ANSWERS

Question #1. There is incomplete A-V dissociation due to both 1) relative sinus slowing and 2) acceleration of the junction, producing an accelerated junctional rhythm. There are two capture complexes. There is a wide QRS complex, probably left bundle branch block. The ST segment abnormalities suggest digitalis effect.

Question #2. The most likely clinical diagnosis is digitalis toxicity in a patient with preexisting bundle branch block.

Question #3. The site of impulse initiation of the wide QRS complex is in the atrioventricular junction.

Question #4. There is no discernible A-V block because the second capture complex in each strip has a normal P-R interval.

DISCUSSION

A systematic and logical approach to this tracing is the key to its interpretation. First, the mean rate of the QRS complexes is normal. The QRS duration is wide (120 ms). The R-R intervals exhibit a dominant regular QRS rhythm with one premature QRS complex. After subtracting the effects of P waves superimposed on the other QRS complexes, the morphology of the premature QRS complex is identical to that of the others. The most common cause of a premature QRS complex that is identical to that of the dominant QRS complexes is a premature atrial complex. In both the top and bottom strip, a P wave precedes each premature QRS. We can assume causality for this P wave. First, both junctional and ventricular rhythms are regular, unless there are PVCs, which is not the case in this tracing. Second, the P-R interval preceding each premature QRS is the same, namely 290 ms. Note that this P wave is actually not premature because all the P waves in both strips have constant P-P intervals. If the preceding P wave did not produce the premature QRS, that QRS would have been at the R-R interval of the dominant QRS complexes. If one assumes that this P wave produced the premature QRS, it must be an example of a capture complex (see below). By comparison, most of the time the P waves and QRS complexes are not associated, except for the capture complex. This finding is an example of incomplete A-V dissociation (see below). Because the dominant QRS complexes are identical to those of the capture complex, they must also be examples of supraventricular complexes, i.e., those produced by impulses at the level of the His bundle or above. Because there are many QRS complexes without preceding P waves and the rate of the dominant QRS complexes is exactly 68 bpm, the dominant rhythm must be an accelerated junctional rhythm. The cause of the A-V dissociation in this case is relative slowing of the primary pacemaker (sinus rate of 65 bpm) with an acceleration of a subsidiary pacemaker (junctional rate of 69 bpm).

Is the prolonged P-R interval preceding the capture complexes an example of A-V block? The answer is no because the P wave occurred in the ST segment when the A-V node was still in its relative refractory period and would have caused P-R prolongation. Therefore, this prolonged P-R interval represents physiologic conduction delay, not A-V block. In fact, there is no A-V block in this tracing. For example, in the top strip carefully compare the R-R interval of the accelerated junctional rhythm (the third and fourth complexes in the top strip) with the R-R interval that includes the premature QRS (the seventh QRS) and the next QRS complex (the eighth QRS) in that strip. Note that the eighth QRS is also premature, but not by much. Nevertheless, this QRS could only occur because of a prior atrial impulse. The P wave immediately preceding this QRS has a normal P-R interval and occurred at a time when the A-V node was completely re-
covered. Therefore, there is normal conduction and no 
A-V block.

**Accelerated junctional rhythm**
In an accelerated junctional rhythm, there are three or 
more consecutive impulses from the junction at rates be-
tween 61–100 bpm. An accelerated junctional rhythm al-
ways implies a primary disturbance of the junction.

**Clinical causes of accelerated junctional rhythm or junctional tachycardia**
1. The most common cause is digitalis toxicity. In pa-
patients with underlying atrial fibrillation, the onset of 
the junctional arrhythmia changes the rhythm catego-
rization from irregularly irregular to regular. Not un-
commonly, there is exit block with Wenckebach peri-
odicity out of the junctional focus, producing a regu-
larly irregular rhythm (group beating).
2. Acute inferior myocardial infarction.
3. Post-operatively after cardiac surgery: mitral valve 
surgery (53% incidence), coronary artery bypass sur-
gery (16% incidence)
4. Myocarditis
5. Hypoxia.

**Glossary**

**A-V dissociation.** This term causes much confusion. 
Langendorf and Pick (2) defined A-V dissociation gener-
cally as a condition in which the P waves and the QRS 
complexes do not have a relationship to one another. 
There are two major subsets: 1) complete A-V dissoci-
tion, in which the Ps and QRSs are without relationship 
all of the tune, and 2) incomplete A-V dissociation in 
which the Ps and QRSs are without relationship most 
of the time. The two ECG manifestations of incomplete A-V 
dissociation are the presence of either: 1) fusion com-
plexes (see below) or 2) capture complexes (see below). 
The causes of A-V dissociation can be divided into four 
groups as follows.

The four basic disorders of impulse formation or con-
duction producing A-V dissociation

1. Slowing of the primary pacemaker: example: sinus 
bradycardia with a junctional escape rhythm
2. Speeding of a subsidiary pacemaker: examples: most 
commonly ventricular tachycardia or accelerated 
ventricular rhythm (requires no A-V block) or, 
less commonly, junctional tachycardia or accelerated 
junctional rhythm (requires no A-V block).
3. Third degree A-V block: defined as complete failure 
of conduction of all atrial impulses. The escape 
pacemaker originates from one of two sites, either 
1) the A-V junction producing the normal su-
praventricular QRS complex seen in that heart, in-
cluding narrow QRS complexes or ones with right 
or left bundle branch block, or 2) ventricular, or 
sub-His foci producing a wide QRS complex lack-
ing the classic pattern of either right or left bundle 
branch block.
4. Combinations, usually of 1 and 2 above, example: 
relative slowing of the sinus in association with an 
accelerated junctional rhythm. The diagnostic hall-
mark is the presence of a capture complex.

**Capture complex.** A capture complex is defined as pre-
mature activation of the ventricles (ventricular capture) 
by an anterogradely conducted supraventricular impulse. 
The presence of a capture complex during A-V dissocia-
tion defines incomplete A-V dissociation, and unequiv-
cally excludes third-degree A-V block. Most capture 
complexes have the same morphology as the usual su-
praventricular QRS complexes produced in that heart. 
The P wave producing the ventricular capture is usually 
superimposed upon the ST segment or T wave caused by 
the prior QRS complex.

**Ventricular fusion complex.** A ventricular fusion com-
plex is defined as simultaneous activation of the ventricles 
by waveforms that originate from two separate areas and 
that blend together. This fusion results in an electrocar-
diographic complex that is intermediate (mixed) in form 
between the deflections resulting from activation by ei-
ther impulse acting separately. The Wolff-Parkinson-
White pattern is the prototypical example of a fusion 
complex. Fusion complexes most commonly occur dur-
ing periods of ventricular tachycardia or during the onset 
or offset of ventricular demand pacing.

**Atrioventricular block.** Atrioventricular block is de-
fined as delay or failure of an impulse to be conducted 
from the atria to the ventricles (anterograde conduction) 
(1). However, in a normal heart, activation of the A-V 
node-His-bundle-branch axis by a premature atrial com-
plex may produce a prolonged P-R interval as a manifes-
tation of physiologic conduction delay, not A-V block. 
Note that in first degree A-V block, which must have a 1:1 
atrio-ventricular relationship, there is no block, but 
merely conduction delay. If the atrial impulse arrives at 
the A-V node at a time when it is not in its normal refrac-
tory period, one should expect P-R interval prolongation 
or even block of the impulse. In contrast, if the P wave 
arrives at a time when the A-V node has recovered and, 
therefore, has an opportunity to conduct normally but 
does not, there is A-V block.

**REFERENCES**

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