MCQs: answers

Chapter 4 - Active Supraventricular Arrhythmias

4.1 Correct answer: C

Comment: P’ waves, as with any other atrial wave, can be conducted normally, be blocked or be conducted slowly (Figures 4.1 and 4.3). This will occur when the P’ wave of a premature supraventricular complex occurs during the relative refractory period of the AV junction, which will cause it to be conducted slowly with the P’R interval lasting possibly even more than 0.20 s.

4.2 Correct answer: C

Comment: The P’-QRS or QRS-P’ relationship in each type of tachycardia can be seen in Figure 4.19. As stated, a P’ wave in a supraventricular paroxysmal tachyarrhythmia that originated in an exclusive junctional circuit (AVNRT) is conducted retrogradely to the atria with just a slight delay in the conduction to the ventricles. This explains P’ wave being hidden within the QRS complex or recorded quite close to its final portion (Figure 4.19A and B). This situation gives rise to a slight change in the QRS morphology, which is seen as an “r” wave in V₁ and as a small “s” wave in other leads (Figure 4.18).

4.3 Correct answer: C

Comment: The typical pattern recorded during sympathetic overdrive can be observed in Figures 4.7 and 4.8. The other possibilities cannot be related to sympathetic overdrive. In any case, the latter can be found in cases of vagal overdrive.

4.4 Correct answer: B

Comment: The diagnoses of reentrant junctional paroxysmal tachycardia, chaotic atrial tachycardia, or atrial flutter are ruled out by the presence of AV dissociation. Furthermore, chaotic atrial tachycardia is irregular and most of the P’ waves are conducted, even though their morphology is different. Therefore, the correct diagnosis is junctional tachycardia.
secondary to an ectopic focus, which is frequently associated with AV dissociation (Figure 4.27).

4.5 Correct answer: B

Comment: The evidence that the P' wave is recorded after the QRS complex and is separated from it, but with RP'<P'R, suggests reentrant tachycardia in a patient with a Wolff–Parkinson–White syndrome (AVRT) (Figures 4.16 and 4.18). It is not a junctional reentrant tachycardia with the circuit located exclusively in the AV junction (AVNRT) (Figure 4.17) because, in this case, the P' wave is recorded within the QRS complex and is not seen, or it is quite close to the QRS complex and modifies its morphology, appearing to be a part of it. In addition, 2x1 atrial flutter is ruled out by the lack of two atrial waves for each QRS complex. The additional fact that the P' wave is negative in lead I (Figure 4.16, see the morphology of ST in I and V6) strongly suggests that the accessory pathway is located on the left side; thus, when atria are activated from left to right, leads I and V6 are facing the depolarization vector tail and are recorded as a negative P' waves.

4.6 Correct answer: C

Comment: Flutter F waves that depolarize the atria cannot coexist with another conducted AV activity. Nor can a Wenckebach-type FR conduction generate a regular RR interval, even if it were atypical. This is due to flutter waves above the AV node that dissociate from a junctional ectopic tachycardia below the AV node. This is sometimes seen in digitalis toxicity (Figure 4.28B).

4.7 Correct answer: C

Comment: Remember that the three characteristics of parasystoles are a variable coupling interval (generally >80 ms), interectopic spaces that are multiple or nearly multiple among them, and, when the recording strip is long, the presence of fusion complexes. Therefore, the correct answer is “variable coupling interval.”

4.8 Correct answer: A
Comment: It is evident that the advanced right bundle branch block morphology (rsR') supports the diagnosis of aberrancy (Table 4.3). The other morphologies are more frequently found in premature ventricular complexes.

4.9 Correct answer: C

Comment: Runs of classic and parasystolic ventricular tachycardia are not so irregular, nor is their morphology so variable (see second strip). Also, in ventricular tachycardia, narrow complexes (captures) that are seen at the end of runs are always early, while in this case they sometimes are early (1) but sometimes they are not (2) (Figures 4.52 and 5.23).

This is not a case of atrial fibrillation with a different conduction degree through the right bundle branch because: a) a “pure” R wave morphology is seen, often with a notch in the R wave descending slope, instead of a rsR complex, and b) when conduction aberrancy through the right bundle branch is present, an advanced right bundle branch block morphology is usually seen without so many intermediate morphologies present.

The irregularity of QRS complexes and their changing morphology with no relation to the preceding diastole or coupling interval decisively supports the diagnosis of atrial fibrillation in the presence of Wolff–Parkinson–White syndrome with different preexcitation degrees. They vary from very slight degrees (6th complex in the first strip, for example) to frequent major degrees, which in this case occur in the form of frequent repeated complexes – thus resembling ventricular tachycardia – but with an irregular cadence. When pre-excitation is extreme, it may be difficult to diagnose, precisely because the δ wave is too large. However, the progressive appearance of a wide complex (see the first strip) together with the fact that narrow complexes are sometimes early and sometimes late, suggest the diagnosis of atrial fibrillation in the presence of Wolff–Parkinson–White syndrome.

4.10 Correct answer: C

Comment: This is not an inspiratory artifact because the QRS complex not only changes its morphology, but also widens. Respiration can modify the morphology of the QRS complexes, especially in intermediate precordial leads, but not their width. This is probably not a premature ventricular complex, nor a couple, nor a ventricular tachycardia run; the coupling intervals of complexes 8, 11, and 14 are different and the morphology with a slurred
S wave in V₆ and the decrease in the R wave amplitude favors conduction aberrancy through the right bundle branch rather than ectopy.

Bearing in mind that this is a case of conduction aberrancy, what mechanism can explain it? The wide isolated complex (8th) and the first one in the couple (11th) and the run (14th) can be explained by classic conduction aberrancy (long-short cycle). The 11th and 14th complexes, even if their previous diastole is not very long, show a very short coupling interval (≈300). Although it has already been stated that this mechanism of classic aberrancy (Phase 3 – long-short cycle) is not very useful in atrial fibrillation, in this case it seems to be valid because the pattern with a wide QRS complex corresponds to a classic right bundle branch block (qRS in V₆). This strongly favors aberrancy over ectopy. However, the wide complexes, with the exception of the first complex, in each case (12th, 15th, and 16th complexes) cannot be explained by classic aberrancy. One explanation may be aberrancy as the result of a transeptal retrograde concealed conduction in the right bundle branch in the previous QRS complexes. The right bundle branch is not retrogradely depolarized by the stimulus descending through the left bundle branch in the 8th QRS complex, but this does occur in the 11th and 14th QRS complexes (retrograde concealed conduction through the right bundle branch), which causes the right bundle branch to remain in refractory period and, consequently, the following QRS complexes (12th and 15th) cannot be conducted through it but instead through the left bundle branch only and thus explain the right bundle branch block morphology. The 16th QRS complex is also wide because the 15th QRS complex may also have retrogradely penetrated the right bundle branch. This 16th complex is no longer retrogradely conducted in the right bundle branch and, consequently, this sustained aberrancy phenomenon ceases.

4.11 Correct answer: C

Comment: Ectopic supraventricular tachycardia runs at a slow rate (≈100 bpm) are shown. The P wave morphology allows for the ruling out of sinus tachycardia. Macro-reentry atrial tachycardia morphology is more often similar to that of atrial flutter (common, inverse, or atypical). Also, macro-reentrant atrial tachycardia, like atrial flutter, usually presents fast rates, not slow rates, as in this case.

Runs appearing in relation to a critical shortening of the sinus RR interval is one of the most important characteristics that define incessant junctional reentrant tachycardia (I-JRT), as shown here (note AB>BC). There is no such critical shortening of the previous RR interval in incessant ectopic atrial tachycardias (I-EAT). Furthermore, an evident warming up of the
focus is usually observed (Figure 4.10), which cannot be observed here, and the first P wave of the tachycardia in I-EAT presents the same morphology as the following P wave. Therefore, this is a case of I-JRT. Currently, it is known that this type of tachycardia (I-JRT) is brought about by a reentrant mechanism with a retrograde arm that is an anomalous pathway with slow retrograde conduction (Figure 3.10). The differential diagnosis between both types of incessant tachycardia can be seen in Table 4.7 (Figures 3.10 and 4.21).

4.12 Correct answer: D

Comment: This is a case of recording artifacts that can be mistaken for wide QRS complex fast arrhythmia runs. The morphology of the complexes are monomorphic, appearing as self-limited ventricular flutter runs (monomorphic QRS complexes with no repolarization waves). However, self-limited and repetitive ventricular flutter runs are seen exceptionally, and they are never asymptomatic, as is this case. Furthermore, if carefully observed, different notches that correspond to the authentic QRS complexes can be observed interspersed in the middle of these presumed runs. This is proven by the superimposition of the sinus node cadence (AB) on the runs (arrows), thus demonstrating that the former is coincidental, with the notches corresponding to sinus node QRS complexes that are masked by the artifact.

4.13 Correct answer: B

Comment: Atrial waves show a wide and undulating morphology at 240 bpm, with no isoelectric baseline, which are predominantly positive in inferior leads (III), and negative in V1. This is a typical reverse (or inverse) atrial flutter morphology. The different monomorphic atrial waves in supraventricular tachyarrhythmias are shown in Figure 4.69. Common flutter waves have a saw-shaped morphology and are mainly negative in II, III, and ventricular fibrillation (VF), and positive in V1, with no isoelectric baseline between the “F” waves. Atypical flutter presents negative “F” waves in lead I and positive “F” waves in leads II, III, VF, and V1 (Figure 4.61). They may be similar to macro-reentrant atrial tachycardia, which present atrial waves <200–220 bpm. Atypical flutter presents atrial waves >220 bpm.

4.14 Correct answer: D

Comment: A) This is a tachyarrhythmia with a narrow QRS complex. The cadence of the arrhythmia in the first strip is almost, but not completely, regular. For example, the first RR
interval lasts 760 ms and the second 800 ms. Throughout the tracing this difference is repeated, although this is not always seen in a long recording. In fact, a certain irregularity can be present in arrhythmias, especially those secondary to an ectopic focus, but they may be also seen in different types of atrial arrhythmias, including atrial flutter, when there are different types of AV block, especially in a repetitive form (Figure 7.6F and H). This is an elderly patient with advanced cardiac disease presenting a narrow QRS complex tachycardia without any apparent atrial waves at 150 bpm. The last part of the QRS complex presents a notch that is also observed in the sinus rhythm ECG tracing, so this cannot be related to any atrial activity. First, atrial flutter should be suspected, because this is the most frequent rapid (around 150 bpm) and regular (or almost regular) supraventricular arrhythmia in this type of patient. In this regard, carotid sinus compression (CSC) is the best procedure to perform in order to assess the response when the AV node is slowed down. The responses of the different types of arrhythmias to CSC are shown in Table 1.4. In this case (B), it clearly helped to “open” the arrhythmia a little, enough to clearly show the presence of flutter “f” waves at 300 bpm (see points); the recording quality of the tracing is not very good, but this is how patient ECGs often appear. This case is a good example that demonstrates the importance of carotid sinus compression, the clinical correlation, and the correct interpretation of tracings with artifacts.

4.15 Correct answer: C

Comment: The most appropriate decision is C for the following reasons: 1) the patient is not unstable hemodynamically and therefore it is not urgent to perform a CV; 2) the patient presents with unpleasant palpitations that have not stopped with the pill-in-the-pocket approach and once in the hospital the arrhythmia has to be suppressed by drugs as soon as possible (intravenously); 3) in this case, the best drug may be adenosine because the patient presents with AVNRT. Giving a drug such as adenosine, which especially blocks the AV node, is not dangerous in these cases. However, we would like to emphasize that the ECG criterion that favors AVNRT is the presence of r’ in V1 or in the S wave in some leads that does not exist in previous ECGs during sinus rhythm. There are also some clinical criteria that favor the reentrant AV node circuit instead of an accessory pathway circuit: 1) female gender, 2) the presence of a pounding feeling in the neck, and 3) the episode of tachycardia (that probably has an inflammatory origin) often starts after 10–15 years of age. Bear in mind that if the ECG suggests an AVRT tachycardia (Figure 4.18B with RP’<P’R), the administration of adenosine would not be recommended (see Chapter 4, Junctional reentrant tachycardia).
4.16 Correct answer: C

Comment: The best approach is C, for the following reasons. 1) It seems that this is not a candidate for ablation as a first option. It is seldom recommended, less so in a patient who presents a first episode of atrial fibrillation a few months ago, and the current episode started 6 days ago is relatively well tolerated. This patient is a candidate for programmed, not urgent, CV.

Furthermore, the probability to success with CV will increase with an amiodarone and ACE-1 pretreatment during the 3 weeks of previous anticoagulation. Furthermore, there is some possibility (±10–20%) that the patient returns to sinus rhythm during the 3-week pretreatment period. The urgent CV is not recommended because the patient tolerates the arrhythmia well and it is worthwhile to give the above treatment before CV.

4.17 Correct answer: C

Comment: The first option to prevent episodes of paroxysmal atrial fibrillation (PAF) is to avoid triggers such as wine and a diet that favors aerophagia. Wine is a trigger of PAF, especially when taken in the evening. The wine coincides with the nocturnal vagal predominance and triggers more PAF. Therefore, our first option is “C.” This includes coughing immediately if the episode starts and, if necessary, taking drugs such as propafenone. If the patient is going to drink during dinner and the crisis starts, the pill-in-the-pocket approach is recommended (see Chapter 4). If preventive long-term treatment is necessary in a patient without heart failure, propafenone (some prefer flecainide) or dronedarone may be the best options. Proceeding to catheter ablation sometimes takes place (see Chapter 4).