaker occurs with the interruptible point being the pacemaker. This situation can be prevented by ensuring that the atrial lead is implanted correctly with good atrial signal sensing and no or minimal contamination with distant ventricular signals.

FIGURE 1: Mechanisms of rapid ventricular pacing in a dual chamber pacemaker.

tachycardia at 156bpm with 1:1; A-sensing V-pacing needs to be considered (Figure 1.3). Another possibility is that this is atrial flutter with an atrial rate that is double, 312bpm, which would be within the usual rate range of atrial flutter and that the pacemaker settings have resulted in a 2:1 response. Analysis of the ECG does show a small deflection in V1 occurring in the ST. This could be a P-wave of an atrial tachycardia. If there were a second deflection half way between 2 P-waves this would be compatible with atrial flutter. As the QRS obscures this halfway point, it is impossible to exclude atrial flutter. If the pacemaker upper rate limit were not set as high, then the pacemaker's response to the high rate A-sensing could be 2:1 or "Wenckebach".

- Pacemaker mediated tachycardia (PMT): a tachycardia that would not occur without the pacemaker and in which the dual chamber pacemaker is a component of a re-entry circuit. A recurring re-entry can occur if after V-pacing, the A lead senses an impulse and then triggers a V-paced event which again is sensed and the re-entry is perpetuated. What can the A lead sense to trigger a V? It is either retrograde V to A conduction via the AVN with a retrograde P-wave (Figure 1.4) or farfield V activity interpreted by the pacemaker as atrial activity (Figure 1.5). This tachycardia typically runs at exactly the upper rate setting of the dual chamber pacemaker.

### HOW CAN ONE DIFFERENTIATE BETWEEN PMT AND AN SVT WITH A-SENSING AND V-PACING?

Various bedside manoeuvres can be tried. Please remember to record a continuous ECG during any manoeuvre as the effect may be transient.

- Vagal stimulation e.g. carotid massage: vagal stimulation may cause block of retrograde V to A conduction as in PMT (Figure 1.4) and terminate the tachycardia.
- Adenosine IV bolus: although this can be tried, and if the tachycardia terminates, the interpretation is the same as with vagal stimulation, the following step is recommended.
- Application of a magnet to the pacemaker: In order for a pacemaker to sustain PMT or pace the ventricles rapidly in response to an SVT, it must sense A so that it can trigger V. Almost all pacemakers come with a feature that, in a magnetic field, a magnetic-reed switch stops the pacemaker from sensing anything. The pacemaker output will continue and it will still pace. However, the pacing rate will no longer be triggered by atrial activity and the pacemaker will revert to the "magnet rate". Thus, whether it is a PMT or an SVT triggering rapid V-pacing, the magnet would have the same effect, viz., the pacemaker rate would drop to the set lower rate. It is only the

response to the removal of the magnet after slowing the ventricular rate that differentiates between PMT and an SVT with rapid V-pacing.

- In a PMT, the re-entry would have been interrupted by the application of the magnet resulting in the V-pacing slowing down, and the atria returning to a normal rate, in a very similar way to carotid massage in an AV junctional re-entry tachycardia; removal of the magnet would not usually be followed immediately by resumption of the original tachycardia. On the other hand, in an SVT which is conducted to the ventricles via the pacemaker, sensing by the atrial lead, which is transiently suppressed by the magnet causing pacemaker slowing, will resume after removal of the pacemaker resulting in the immediate resumption of the fast ventricular paced rhythm.

### HOW CAN THIS BE CONFIRMED?

Interrogation of the pacemaker with a programmer allows one to "see" what the pacemaker wires are "seeing". The endocardial electrogram traces are equivalent to those during an electrophysiological study. (Figure 2) Thus, the correct answers were: 1 (d) and 2 (c).

### WHAT CAN BE DONE?

- Programming of the pacemaker should prevent what was observed in this patient.
- An appropriate upper rate or upper tracking rate of the pacemaker must be set according to the patient's physiological needs. The upper rate of 160 or 170 set in this 35-year-old patient may be acceptable only if he is very physically active.
- To avoid PMT, the post ventricular atrial refractory period (PVARP) can be lengthened so that the atrial lead ignores atrial activity soon after ventricular activity. Thus, atrial lead signals from farfield sensing or retrograde V-A conduction would not be sensed and trigger rapid ventricular pacing. Also the atrial lead sensitivity could be reduced to prevent over-sensing of farfield signals. Many pacemakers have "PMT intervention" as an optional additional programmable feature: if the pacemaker detects that the ventricles are being paced at the upper programmed rate, PMT could be occurring. Automatic PMT intervention will cause the intermittent non-sensing of the atrial lead in an attempt to try to interrupt the circuit and terminate the tachycardia.
- To avoid SVT sensing, tracking and rapid ventricular pacing, mode switch should always be turned on in dual chamber pacemakers. This would allow the pacemaker to switch from a DDD mode after sensing a high atrial rate to DDI mode which does not allow tracking.

**CONCLUSIONS / LESSONS**

- The standard ECG may not record pacing spikes reliably. Apparent absence or variation in spike amplitude should never be interpreted as pacemaker malfunction.
- In a dual chamber pacemaker, ventricular pacing at the programmed upper rate should make one suspect PMT – pacemaker mediated tachycardia.
- Application of a magnet to the pacemaker during the paced tachycardia will slow down the pacing.
- On removal of the magnet, immediate resumption vs. termination of the tachycardia differentiates between a pacemaker-tracked SVT and PMT, respectively.
- In sinus node dysfunction or sick sinus syndrome (as in this patient), AV conduction is usually normal and atrial single chamber pacing is all that is needed. Unnecessary dual chamber pacing may result in unintended consequences and complications.



**FIGURE 2: Dual chamber pacemaker set with mode switch: underlying atrial flutter.**

The programmer strip recording of the ECG Quiz #24 patient after mode switch was turned on shows the surface ECG at the top, the marker channel and the atrial endocardial electrogram. The atrial rhythm is atrial flutter at 312bpm. The pacemaker “mode switch”, now programmed for an atrial rate of 165bpm, kicked in, converting the pacemaker mode from DDD to VVI at 75bpm. For the most part, there is 4:1 AV node conduction with ventricular sensing (VS); a paced ventricular event (VP) occurred when the AV conduction slowed transiently. The pacemaker had originally been implanted for sinus node dysfunction.