002 Sinus Rhythm, atrial rate 90
Mobitz II AVB, Ventricular rate 50
Left Atrial Enlargement
Left Ventricular Hypertrophy
RBBB

a) Long R-R intervals (dropped beats) equal to multiple (2X) of P-P intervals
b) P-R intervals constant and within normal limits (160 msec)
c) P waves are broad (120 msec) and the V1 P waves are 1 mm negative for 1 mm.
d) R waves 1.0 mV in aVL, Ventricular Activation Time* 50 msec in V6
e) QRS duration 140 msec, latest R wave summit in right precordial lead (V1)

Note: Mobitz II results from infranodal conduction system disease, which unlike disease in the A-V node responsible for Mobitz I block, does not delay A-V conduction, but will periodically fail to conduct. Support for infranodal conduction system disease is evident in this example by the presence of RBBB.

* Ventricular Activation Time (VAT, also called the "intrinscoid deflection) is measured from the onset of the QRS to the summit. An interval of 50 msec in a left precordial lead (in the absence of LBBB) is a highly specific criterion for left ventricular hypertrophy. V5 is magnified in Figure 002 and the dashed lines indicate the onset and summit of the R wave.

002: Sinus P waves are seen as arrows in the top (A) column on the Ladder, and conduction through the AV Node (AVN) is seen as diagonal lines linked to arrows representing R waves in the Ventricular (V) column. Non-conducted beats are designated by interrupted diagonal arrows that traverse the AV Node but fail to depolarize the ventricle because of infranodal block.
Sinus Rhythm, atrial rate 65
Mobitz II AVB, ventricular rate 65*
*Junctional Escape beats "compensate" for non-conducted beats
Anteroseptal Myocardial Infarction, recent
Bifascicular Block (RBBB and Posterior Fascicular Block)

   a)  P-R intervals 180 msec and constant prior to dropped beats 
   b)  Escape beats resemble native beats, therefore junctional in origin 
   c)  Beats following escape beats are conducted from P waves hidden in T wave 
   d)  Q waves and ST elevations in V1-4 indicate evolving infarction 
   e)  QRS duration 160 msec, latest R wave summit in right precordial lead (V1) combined with right axis deviation in frontal plane indicate bifascicular block.

Note: Infranodal conduction system disease in association with a myocardial infarction suggests that the ischemic process has penetrated deep within the interventricular septum. The variation in height of the R waves in the right precordial leads results from different degrees of recovery of the right bundle branch depending on the preceding R-R interval--the R waves are tallest when the R-R interval is shortest in the conducted beat following the escape beat. The prolongation of the P-R intervals in the beats following the escape beats results from the relatively recent activation of the AV node by the junctional escape focus.

*Junctional escape beats usually occur at fixed intervals, but in this instance the escape intervals are 1410 and 1360 msec. The differing rhythms preceding each of the dropped beats reset the escape focus at different times. The interval between the non-conducted P waves and the junctional beats is constant (650 msec).

A junctional escape beat is noted on the Ladder by (*). The calipers set to the P-P interval locate a P wave superimposed on the T wave. AV conduction is prolonged by 40 msec (shallower slope) when the R-P interval is short following the junctional escape beat.
014  Sinus Tachycardia, atrial rate 120
Third degree A-V Block*
Junctional (or His Bundle) Escape Rhythm, rate 58
Inferior Wall Injury or Infarction
Anterior Ischemia

a) Every other P wave is partially hidden in the T waves.
b) The A and V rates are constant and almost 2:1, but the P-R relationship varies
c) QRS complexes are narrow, therefore supraventricular (or His) in origin
d) Inferior lead S-T segments are minimally elevated with T inversions and reciprocal S-T depressions in I and aVL.
e) T waves symmetrically inverted in V1-4

Note: The rapidity of the sinus rate suggests that this rhythm is a result of an acute, poorly tolerated process. The S-T elevations in the inferior leads and reciprocal depressions in I and aVL indicate injury or infarction of the inferior wall, which could provide a mechanism for the high degree of A-V block, e.g., the Bezold-Jarisch reflex. This vagotonic reflex is activated by stretch receptors in the inferior left ventricular wall, and can result in varying degrees of A-V block that is usually, but not always transient.

014: Alternate P waves are partially obscured in the T waves but can be seen clearly in Lead II. Although there is P-R proximity that suggests AV conduction, the constant R-R intervals, and changing P-R relationships throughout the strip belie any atrial influence on the ventricles.

*Although none of the P waves conduct, it could be argued that none of the P waves were permitted to conduct because they were either preempted by depolarization from a junctional rhythm or occurred in the refractory period within the preceding T wave. Other ECGs in this patient demonstrated complete A-V dissociation despite differing P and QRS relationships.
Atrial Bigeminy (Sinus Rhythm Alternating with Atrial Premature Complexes), rate 82. Early precordial R-wave transition

a) Alternating complexes follow 860 msec and 610 msec intervals.
b) There are 2 populations of P waves followed by...
c) Alternating P-R intervals of 180 and 240 msec
d) Precordial lead R waves exceed S waves in V2—a nonspecific finding that could result from right ventricular hypertrophy, posterior infarction or septal fascicular block.

Note: Atrial Premature Complexes (APCs, marked by asterisks on the Ladder) are usually followed by a pause that is less than a compensatory pause* because, unlike ventricular premature complexes, atrial premature beats reset the sinus node. The pause results from the transit time required to enter the sinus node and reset it, followed by the time required for the ensuing sinus discharge to traverse the sino-atrial junction to depolarize the atria. Thus the entrance and exit transit times are added to the interval between the P waves.

*Compensatory pauses are seen following most ventricular premature complexes, and result from the lack of any influence on the sinus node -- the sinus node acts as a metronome to keep the heart in step with the sinus beat following the premature beat on schedule. The premature beat occurs early by definition, and the ensuing sinus beat is late by the same time interval that the premature beat was early. For example, if a premature beat is 400 msec early, the post-extrasystolic beat "compensates" by occurring 400 msec late. The sum of these two intervals equals 2 normal sinus cycles for that patient. Compensatory pauses are seen in ECG 324 (click on Menu to select that ECG).

019: Alternate P wave arrows in the atrial (A) column are designated with asterisks (*) indicating an ectopic focus. The alternating sinus and ectopic P waves exhibit subtle differences in P wave morphology in the limb leads. The resulting rhythm closely resembles that seen in ECG 027, but in the latter case the alternate P waves have constant morphology.
Sino-Atrial Wenckebach (Mobitz I) Exit Block, rate 76
Recent Inferior Wall Myocardial Infarction
Early Precordial Transition (consider Posterior Infarction)

a) Alternating long (930 msec) and short (630 msec) intervals
b) Constant P-R intervals (200 msec)
c) Q waves and ST elevations in inferior leads (II, III, aVF)
d) Reciprocal ST depressions in leads I and aVL
e) R>S in V2, >40 msec

Note: The rhythm disturbance bears the "footprints of Wenckebach," in which the pauses are less than twice the length of the shortest intervals, but differs from A-V Wenckebach in that both atrial and ventricular complexes "drop out" and the P-R intervals remain constant. This recording also resembles the previous tracing (019) in which there are alternating short and long intervals, but the P wave morphology is the same on all beats in 027 suggesting that the P waves all arise from the same focus, namely the sinus node. The Ladder has been modified to depict decremental conduction in the sino-atrial junction leading to a 3:2 conduction block from the sinus node to the atrium. The setting in which this rhythm occurs (inferior wall infarction) suggests that the Bezold-Jarisch Reflex is responsible.

The Ladder designations have been modified to represent the sinus node, the sino-atrial junction, and the atrium in order to demonstrate the decremental conduction occurring at the level of the SA junction preceding the dropped beats, resulting in "the footprints of Wenckebach." The timing of sinus node depolarization was calculated by dividing the interval between every other P wave by 3 (2 + 1 = 3) so that 3:2 conduction could be plotted. The identical configuration of all P waves can be contrasted with the alternate P waves in ECG 019, but the resulting "bigeminal" rhythm (defined as "group beating by twos") is almost identical.
Sinus Tachycardia, atrial rate 118
Mobitz I (Wenckebach) A-V Block, ventricular rate 72
Acute Infero-posterior Myocardial Infarction
Left Atrial Enlargement

a) Longest R-R intervals less than 2X the shortest
b) AV Block (2:1, 3:2) with decremental conduction (P-R prolongs before failure to conduct).
c) Q waves in II, III and aVF with S-T elevations denote acute inferior wall MI
d) Prominent R with S-T depression in right precordial leads depict acute posterior MI
e) Negative P wave deflections >40 msec and 0.1 mV indicate left atrial enlargement

Note: This ECG was part of a series that exhibited different manifestations of the Bezold-Jarisch reflex during evolution of an acute infero-posterior myocardial infarction (see 040).

The partially obscured P waves can be found by setting the calipers to fit visible 2 P wave intervals and swiveling the calipers to the right and left. After finding the P waves, sinus tachycardia is indicated by closely spaced arrows in the atrial column, and decremental conduction is seen in the slope of the lines connecting the atrial and ventricular depolarizations exhibiting 3:2 and 2:1 (n/n-1) conduction. The injury current (ST elevation) and Q waves in II, III, and aVF establish an evolving inferior wall infarction, while the tall R waves and ST depressions in right precordial leads indicate posterior wall involvement as well.
Sinus Tachycardia, atrial rate 116
Third Degree A-V Block, ventricular rate 57
Acute Infero-posterior Myocardial Infarction
Left Atrial Enlargement

a) Ventricular rhythm is regular and unaffected by relationship to P waves
b) P-R "relationships" suggest 2:1 conduction, but drift is seen over 10 seconds
c) Q waves in II, III and aVF with S-T elevations denote acute inferior wall MI
d) Prominent R with S-T depression in right precordial leads depict acute posterior MI
e) Negative P wave deflections >40 msec and 0.1 mV indicate left atrial enlargement

Note: Compare the relationship between the P and QRS in the first and last complexes. The subtle drifting apart of the atrial and ventricular complexes may be overlooked by focusing on adjacent complexes, but is obvious when comparing the first and last beats. Both the P-P and R-R intervals are constant. The junctional origin of ventricular depolarization is indicated by the asterisks (*) in the Ladder Diagram and supported by the similarity with the conducted QRS complexes in 030.

At first glance this ECG appears to show first degree AV block because of the wide separation of visible P waves from the ensuing QRS complexes. However partially obscured P waves can be located by setting calipers at ½ the interval between clearly visible P waves, revealing sinus tachycardia at about twice the ventricular rate. The juxtaposition of alternate P waves to every QRS complex suggests 2:1 conduction, but the relationship is inexact, changing progressively from 240 to 340 msec in the first to the last complexes on the rhythm strip. This subtle variation in PR relationships (not actual PR intervals) results from the junctional escape rate (57) being slightly less than half the atrial rate (116).
Sinus Arrhythmia, atrial rate 54-68
Accelerated Junctional Rhythm, rate 58
Resulting in Atrioventricular Dissociation

The R-R intervals are constant and indicate a rate of 58 bpm until the last QRS, which is preceded by a P wave indicating "ventricular capture" by the atrium. The next-to-last QRS is also preceded by a P wave, but it has the same R-R interval of the preceding beats. The relationship between P and QRS complexes varies; some Ps occur within the QRS.

The P waves are hidden in the QRS, but emerge as the atrial rate accelerates. Asterisks (*) on the Ladder indicate junctional origin of 8 out of 10 QRS complexes.
Note: This manifestation of A-V Dissociation is not a result of A-V Block, as evidenced by the ability of the last P wave on the strip to capture (conduct to) the ventricle with a normal P-R interval (160 msec). Many of the P waves are superimposed upon the QRS complexes in the middle of the strip. The proof of conduction is the abrupt change in the previously constant R-R interval when the P wave is appropriately timed as the atrium accelerates and exceeds the junctional rate.

The following algorithm summarizes the broad spectrum of rhythms exhibiting A-V Dissociation, with the rhythm in 056 highlighted.

On the left side of the algorithm, the atrial rate exceeds the ventricular rate as a result of AV Block. When the ventricular rate exceeds and/or is independent of the atrial rate, mechanisms other than block are responsible as shown on the right. In 056, sinus arrhythmia allowed the atrial rate to fall below the intrinsic junctional rate, so the junction took over until the atrial rate accelerated and overtook the junctional rate. The junctional rate of 58 is near the upper rate limit for an "escape" rhythm and may be considered "usurpation" by an accelerated junctional rhythm.
Coarse Atrial Fibrillation
Rapid Ventricular Response, ventricular rate 120
Aberrant Ventricular Conduction ("Ashman Phenomenon")
Left Ventricular Hypertrophy

a) Atrial activity rapid (>300) and irregular in rate and morphology
b) Ventricular response rapid and irregular (R-R 340-750 msec, 80-176 bpm)
c) Aberrant (RBBB) QRS complexes following "long-short sequences"
d) R wave in V5 2.8 mV

Note: In this example intermittent aberrancy (wide QRS complexes exhibiting right bundle branch block) is not due to absolute prematurity (shortest prior R-R interval), but rather "relative prematurity" in that there is a long, followed by a (relatively) short R-R interval leading to the aberrant complexes. Since the duration of the refractory period of the right bundle is proportional to the length of the previous R-R interval, a relatively short R-R interval immediately following a long R-R will encounter a refractory right bundle and produce an aberrant complex.

The atrial rate is rapid and the rhythm is irregular, typical of atrial fibrillation with rapid ventricular response. Two QRS complexes exhibit RBBB in the V1 rhythm strip, both preceded by a long-short interval sequence. These aberrant beats are called "Ashman Beats" and result from an increase in the refractory period of the right bundle branch following a long preceding R-R interval. Other R-R intervals on this ECG are actually shorter (as shown) but the right bundle branch refractory period has not been prolonged by a preceding long R-R and conduction is normal. The ventricular rate (120) was determined by measuring the number of R-R intervals occurring within 6 seconds (12) and multiplying by 10 (6s X 10 = 1 minute). It is preferable to calculate rate, particularly if the rhythm is irregular, by taking a large "bite" rather than by "counting little squares." A 150 mm ruler facilitates a 6 second sample. The width of a standard ECG recording is 10 seconds, and 20 R-R intervals are seen here, so the rate is 120.
Sinus Rhythm, atrial rate 54
Mobitz I (Wenckebach) A-V Block, ventricular rate 42
Left Ventricular Hypertrophy with secondary ST-T changes

a) "Footprints of Wenckebach":
   - Decremental conduction (P-R prolongs before non-conducted beat)
   - Longest R-R intervals (non-conducted beat) less than twice shortest R-R
   - R-R shortens as P-R lengthens (see diagram*)

b) LVH Voltage (Cornell Criterion: R aVL + S V3 = 2.5 mV) with ST-T changes

Note: The slow atrial rate suggests that this rhythm is chronic and well tolerated. The longest RR interval (2080 msec) is less than twice the shortest RR interval (1095 msec) because the pause begins with the longest PR prolongation (last conducted beat) and ends with the resumption of conduction and the shortest PR interval.

*In "classical Wenckebach" the R-R shortens (900 to 825 msec) as the P-R lengthens because the delta P-R progressively decreases (100 to 25 msec) despite the absolute P-R increase.
"Atypical" Atrial Flutter, rate 200
"Physiological" A-V Nodal Block, ventricular rate 90
Right Bundle Branch Block
Nonspecific ST-T changes

a) Atrial rhythm rapid and regular
b) Variable AV conduction (Mobitz I) with irregular ventricular response
c) Flat T waves over left ventricular leads

Note: The atrial rhythm is regular and rapid, but at the lower limit for atrial flutter (range 200-400) but can be slowed by antiarrhythmic drugs. The regular undulations in the baseline suggest continuous cyclic depolarization typical of flutter. The mechanism underlying flutter* is "macro-reentry" in contrast to the repeated firing from an ectopic focus in atrial tachycardia. AV block is invoked during rapid atrial arrhythmias in the atrioventricular node, and is characterized by a Wenckebach pattern that can be seen by the varying flutter wave-to-QRS intervals. This decremental conduction from alternate flutter waves can be best seen at the beginning of the recording; the QRS complexes can be seen to move farther from the conducting flutter wave before being completely blocked. The flutter is again conducted 2:1 with subtle increases in the conduction interval in the 4th through 6th conducted beats.

* The macro-reentry mechanism underlying "typical" atrial flutter is illustrated in the SVT Tutorial elsewhere on this website. Click on SVT Tutorial/Contents/Atrial Flutter. This example is termed "atypical" because of the relatively slow rate and the inferior leads do not exhibit the classical saw-tooth appearance resulting from the classical counter-clockwise reentry loop through the isthmus area within the right atrium shown in the Tutorial.
"Typical" Atrial Flutter, rate 360
Variable Second Degree A-V Block, ventricular rate 128
Right Axis Deviation
Incomplete RBBB
Left Ventricular Hypertrophy

a) Atrial rhythm rapid (360) and regular throughout; "saw-teeth" in aVF
b) Ventricular rate less that atrial, with varying conduction intervals
c) RSR' in V1 and V2. QRS duration 100 msec.
d) R in III taller than R in II, axis > 90°.
e) R wave 2.3 mV in V6 (Liu-DeCristofaro Criterion, R>1.8 mV)

Note: This ECG depicts atrial flutter near the upper range (400), in contrast to 078 which showed atrial rate at the lower range (200). The undulating baseline ion V1 resembles the coarse fibrillation seen in 074 but in this instance the atrial rhythm is precisely regular. To confirm precise regularity, use the calipers and take "big bites" (3-4 seconds) with the caliper points on the peaks of the atrial waves in V1, and then slide or swivel the calipers over the ECG (avoiding "contamination" by superimposed QRS and T waves) to see that the caliper points invariably fall on peaks throughout the V1 rhythm strip. The saw-tooth pattern in leads II, III, and aVF indicates that the macro-reentry loop is in the right atrium as shown in the SVT Tutorial. Right QRS axis in the presence of LVH is a criterion for biventricular hypertrophy.

079: "Big bites" with the calipers establish that the atrial rhythm is regular when the points consistently fall on the same part of the atrial complexes when they are not distorted by superimposition of QRS or T waves.
Sinus Bradycardia, atrial rate 40
Junctional Escape Rhythm, rate 46
Resulting A-V Dissociation (by "default")

a) P and QRS at different rates and rhythms, ventricular rate faster than atrial
b) P-P intervals constant (1500 msec or 40 bpm)
c) R-R intervals without preceding P waves similar (1265 to 1305 msec)

Note: This ECG resembles 056 in that both have sinus bradycardia combined with a junctional rhythm with resulting AV Dissociation with ventricular rate faster than atrial. Although the sinus node-originated QRS complexes (e.g., short R-R intervals preceded by p waves in beats 3, 6, and 9) are different in appearance, all of the complexes are supraventricular in origin (QRS <100 msec). Using the calipers will help solve the rhythm since the spacing between similar elements is nearly constant:
  P-P intervals 1500 msec (40 bpm)
  R-R intervals between complexes not preceded by P waves 1260-1290 msec
  Intervals following sinus-generated beats are constant when measured from P waves

The Ladder depicts the junctional beats with an asterisk (*) indicating their site of origin, while the sinus beats are shown conducted anterograde from the atrium. The retrograde "concealed conduction" from the junction delays or blocks the ensuing atrioventricular conduction when the R-P interval is short. The junctional escape beats have different R-R interval when they occur without intervening P waves than when they follow a conducted beat; this shortening is evidence that that the junctional focus was reset by the passage of the impulse from atrium to ventricle.
132 Supraventricular rhythm, rate 85 (82-89)
Left Axis Deviation (possibly due to Inferior Myocardial Infarction)
Right Bundle Branch Block

a) P waves not seen
b) R minus S in aVL greater than in lead I, QRS positive in aVR
b) QRS duration 120 msec, latest QRS summit in V1

Note: This 86 year old woman was recovering from mitral valve replacement and the subsequent arrhythmias (133 and 134) caused considerable hemodynamic instability, so that it was imperative to establish their nature in order to deploy appropriate management.

132: P waves are not clearly seen in this seemingly regular supraventricular rhythm, which led us to believe initially that it was an accelerated junctional rhythm. However on closer inspection, the R-R intervals are irregular, whereas junctional rhythms are usually regular. (Use the calipers to confirm that there is considerable variation in the R-R intervals). The nature of this arrhythmia will become clearer after examining the next three ECGs (133, 134 and 135) obtained shortly afterwards. Subsequent ECGs indicated that there are P waves hidden in the S-T segments conducting anterograde with long P-R intervals (first degree AV block).
Paroxysmal Atrial Tachycardia with RBBB, rate 140, alternating with Accelerated Idioventricular Rhythm (AIVR) rate 92
Resulting in AV Dissociation and...
Fusion Beats

a) Narrower (120 msec) QRS complexes with RBBB have rate of 140
b) Wider complexes (160 msec) with LBBB configuration have rate of 92
c) Ventricular rate exceeds atrial rate when atrial rhythm slows
d) Fusion beats during transition

Note: The presence of both supraventricular and ventricular complexes is confirmed by the presence of fusion beats (beats 2 and 12) intermediate in appearance between the two "parent beats." The ST-T waves associated with the atrial tachycardia exhibit variable degrees of distortion, suggesting the presence of dissociated P waves with different timing. Two isolated P waves are seen, one inverted P in Lead III following the first QRS, and the other upright P in aVF preceding the 8th QRS on the recording.

Two different rhythms alternate: A rapid rhythm with QRS complexes resembling those in 132 and a slower rhythm with wider QRS. Two fusion (F) beats, designated by colliding arrows in the ventricular column on the Ladder are seen at the transition points between the 2 rhythms. These fusion beats confirm that the wider QRS with slower rate must be of ventricular origin, and is an accelerated idioventricular rhythm (AIVR). Atrial activity is indicated on the Ladder with asterisks in the atrial column, indicating an ectopic atrial rhythm that accelerates to tachycardia, with the P waves obscured within the ST-T portion of the narrower QRS complexes. The first P wave initiating the accelerating atrial rhythm is best seen in Lead III because it occurs at the end of the T wave of the preceding ventricular beat. The P waves will be clearly seen on the next ECG (134) on the right atrial electrogram.
Paroxysmal Atrial Tachycardia with RBBB, ramping from 88-140, alternating with Accelerated Idioventricular Rhythm (AIVR) rate 92
Resulting in AV Dissociation and...

Fusion Beats

a) Intracardiac P waves are seen in V1
b) AIVR present in beats 1-3 and 11-13 (upward arrows denote ventricular origin)
c) Atrial Tachycardia in beats 4-10 and 14-19
d) P waves briefly drive tachycardia 1:1 as atrial rate ramps up before

Note: Atrial electrodes (placed by the surgeon before closing the chest after valve replacement) were accessed for recording electrograms in the V1 lead position on the ECG. These recordings clarified some aspects of the complex arrhythmias, and were also used for pacing (see 135) that stabilized her rhythm and facilitated uneventful recovery. Her sinus rhythm resumed after 48 hours of atrial pacing.

134: The plumb line is used to differentiate the P waves from the QRS complexes on the intracardiac rhythm strip replacing V1 on the last beat on the far right. The atrial signals are magnified because the exploring lead is in contact with the right atrium, and are seen to occur during the S-T segment of the surface ECG. The irregular ectopic atrial activity is plotted on the Ladder with asterisks (*). It can be seen that there is anterograde conduction when the P waves emerge from the ventricular refractory period and accelerate to overtake the ventricular rhythm. When atrial activity slows, the accelerated idioventricular rhythm (AIVR) takes over for three beats until the ectopic atrial focus recovers and ramps up the rate again. Although the P waves are negative in the inferior leads and follow the QRS complexes at the start of the rhythm strip (suggesting retrograde VA conduction) the atrial activity is independent of the AIVR. Note the P wave within the first QRS complex in the 3 beat segment of AIVR. The next ECG (135) establishes that anterograde AV conduction is intact in this patient.
135  Atrial Pacing, rate 90, with 1:1 capture
Right Bundle Branch Block
Digoxin toxicity

a) Pacing "spikes" initiate P waves which conduct to ventricle
b) Broad QRS with late R wave summit in V1
c) Prior Arrhythmias -- sinus node suppression and paroxysmal atrial tachycardia

135: This ECG clarifies several confusing aspects seen on this patient's previous tracings. The QRS complexes are identical to those in 132 and the narrower complexes in 133 and 134, confirming that 132 had to represent a supraventricular rhythm. The bursts of rapid rhythm in 133 resemble these complexes as well. Lastly, the integrity of atrioventricular conduction is affirmed by 1:1 capture at a normal P-R interval (160 msec). In summary, the principal problem following open heart surgery in this patient was the lack of competent sinus node function, which in turn led to competing takeovers by an unstable atrial rhythm and AIVR. Intact AV conduction prompted utilization of atrial pacing to stabilize the rhythm. It was later learned that the patient had elevated serum digoxin levels, which helped explain the suppression of the sinus node, accelerated atrial rhythm, and AIVR.
Sinus Tachycardia, rate 112
Parasystolic Ventricular Premature Complexes (VPCs)
Fusion Beats
Left Atrial Enlargement
*LVH

a) Regular, rapid supraventricular rhythm with normal P waves
b) Variable coupling of VPCs to sinus beats with constant inter-ectopic intervals
c) Fusion beats
d) Broad P waves with negative deflections .04 sec and 0.1 mV in V1
e) Prominent R in LV leads, delayed activation time in V6, inverted T waves

Note: There are 4 premature FLBs (funny looking beats) that occur after the sinus P waves. The most premature complexes (beats 4 and 14) are wider and exhibit taller R waves. When these beats are compared in leads II and III they look similar to each other but have T waves of different depths. Most Ventricular Premature Complexes (VPCs) occur at fixed "coupling intervals" after the native QRS as a result of a micro-reentry pathway that links them to the prior ventricular depolarization. In this ECG these complexes have differing relationships with the preceding R waves, but are coupled to each other with 1550 msec spacing. This fixed inter-ectopic interval suggests that they arise from an ectopic focus independent of the rest of the conduction system -- a parasystolic focus. The differing P-R relationships result in different degree of fusion, with more expression of the native QRS in the later fusion beats. LVH cannot be diagnosed by strict voltage criteria, but the left atrial enlargement, delayed ventricular activation time (VAT), and ST-T changes permit the diagnosis by the Romhilt-Estes point system.

142: The VPCs occur at slightly different intervals after the preceding P wave causing different degrees of fusion. The interectopic (VPC to VPC) intervals are constant, and the long interval between the calipers is approximately twice 1550 msec (3190 msec).
Ventricular Tachycardia, rate 135
A-V Dissociation with Fusion (atrial rate 78)

Note: Wide complex tachycardias are often difficult to diagnose with certainty, but if A-V dissociation and fusion beats are present, the diagnosis of ventricular tachycardia is assured, as in this example. The fusion beats can be found in the V1 rhythm strip by looking for narrower QRS complexes with shallower T waves: beats 4, 11, and 16. A-V dissociation can be found by looking for P waves that are not obscured by QRS complexes (marked with arrows below the rhythm strip). Hidden P waves can be found by setting the calipers for an observed P-P interval, then swiveling forward and backward to look for distortion of the QRS or T wave. Fusion beats will represent participation in the depolarization from ventricular and supraventricular foci. In the Ladder, beats of ventricular origin are designated by asterisks (*) and upward arrows, while fusion beats are shown by colliding arrows in the ventricular column.

146: P waves are marked with thin arrows at the bottom of the figure, and obscured P waves can be plotted by setting the calipers for observed P-P intervals and dividing by 2.

The spectrum of A-V Dissociation is illustrated with Ventricular Tachycardia highlighted.
Atrioventricular Nodal Reentry Tachycardia (AVNRT), rate 160
Left Axis Deviation
Low Voltage in Limb Leads
Left Ventricular Hypertrophy (Cornell Criterion: R in aVL plus S in V3 = 2.5 mV)

160: Narrow complex tachycardia bespeaks supraventricular origin; the absence of obvious P waves renders unlikely either sinus or atrial tachycardia. If retrograde P waves can be seen after the QRS, AVNRT is the probable cause. The "pseudo R waves" in this patient with dilated cardiomyopathy are actually tall Ps. Reentry within the AV Node is diagrammed in the Ladder.

161: The previously seen "pseudo R waves" in V1 have moved in front of the QRS complexes and are shown to be large P waves indicating right atrial enlargement.
**163** Sinus Rhythm, rates 56-60
Accelerated Idioventricular Rhythm (AIVR) rate 58
Resulting in Atrioventricular Dissociation and...
Fusion Beats

Note: This ECG depicts a blend of two independent rhythms with more influence of the ventricular component on the far left and far right, and the conducted supraventricular beats in the middle. The beat with the narrowest QRS and smallest T in the rhythm strip is conducted from the sinus node, while those with widest QRS and tallest T waves are of ventricular origin. AIVR is usually a benign rhythm disorder seen after myocardial infarctions, following spontaneous or iatrogenic thrombolysis and reperfusion. Although voltage criteria for left ventricular hypertrophy are met, the presence of fusion invalidates the measurements.

163: P waves occur prior to every QRS, but the P-R interval is shortest when the QRS is wide and longer when the QRS is narrow and the T wave is lowest; they undergo transition from wide to narrow and back. Atrial and ventricular rates average 58 bpm; the atrial rate accelerates toward the center and then slows toward the right, while the ventricular rate is constant at 58. The ventricular rate exceeds atrial in this form of AV dissociation.
Multifocal Atrial Tachycardia (MFAT) rates 140 -160
Rate-related Left Bundle Branch Block (LBBB)
Old Inferior Myocardial Infarction

a) P waves are rapid, irregular in timing, vary in configuration, and precede every QRS
b) When rate accelerates, QRS becomes wide with secondary T wave changes
c) Before bundle branch block appears, deep, broad Q waves are seen in inferior leads

Note: This ECG was thought by many to represent the onset of "ventricular tachycardia," but the gross irregularity and presence of P waves (sharp deflections superimposed on the T waves) disproves this interpretation. The LBBB is not strictly rate related, in that some of the normally conducted beats have shorter R-R intervals than some of the LBBB beats, but the trend on this and other ECGs from this individual was to develop LBBB when the atrial rate accelerated. It is interesting to note the masking of the Q waves in aVF and the emulation of a lateral infarction in aVL when the LBBB commences.

Prior to the onset of the wide complex irregular tachycardia, P waves of differing configuration can be seen (arrows) and are seen to continue throughout the strip. Note the different shapes of the P waves in the limb leads as the rate accelerates, and the Q waves in II, III, and aVF indicative of an old inferior wall infarction. The wide QRS complexes have an RSR' pattern in V6, indicating delayed conduction in the territory served by the left bundle. Although this wide complex tachycardia resembles ventricular tachycardia, the irregularity and 1:1 relation with the P waves indicates supraventricular origin.
Ventricular Tachycardia, rate 234
V-A Conduction (2:1)

Note: Wide complex tachycardias can be of either supraventricular or ventricular origin, and it is often difficult to differentiate them. The superior axis and the monophasic appearance in the right precordial leads favor ventricular origin of the tachycardia. The important point is that the rate is extremely rapid and poorly tolerated, and regardless of cause should be treated with prompt countershock to minimize myocardial ischemia.

Upward arrows below the rhythm strip point to P waves that seem to occur in a 2:1 ratio after alternate QRS complexes. Some uncertainty about the relationship between atria and ventricles occurs when the P waves are not as clearly visible at the onset of the rhythm strip, as well as in the inability to discern P waves in the limb leads to confirm that they are conducted from the ventricle to the atria. Ventricular tachycardia may be dissociated (completely independent of atrial activity) as seen in 146, conduct retrograde 1:1 through the AV node (see 658 and 659), or conduct intermittently as seen here and in 702.

The rapid ventricular rate exceeds the atrial rate, qualifying as the highlighted form of AV Dissociation: Ventricular tachycardia.
Atrial Fibrillation
Left-sided Accessory Pathway Conduction, rate 210

a) Rapid, irregular rhythm
b) Wide (160 msec) QRS with "Right Bundle Branch-like" configuration

Note: The individual QRS complexes resemble those of ventricular tachycardia because they are generated in the same manner as ventricular ectopic beats, spreading across the myocardium fiber-to-fiber from the point of insertion of the accessory pathway without benefit of the His-Purkinje system utilized in normal ventricular depolarization. The rhythm is grossly irregular--too irregular to be ventricular tachycardia and thus a result of atrial fibrillation. Some R-R intervals reveal the ability to conduct beats at a rate of over 300. The frontal plane axis is markedly rightward and the R waves are widest in V1.

The QRS configuration is typical of beats of ventricular origin, but the gross irregularity of this rapid rhythm is caused by atrial fibrillation and is incompatible with ventricular tachycardia. The right bundle branch block configuration indicates that the bypass tract is left-sided, initially activating the left ventricle and slowly spreading the wave of depolarization over the right ventricle. Compare this ECG with the next tracings from the same patient (170 and 171).
Atrial Fibrillation
Accessory Pathway conduction, rate 220
Fusion beats

a) Rapid, irregular rhythm
b) Wide (160 msec) QRS with "ventriculoid" configuration
c) Occasional narrow QRS complexes of variable appearance

Note: This ECG is from the same patient as 169 and provides more definitive proof of the mechanism of the arrhythmia. Once again the rate is rapid, the rhythm irregular, and the majority of the beats resemble those of ventricular origin. However on this recording one normal QRS complex (10th beat) and 4 species of fusion beats are seen. The 5th beat is narrow and reveals a near 50:50 blend of the "parent beats" (His-Purkinje and Bypass Tract), whereas the 9th beat, and the two beats seen at the end of the V1-3 recordings reveal more evidence of pre-excitation from the bypass tract contribution (tall R waves with effacement of S waves). This is a poorly tolerated and potentially fatal arrhythmia and should be terminated by countershock without delay.

This recording demonstrates three variations of AV conduction.
1. A-V Nodal and His-Purkinje Conduction (N)
2. Fusion Beats with Pre-Excitation (F)
3. Bypass Tract Conduction (majority)

One normally conducted beat (N) utilized the AV node and His-Purkinje system. Fusion beats (labeled F) are hybrid beats depolarized by both the bypass tract and the His-Purkinje to variable degrees. The rest are conducted down the left postero-septal bypass tract and resemble beats of ventricular origin (as are all of the beats in 169).
Sinus tachycardia, rate 108
Wolff-Parkinson-White Syndrome

a) P waves upright in I and aVF, rate >100
b) Slurred upstrokes of QRS complexes
c) P-R intervals 100 msec*

Note: This recording was made a few minutes after countershock had terminated the rhythm seen in 168 and 169 and demonstrates the cardinal features of combined accessory pathway pre-excitation and His-Purkinje conduction (fusion) in WPW. In ECGs 168 and 169 the widest and tallest R waves are seen in V1. In 171 the initial portion of the QRS is caused by accessory pathway conduction ("delta waves") best seen in V1.

* The PR intervals should be measured in leads in which the delta waves are prominent. If the pre-excitation is isoelectric, as in lead I, the PR interval measured in that lead may appear to be normal.

171: The standard limb leads are magnified to demonstrate the P-R interval as seen in these 3 leads. The onset of the P waves are aligned with a bold grid line, and the plumb line is placed on the onset of the QRS in Lead I, yielding a normal PR interval of 140 msec. However in Leads II and III an up-sloping delta wave is seen 100 msec after the onset of the P wave in III, confirming the timing of onset of activation due to accelerated AV conduction via the bypass tract.
Sinus Rhythm with First Degree AV Block (P-R 810 msec)...
or Junctional Rhythm with Retrograde P waves, rate 53
Nonspecific S-T and T waves, consider Ischemia

a) There is a seemingly constant P-R (and R-P) relationship throughout; P-R= 810
b) T waves are flat in most leads and inverted in I, aVL, and V4-6

Note: Without additional data, this ECG could either be interpreted as first degree A-V block
with a very long P-R interval or a junctional escape rhythm with retrograde P waves. The
exceptionally long P-R interval (over 800 msec) would seemingly render the first possibility
very unlikely, while the latter diagnosis is supported by a rate typical for junctional escape
rhythm and the suggestion that the P waves in III are inverted (and therefore conducted
retrograde from the junctional focus).

The 2 possible mechanisms to explain this rhythm are placed on the Ladder, and the
calipers measure the P-R interval (810 msec) if anterograde conduction is the mechanism of
this arrhythmia. The following ECG obtained on the same patient will confirm the mechanism
underlying this arrhythmia. (Hint: Measure the R-R and P-P intervals with the calipers; a
junctional rhythm should be regular, while sinus rhythm will usually show concurrent variation
in P-P and R-R intervals, while maintaining constant P-R intervals).
**178** Sinus Rhythm, rate 53  
Mobitz I (Wenckebach) A-V Block  
Nonspecific S-T and T waves, consider Ischemia

a) Upright P waves are seen in I, II, and aVF (at same rate as ECG 177)  
b) Decremental A-V conduction with non-conducted beat interval <2X R-R interval  
c) T waves are flat in most leads and inverted in I, aVL, and V4-6

Note: Progression to second degree A-V block proved in retrospect that **177** was indeed **first degree A-V block** because **178** had the same P wave rate and configuration and exhibited the classic pattern of Wenckebach, namely progressively prolonging P-R interval prior to conduction failure, a pause of shorter duration than 2 of the shortest intervals, and (on the right of the strip, after the dropped beat) decreasing R-R intervals while the P-R is prolonging.

**178**: Although **177** could have resulted from either of the two contrasting mechanisms, a measurement of the R-R intervals in **177** would have revealed slight variations in the PP and RR intervals with constant PR intervals, confirming that anterograde conduction of sinus bradycardia with first degree AV block was responsible for the rhythm in **177**. The last PR interval before the dropped beat in this ECG measured 1340 msec!
255 Sinus Rhythm, rate 85
Multiple Atrial Premature Complexes
Ashman-type Aberrancy
Interventricular Conduction Defect

a) Normal P waves with regular rhythm in beats 2-4
b) Frequent premature P waves partially obscured in T waves
c) Right bundle branch aberrancy on beats 7, 11, and 15

Note: The first premature P wave is conducted to the ventricles with a normal QRS configuration; thereafter the premature beats have an aberrant (RBBB) appearance that resembles premature ventricular beats. Typically (but not invariably) atrial premature complexes are followed by pauses that are not "compensatory" as are those seen following ventricular ectopic beats. These pauses result from a delayed appearance of the next sinus P wave. The premature atrial depolarization traverses the sino-atrial junction retrograde to reset the sinus node, and the ensuing P wave is further delayed by the time required for the next sinus impulse to traverse the sino-atrial junction anterograde to depolarize the atrium. The sinus P-P (and R-R) intervals are 710 msec, while the pauses following the premature beats are 860 msec, a difference of 150 msec caused by the combined transit times entering and leaving the sinus node. The ensuing premature P waves (beats 7, 11, and 15) thus follow in the aftermath of longer preceding P-P (and R-R) intervals, and are therefore relatively more premature and aberrantly conducted. This phenomenon (long-short-aberrancy) is often seen in atrial fibrillation (see 074), and the aberrant beats are called "Ashman beats."

255: The premature atrial P waves distort the T waves of the preceding sinus beats, and are designated by asterisks (*) on the Ladder. Aberrant (RBBB) conduction may result from absolute or relative prematurity in the timing of the premature atrial complexes. In this ECG, both mechanisms can be invoked.
Sinus Rhythm rate 88-96
Atrial Premature Complex
Ventricular Parasystole
Left Ventricular Hypertrophy

a) Upright P waves in I and aVF
b) Early P wave (9th beat) followed by pause (increase P'-P interval)
c) Frequent wide complex premature beats with equal inter-ectopic intervals
d) R in aVL 15 mm, secondary ST-T changes

Note: At first glance this ECG resembles "frequent atrial premature complexes with RBBB aberrancy" because many of the aberrant beats are preceded by P waves and the initial half of the QRS complexes resembles the native beats. However, the P waves preceding the aberrant beats are not premature, but the P-R is shortened. The one actual premature atrial complex (vertical arrow) is not followed by an aberrant QRS. The ventricular origin of the aberrant beats becomes clear when the P-R relationship is seen to vary (one of the complexes actually precedes the P wave), and each premature complex is followed by a compensatory pause. The inter-ectopic intervals are either constant (1150 msec) or a multiple of that constant (1150 X 3 = 3450), confirming that they are from an independent parasystolic focus with variable exit block. An additional confounding feature of this tracing is that the pause following the APC coincidently emulates a "compensatory pause." Ventricular Parasystole is also seen in 142.

The intervals between the sinus beats and the ectopic beats (coupling intervals) vary while the interectopic intervals are seen to be constant (1150 msec) or a multiple of that constant (1150 X 3 = 3450). The ectopic beats are therefore emanating from a pacemaker independent from and unaffected by the underlying sinus rhythm. The vertical arrow denotes the atrial premature complex which produces a normal QRS.
424 Wide Complex Tachycardia (AVNRT), rate 178
Left Axis Deviation
Left Ventricular Hypertrophy
Interventricular Conduction Delay (IVCD)

Note: This rhythm resembles ventricular tachycardia, and the only solid proof that it is actually of supraventricular origin can be seen in the next ECG (425) obtained earlier the same day during sinus rhythm in which the QRS complexes are very similar in axis, duration, and configuration. The absence of visible P waves suggests that it is atrioventricular nodal reentrant tachycardia (AVNRT), in which atrial depolarization occurs during the QRS complex, especially when the QRS is wide. The ladder indicates that anterograde conduction is down the slow pathway and retrograde reentry is via the fast pathway within the A-V node.

424: This wide complex tachycardia (QRS 140 msec) would be hard to distinguish from ventricular tachycardia if it were not for the next ECG (425) obtained from the same patient in sinus rhythm exhibiting similar QRS configurations. The initial portion of the QRS complexes in Leads II and V3-6 has a sharp rise, unlike beats of ventricular origin.
Sinus Rhythm, rate 99
Left Axis Deviation
Interventricular Conduction Delay; Probable LVH
Left Atrial Enlargement

Note: This ECG confirms the supraventricular origin of the tachycardia seen in 424 because of the close resemblance of the QRS complexes in the two tracings. The QRS complexes are wide (140 msec), but not typical of LBBB, and probably represent left ventricular hypertrophy with interventricular conduction delay (IVCD). The P waves are broad (>120 msec) suggesting left atrial enlargement providing further evidence along with left axis deviation, tall R waves and inverted T waves favoring LVH.

The QRS complexes in sinus rhythm resemble those in 424 and are compatible with left ventricular hypertrophy with interventricular conduction delay. Broad P waves in Lead II with negative terminal deflections in V1 suggest left atrial enlargement complementing the voltage and secondary T wave criteria for left ventricular hypertrophy.
Note: The cause of this wide complex tachycardia with a RBBB configuration was confirmed by recording atrial potentials from an esophageal lead (see 660) that confirmed 1:1 V-A conduction and that the notches in the initial portion of the T waves were P waves.

658: This wide complex tachycardia was well tolerated by the patient, providing an opportunity to establish its origin. The Ladder indicates 1:1 VA conduction confirmed by esophageal lead electrogram in 659. One-to-one V-A conduction could have resulted from either a junctional or ventricular tachycardia.
Ventricular Tachycardia, rate 138
Esophageal Lead Records Ventriculo-Atrial Conduction 1:1
Esophageal Lead after Adenosine Causes VA Conduction Block

The sharp biphasic P waves are seen at the end of the QRS in the Esophageal Lead recording when plumb lines bracket the QRS on the surface leads. When adenosine was administered, VA conduction was blocked while the ventricular rate remained rapid, indicating that the atrium was not responsible for or in a reentry circuit responsible for the tachycardia. The diagnosis of ventricular tachycardia was later re-confirmed during an electrophysiologic investigation during which a left ventricular focus was localized and successfully ablated.
**701** Sinus Rhythm **rate 87**

WPW

a) Upright P waves in I and aVF  
b) Prominent "delta waves" in V1-3

Note: This patient's ECGs were incorrectly read by many observers who thought that they represented either "infero-posterior infarction" or "right ventricular hypertrophy" because of the prominent Q waves in the inferior leads and the prominent R waves in V1-3. An astute cardiology fellow administered adenosine (see 700) and established the correct diagnosis of WPW. A confounding finding on this ECG is the seemingly normal P-R interval; it is actually at the lower limit of normal at 120 msec.

701: WPW is indicated by the slurred upstroke of the QRS complexes seen well in V1-3. The P-R interval was measured by placing the onset of the P wave on a bold grid line and the plumb line on the onset of the QRS. The interval is 120 msec, the lower limit of normal AV conduction. Q waves are seen in the inferior leads, caused by pre-excitation through the bypass tract.
Adenosine Administration causes Transient...
Sino-Atrial Block
Atrioventricular Block
"Pure" Bypass Tract Conduction Revealed

Note: This 15 second continuous rhythm strip demonstrates slowing of the atrial rate with an irregular rhythm resulting from 3:2 and 4:3 sino-atrial Wenckebach ("group beating" with pauses), as the QRS complexes progressively widen and resemble ventricular ectopic beats, but preceded by P waves. The P-R interval remains constant throughout. Toward the end of the strip the atrial rate increases, the rhythm becomes regular, and the QRS complexes undergo a transition back to their original configuration.

The presence of WPW was uncovered by the administration of 12 mg Adenosine which progressively slowed the atrium, possibly by blocking sino-atrial conduction, and completely blocking atrioventricular conduction to reveal depolarization via the accessory bypass tract. The P-R interval remains constant, indicating that the initial portion of the QRS complex was activated by the bypass tract before Adenosine was administered and after its effect was dissipated.
Long Q-T Interval
Sinus Rhythm with Frequent Ventricular Premature Complexes leading to...
Fusion Beats and R-on-T leading to...
Non Sustained Ventricular Tachycardia (*Torsades de Pointes*) Rate 165 and...
"Ventriculo-Atrial Wenckebach"

Note: A catheter electrode was placed in the right atrium to record an electrogram to aid in
diagnosis and treatment of a young woman that had frequent syncopal episodes after taking
her mother's thiazide diuretic pills for pre-menstrual bloating. The long Q-T interval was
augmented by the medication, and frequent premature beats led to fusion beats (\(F\)) and R-on-
T triggers that produced ventricular tachycardia. The episode illustrated was initiated by R-on-
T (arrow), and lasted about 10 seconds and spontaneously reverted to sinus tachycardia. The
Q-T prolongation is best seen on the first beat after termination of tachycardia. *Torsades de
Pointes* ("twisting of the points") is a form of ventricular tachycardia characterized by
spontaneous spiraling of the QRS vector in a given lead from upright to inverted and back as
seen best in these rhythm strips in \(V_1\). *Torsades de Pointes* (plural s in both words is silent)
can be fatal, and is notoriously unresponsive to (and often caused by) antiarrhythmic drugs. In
this patient, the rhythm was stabilized by administration of atropine which produced sinus
tachycardia, preempting the ventricular ectopic beats and shortening the Q-T interval while the
serum potassium level was restored to 4.5 mEq/L from an initial value of 2.8.

702: The **intracardiac Right Atrial** electrogram recorded the P waves as discrete spikes that
can be distinguished from the wider QRS complexes. Fusion beats (\(F\)) occur when ventricular
ectopic beats occur after sinus Ps. When a ventricular ectopic complex occurs within the T
wave of the second fusion beat (arrow next to \(F\)), *Torsades* is initiated. The atrium accelerates
and exhibits periodic pauses resulting from decremental retrograde conduction from ventricle
to atrium with periodic blocked beats; V-A Wenckebach. Sinus rhythm resumed after the
tachycardia spontaneously terminated. The prolonged T waves are especially prominent in the
first beat of sinus rhythm because of the long R-R interval.
703 Ventricular Tachycardia, rate 126
Atrial Rate 72; Atrioventricular Dissociation,
Fusion Beats

Note: The Lead II rhythm strip was not recorded concurrently with the 12 lead ECG

703: Some P waves are evident; hidden P waves are found by setting the calipers. A conducted (sinus) beat (S) is the narrowest QRS, preceded by P. Fusion (F) beats are identified here by their flat T waves and preceded by P. All unmarked beats are ventricular.

See if you can identify sinus (conducted), fusion, and ventricular beats in V1-V3 in the 12 Lead ECG. Note: It was not recorded concurrently with the Lead II rhythm strip.