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Philips DXL ECG Algorithm
Physician's Guide

Notice

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About this Guide

This *Physician's Guide* explains how ECG signals are analyzed by the Philips DXL 16-Lead ECG Algorithm.

NOTE **No automated analysis is completely reliable. Computerized ECG analysis should always be reviewed by a qualified physician.**

Who Should Read this Physician's Guide?

This Physician's Guide is intended for physicians who overread ECGs interpreted by the Philips DXL ECG Algorithm. It also may be of interest to other health care professionals who want to know more about ECG interpretation.

NOTE **This Physician's Guide describes features that may not be available on all Philips equipment. Refer to the documentation supplied with your particular product to learn more about available features.**

Document Conventions

The following conventions are used in the *Physician's Guide*.

NOTE **Notes contain additional important information about a topic.**

TIP **A Tip contains suggested information on using a particular feature.**

Menu items and button names appear in bold no-serif font. Example: Touch the **Setup** button.

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Philips DXL ECG Algorithm

Introduction

Electrocardiography is now more than 100 years old and has become an essential diagnostic tool that is continually being refined and further developed[1]. Early single-channel analog machines, first with photo galvanometers and then later with direct writing galvanometers, have evolved into multi-channel simultaneous digital acquisition systems with a variety of storage and report possibilities. Digital systems enable computerized measurement and interpretation of the acquired signals.

Development of computer-assisted ECG analysis began in the 1960s. Initially used in research facilities, computer interpretation has become an accepted tool to aid physicians in arriving at a final interpretation based on clinical data and a review of the findings.

The adult ECG Criteria Program began in 1971 as a combined development effort between engineers and a worldwide panel of cardiologists. Extensive pediatric analysis was added in 1990. Since then, the Philips algorithm has undergone several modifications and enhancements to not only take advantage of new computer technology and advanced developments in electrocardiography, but also to incorporate revised guidelines proposed by international committees.

The Philips DXL ECG Algorithm provides an analysis of the amplitudes, durations, and morphologies of the ECG waveforms and the associated rhythm. ECG waveform analysis is based on standard criteria for interpretation of these parameters, calculations of the electrical axis, and the relationship between leads.

The algorithm is highly age and gender specific. Patient age and gender are used throughout the program to define normal limits for heart rate, axis deviation, time intervals, and voltage values for interpretation accuracy in tachycardia, bradycardia, prolongation or shortening of PR and QT intervals, hypertrophy, early repolarization, ischemia, and myocardial infarction.

1. W B Fye. “A History of the Origin, Evolution, and Impact of Electrocardiography.” *American Journal of Cardiology* 73:937-949, 1994.

Adult criteria apply if the patient age entered is 16 years old or older, or if no age is specified. Pediatric criteria apply if the patient age entered is younger than 16 years of age. Twelve different age ranges are used for the pediatric criteria to account for the rapid changes that occur in the first few days to months of life.

While increasingly detailed and well developed, no automated analysis is completely reliable, and computerized ECG analysis should always be reviewed by a qualified physician.

The interpreted ECG is a tool to assist the physician in making a clinical diagnosis in conjunction with the physician's knowledge of the patient, the results of the physical examination, and other findings. For example, symmetrical T wave inversion often accompanies severe Left Ventricular Hypertrophy, but may also represent ischemia or a central nervous system event. Without knowledge of the clinical background, any interpretation must be less specific. Serial tracings may help in some cases. For example, ventricular aneurysms may present the electrocardiographic signs typical of evolving myocardial infarction. Without history and previous tracings, there is no way to differentiate the two conditions.

It is also true that humans are better at recognizing artifact and ignoring its effects than current computer programs. Avoiding artifact in the first place will reduce the need for corrections by the overreader. In addition, different overreaders may disagree among themselves about a particular record; a computerized analysis can provide only one interpretation.

What's New in the Philips DXL ECG Algorithm vPH110C?

Detailed information about changes to the algorithm are provided in Chapter 7, "New Features in the DXL ECG Algorithm, vPH110C."

- The algorithm version has changed from PH100B to PH110C. ECG reports print the new version code in the lower right-hand corner.
- The DXL algorithm can now detect STEMI in the presence of LBBB, RBBB, and LVH using proprietary vectorcardiographic criteria.

  STEMI detection is often not advised for conditions that generate secondary ST elevation (STE), such as LBBB, RBBB, and LVH. When these conditions are recognized, it is difficult to separate secondary STE due to the condition from a combination of primary STE due to ischemia and secondary STE due to BBB or LVH.
- The algorithm can be now be configured to lower sensitivity to detect STEMI in the presence of ST elevation confounders such as LBBB, RBBB, and LVH for emergency medical services (EMS) applications where false positives can lead to inappropriate cath lab activation.
- New criteria based on ST segment elevation and depression are used not only to identify the culprit coronary artery in STEMI but also to detect a proximal occlusion in the case of left anterior descending coronary artery.

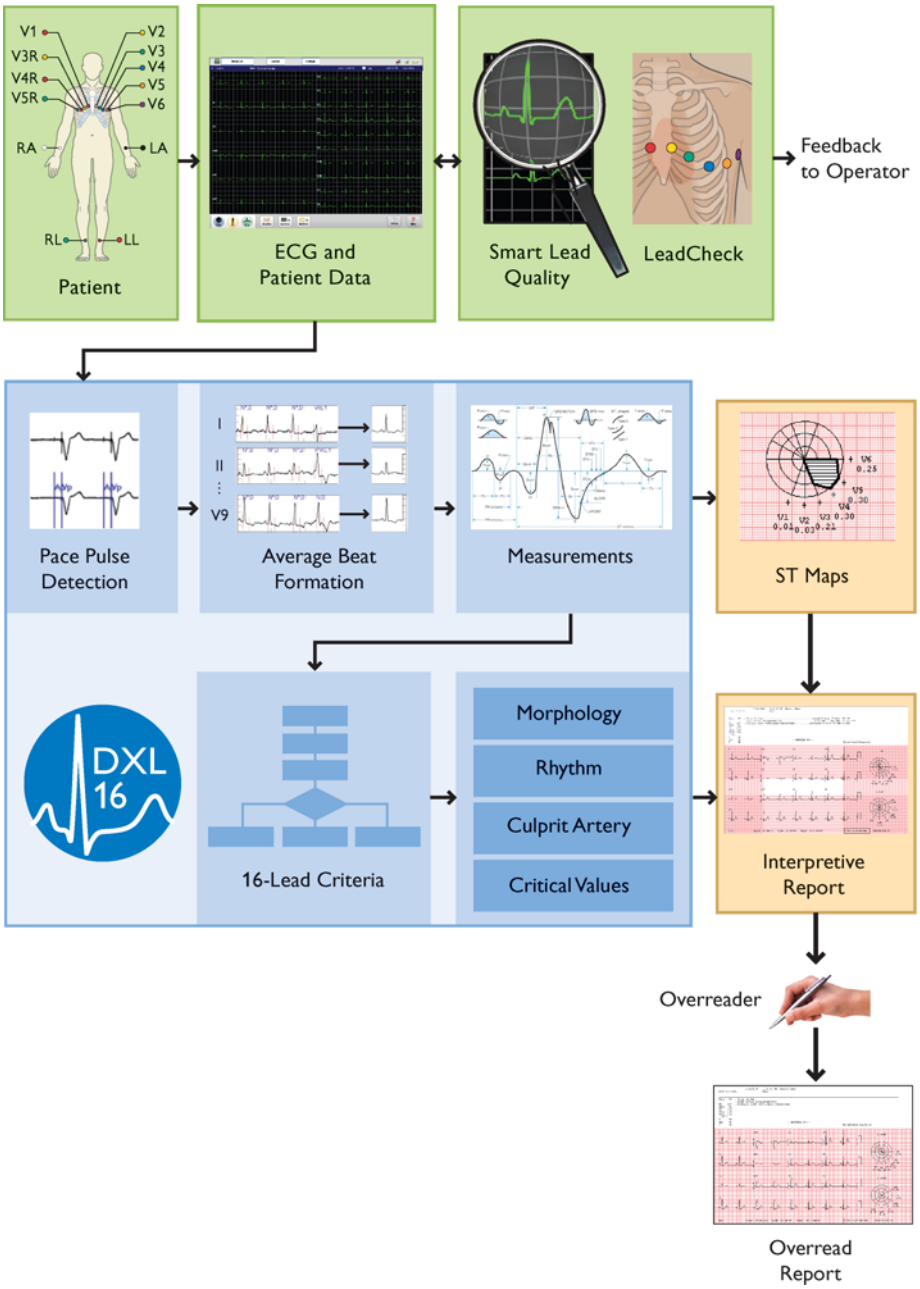
- Deeply inverted T-waves in leads V2, V3, and V4 are interpreted as either proximal LAD occlusion or cerebrovascular accident (CVA)[2].
- You now have four formula options for heart rate correction of QT interval:
  - Bazett
  - Fridericia
  - Hodges
  - Framingham
- Paced rhythm interpretation is extended to include detection of biventricular pacing by Cardiac Resynchronization Therapy (CRT) devices.
- Estimated left ventricular myocardial infarction size by Selvester score is available as a statement, if desired.

How the Philips DXL ECG Algorithm Processes Data

The Philips DXL ECG Algorithm produces precise and consistent ECG measurements that are used to generate interpretive statements. The process begins with the simultaneous acquisition of the twelve conventional leads and any extended right sided or posterior leads, along with patient demographic information.

2. This is a recommendation in: Wagner GS, Macfarlane P, et al. “Standardization and Interpretation of the Electrocardiogram, Part VI Acute Ischemia/Infarction.” *Circulation* 2009; 119:10. Available online at: http://circ.ahajournals.org.

Figure 1-1 The Philips DXL ECG Algorithm Analysis Process



The algorithm follows five steps to produce the interpreted ECG report. Each of these five steps are discussed in greater detail in the following sections.

Table 1-1 Steps to Produce an Interpreted ECG Report

Step	Description	See
Monitoring waveform quality	Examines the technical quality of each ECG lead	page 1-5
Waveform recognition	Locates and identifies the various waveform components	page 1-20
Formation of representative beat	Forms a representative beat for each lead	page 1-22
Generating comprehensive measurements	Measures each component of the representative waveforms and performs basic rhythm analysis, producing a comprehensive set of measurements	page 1-22
Interpretation of the result	Uses extended measurements and entered patient information (age, gender, prescription medication) to select interpretive statements from the program	page 1-30

Monitoring Waveform Quality

Computer-assisted ECG analysis begins by obtaining accurate ECG waveforms through simultaneously acquiring and analyzing 12 or more ECG leads. The analog ECG signal at the body surface is digitized by the Patient Interface Module (PIM), which connects to the applied electrodes.

The ECG waveform data is converted to digital form using multi-channel sigma-delta converters sampled at 8000 sps, which are then downsampled to the ECG range. The converter has the capability of 24-bit resolution (one part in 16,777,216) while eliminating electronic noise. Higher sampling frequencies are sometimes employed for paced spike detection, but the Philips pacing spike detector provides superior performance at much lower frequencies, rendering this unnecessary.

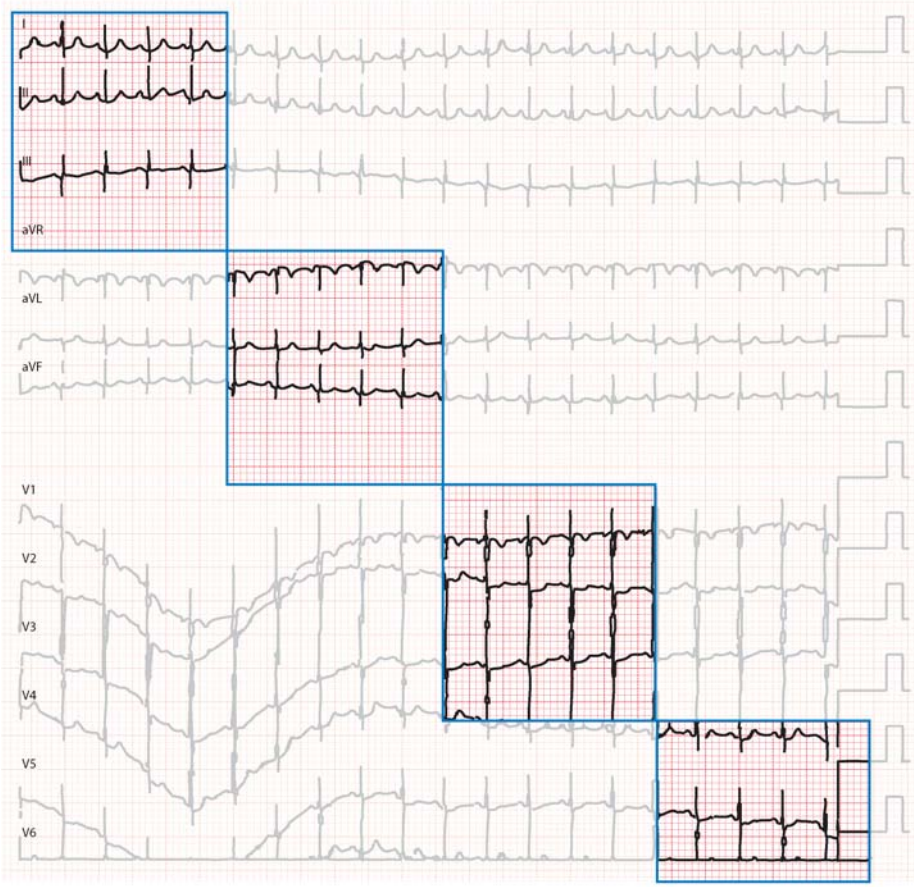
Philips equipment monitors ECG trace quality from the time of lead attachment, to ECG acquisition, and throughout the analysis process. Signal quality feedback to the operator helps to ensure the highest possible quality ECG trace.

During analysis, the ECG is analyzed for muscle artifact, AC noise, baseline wander, and leads-off. Any noise problems not corrected by the operator are described in the interpretive statements on the ECG report. Because only a portion of the ECG is typically printed, the cause of the artifact is not always visible. When viewed within the TraceMaster ECG Management System, the

complete ECG record can be displayed to further identify where the quality problems reside.

Figure 1-2 on page 1-6 clearly demonstrates significant baseline wander in the precordial leads that is resolved during the last five seconds of the ECG, so the baseline wander will not appear on the standard 3x4 report.

Figure 1-2 Baseline Wander in ECG Signal (highlighted areas appear on 3x4 report)



If signal quality issues are severe, the acquisition device may not generate nor print an ECG report. If signal quality issues are significant enough as to prevent ECG analysis, the ECG may be printed without interpretation. The operator must then correct the signal quality problem and retake the ECG.

Thorough and effective patient preparation helps to eliminate most signal quality problems.

The following sections describe these signal quality issues in greater detail.

Identifying Lead Reversals

Incorrect electrode placement and incorrect lead wire connections may cause subtle or obvious quality problems. The DXL Algorithm offers an optional lead reversal detection feature that attempts to detect the most common limb and

precordial lead reversals. The statements generated by the lead reversal algorithm alert the operator to a potential lead reversal include the following possibilities.

- Right arm and left leg electrode reversal
- Right arm and left arm electrode reversal
- Probable extremity electrode reversal
- Probable precordial electrode reversal

Two types of lead reversals are particularly challenging to detect:

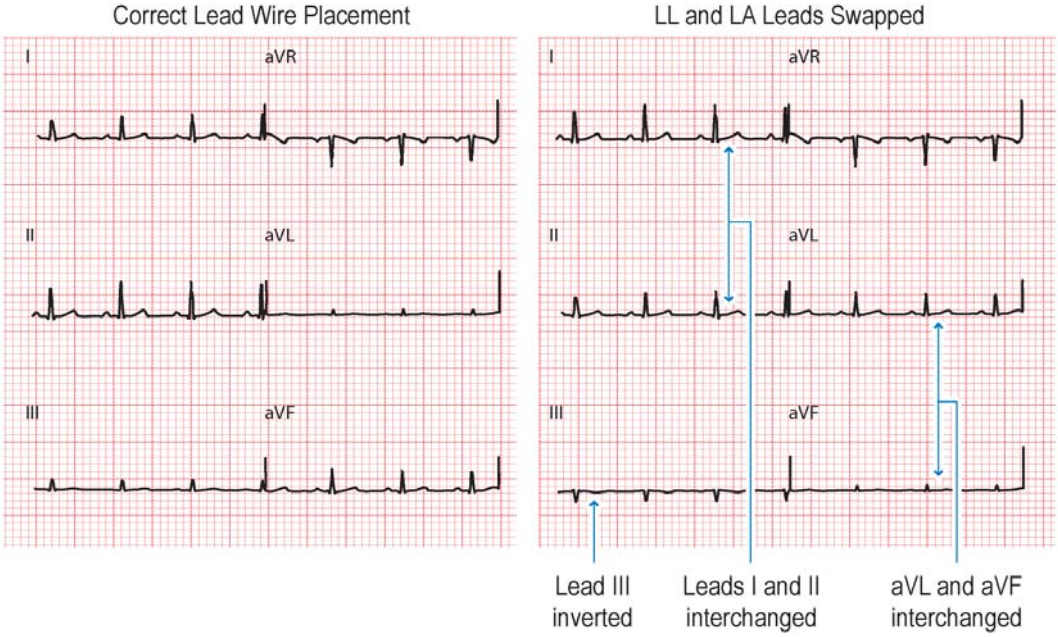
- Reversal of the left arm and left leg lead wires; this instance is not detected by the lead reversal algorithm
- Precordial lead reversals, particularly lead reversals of adjacent precordial leads

Left Arm and Left Leg Lead Reversals

The challenge with left arm and left leg lead placement is that both correct and incorrect lead wire connections may produce a normal looking tracing. Figure 1-3 on page 1-7 shows the limb leads with an LL-LA lead reversal located on the right side. Note in Figure 1-3 that leads aVL and aVF are also reversed, lead III is inverted, and leads I and II have been reversed.

This type of limb lead reversal is difficult to detect, and usually is only detected when comparing serial tracings.

Figure 1-3 Left Arm and Left Leg Lead Reversal



Precordial Lead Reversals

Some precordial lead reversals are also difficult to detect. These lead reversals most commonly occur in adjacent leads and produce a noticeable “drop out” effect in R wave progression; however, this situation can also occur with infarcts. A drop out that disappears on a close serial tracing is almost always due to one of these subtle lead reversals.

Verifying correct lead connections at the time of ECG acquisition provides the best protection against these lead reversals.

Enabling the optional lead reversal detection feature available with the DXL Algorithm on the acquisition equipment will provide a lead reversal detection warning in most situations when this type of lead reversal occurs.

Reducing Artifact

Electrical (alternating current) interference, patient respiration, patient movement, and muscle tremors may add noise and artifact to the ECG signal. Poor quality electrodes or inadequate patient preparation may also degrade the ECG signal.

Alternating current interference in the ECG signal may be defined as one of two following types: *common mode* and *differential mode*.

Common Mode

Some noise sources that interfere with the ECG signal affect all of the electrodes attached to the patient. These common noise sources are removed from the ECG by input circuitry as the signal is acquired and digitized. The amount by which these common mode signals are reduced is referred to as the *common mode rejection ratio*. The common mode rejection ratio for Philips input circuitry meets or exceeds current AAMI and IEC standards. This feature avoids most of the alternating current noise that is produced by motors, lighting, and electrical appliances. This is highly effective when all the electrodes have good contact with the patient, but is less effective if one or more electrodes have poor contact with the patient. Poor electrode contact is usually what has occurred when power line interference is seen in an ECG recording.

Differential Mode

The magnetic fields associated with electrical power interact with the lead wires, which act as miniature antennas. These fields induce electrical signals that appear as high frequency noise on the ECG. The amount of distortion differs from lead to lead, depending on the size of any loop created by the lead wire and its orientation. A good way to prevent distortion is to align all the lead wires with the patient’s body along the head-to-foot axis. Poor contact with the skin for a particular electrode can provide a much stronger signal than the common mode, and thus, produce electrical interference on specific leads.

Recognizing Artifacts

Artifact can be introduced from a variety of sources:

- Power line interference
- Muscles, caused by shivering or disease-induced tremors
- Respiratory, from spontaneous respiration or ventilator
- Baseline drift, generally due to poor electrode/skin contact
- Baseline wander, generally due to poor electrode/skin contact or to patient movement
- Mechanical, generally due to pulling on the electrode cables

The following sections describe each of these circumstances in detail.

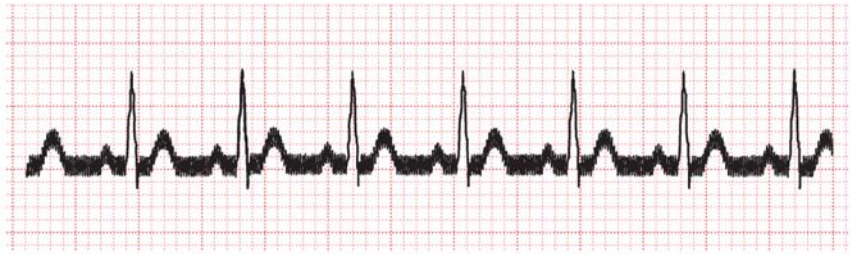
Power line interference

Power line interference originates from 50 or 60 cycle/second alternating current (depending on the local country standard) and usually occurs as a result of a combination of electrical appliances near the patient in combination with poor patient electrode contact.

The interference causes a fast regular waveform that can produce false notching in the QRS complexes, in addition to a thick baseline. Replacing electrodes and removing nearby appliances, or running these appliances on battery power only usually resolves the power line interference issue.

Note that fluorescent lighting may produce interference at three times the rate of the power line (150 or 180 cycles/second).

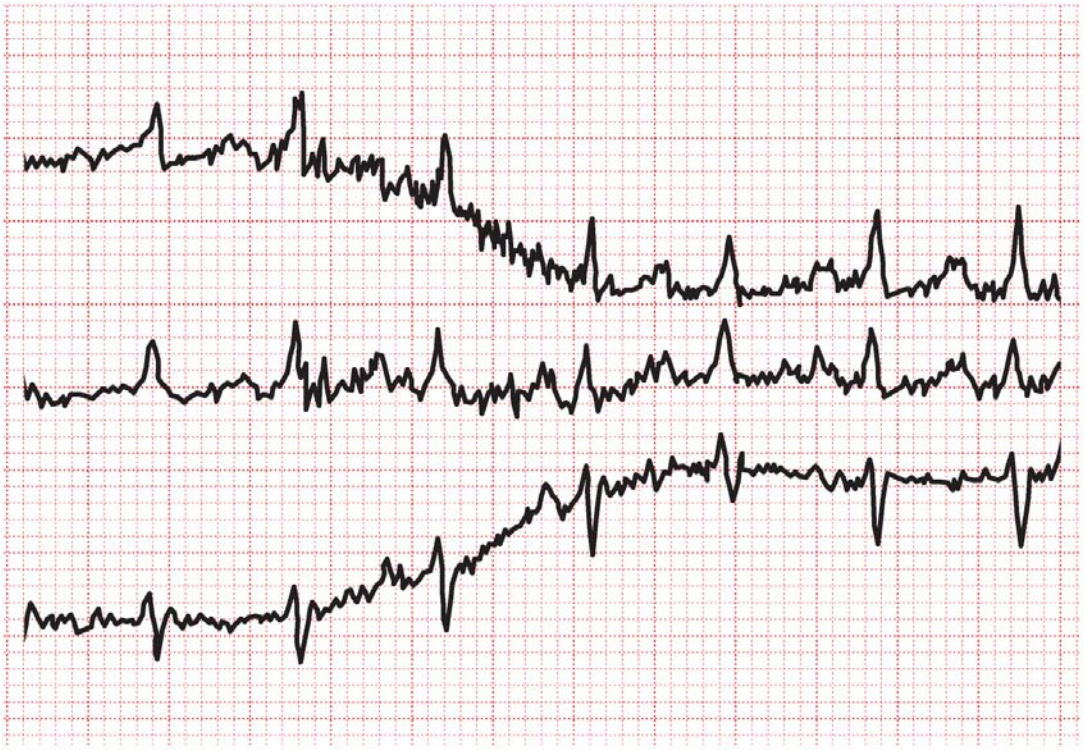
Figure 1-4 Power line interference (50/60 cycles per second)



Muscle artifact

Muscle artifact is usually caused by shivering or tremors due to Parkinson's disease. If it is possible to warm the patient, this may help to reduce shivering. The effect of tremors on the ECG signal may be reduced by moving the limb electrodes closer toward the shoulders or to the hips. Moving the electrodes closer to the shoulders or hips may cause a small reduction of amplitudes and may affect interpretation, however, this will render the ECG more interpretable overall.

Figure 1-5 Muscle artifact and wander

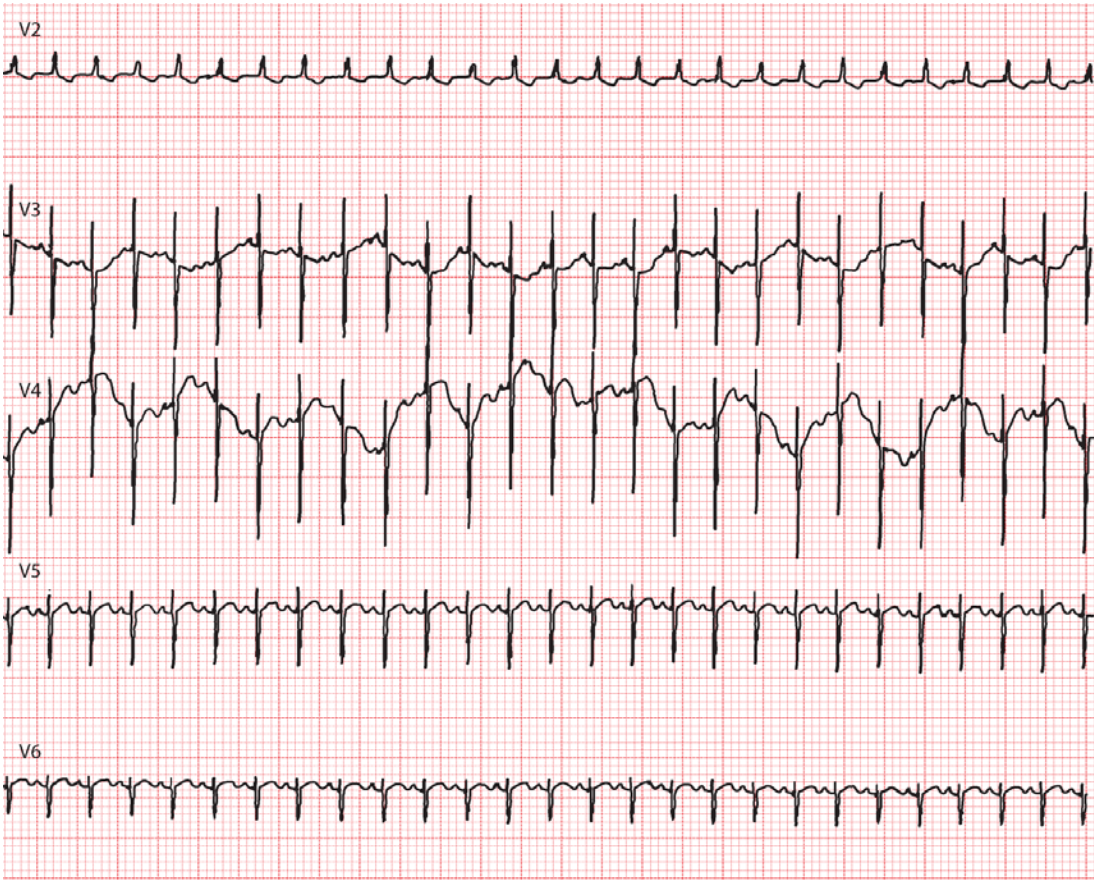


Respiratory Artifact

Respiratory artifact may be caused by spontaneous respiration, or respiration assisted by a ventilator. Intermittent mandatory ventilation provides occasional very large inflations interspersed with the patient's generally smaller spontaneous breaths. This can produce a very large change in heart position if the mandatory breath occurs during the ten second ECG recording. This change is almost impossible to detect on a typical 3x4 report unless rhythm leads have been recorded for a full ten seconds.

More typical respiratory artifact produces a regular oscillation of the baseline superimposed on the ECG, and can be severe with Kussmaul or Cheyne-Stokes breathing. This may cause significant morphological variation that interferes with human or computer ECG interpretation.

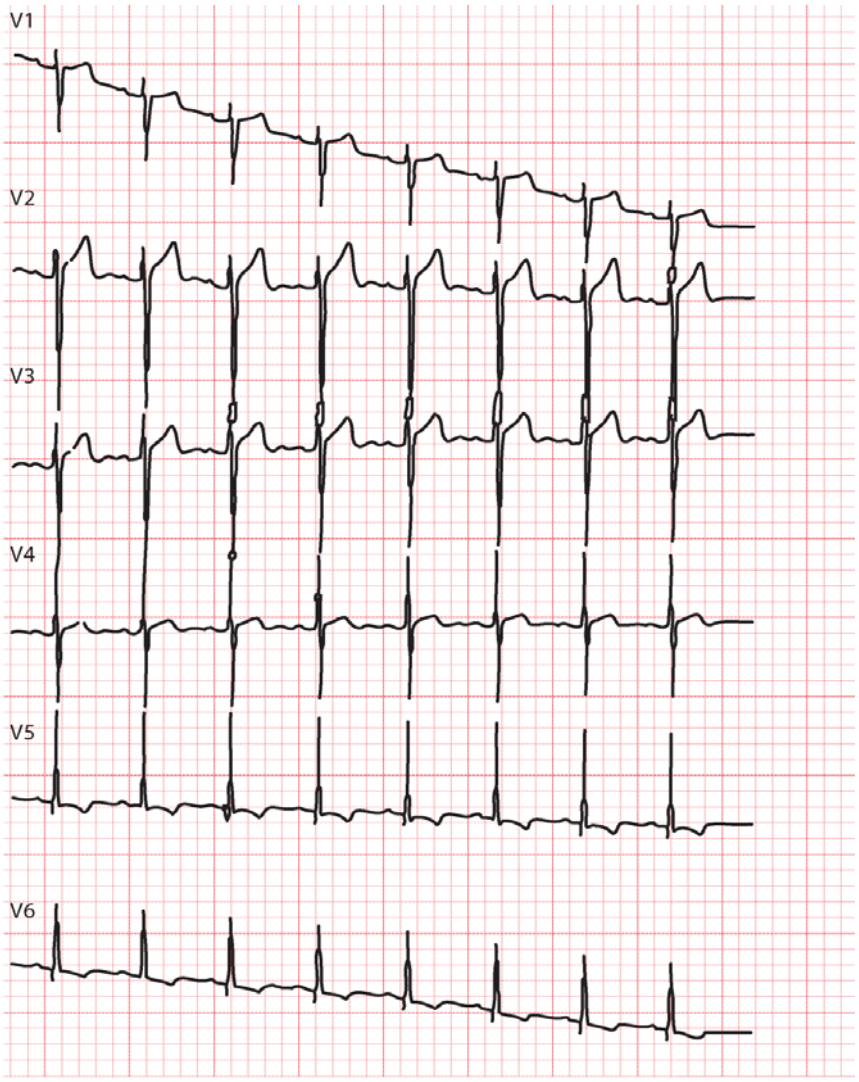
Figure 1-6 Respiratory Artifact



Baseline Drift

Baseline drift is a sloping line superimposed on a particular lead. Figure 1-7 on page 1-12 shows this condition occurring only in leads V1 and V6, where the electrodes have made poor skin contact and are slowly changing their characteristics. Removing chest hair from the patient and possibly replacing or repositioning the electrode(s) will often resolve this problem.

Figure 1-7 Baseline drift

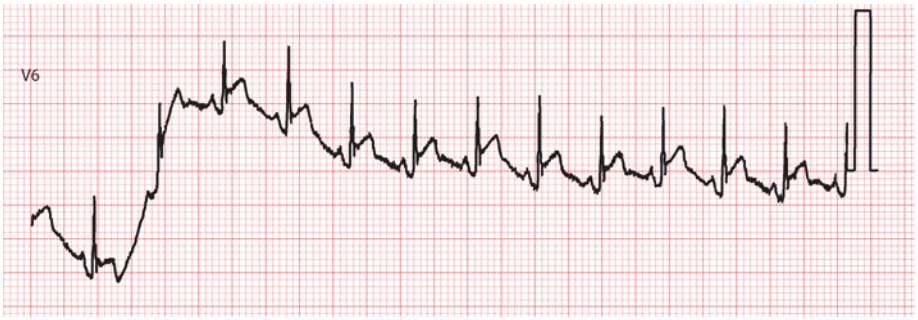


Baseline Wander Artifact

Baseline wander artifact can be varied in nature, but the most common forms of baseline wander are the result of poor electrode contact or patient movement. These types of baseline wander are not repetitive (like respiratory artifact) and can occur at any point in the ECG tracing. Previously, we have presented

examples of respiratory wander and of electrode drift artifacts. It should be noted that only respiratory wander artifact is truly resistant to improvement, but other types of baseline wander can be corrected. While it may be difficult to keep pediatric or neonatal patients calm and quiet, adult patients can generally cooperate with attempts to limit patient movement that causes baseline wander.

Figure 1-8 Severe Baseline wander



Mechanical Artifacts

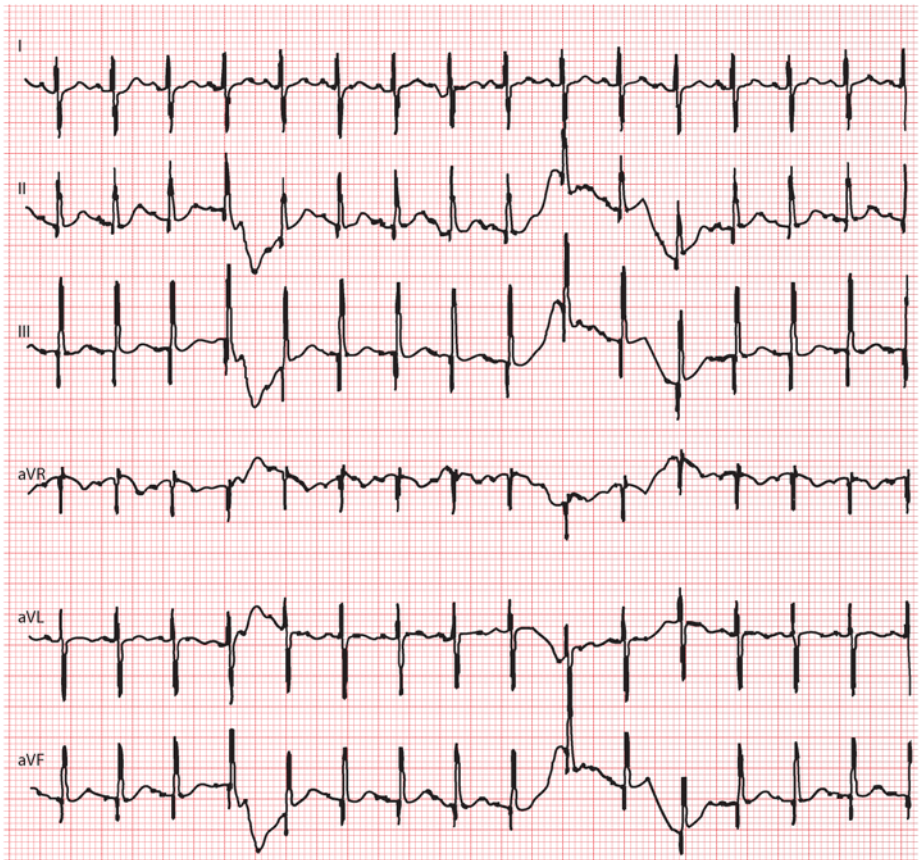
Mechanical artifacts usually occur due to patient movement and the subsequent pulling on the lead wires. Moving the patient interface module so that all of the lead wires and the patient data cable are loose and not too taut may help to alleviate these mechanical artifacts.

It is often easy to identify which electrodes or lead wires are causing the problem:

- A chest lead artifact will appear in just that individual lead.
- Limb leads artifacts will appear as combination patterns.
- If the artifact is largest in one of the augmented leads, then a specific electrode is causing the problem. The two associated limb leads will also have large artifacts (I and III for aVL, I and II for aVR, and II, III for aVF).

Figure 1-9 on page 1-14 illustrates that there is an issue with the left leg electrode. Note that the other two augmented leads will also show artifacts, but these artifacts will be of a smaller magnitude.

Figure 1-9 Mechanical artifact



Using Filters

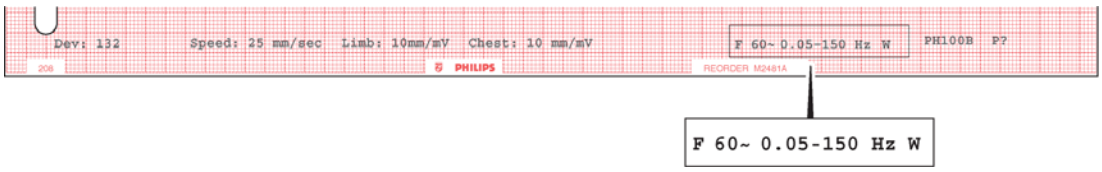
A variety of noise sources may degrade the reproduction of the ECG signal. Philips acquisition devices offer a sophisticated set of digital filters that can be selected by the operator, or can be enabled during device configuration. These filters are used to optimize the displayed or printed ECG waveform.

With the exception of the AC filter, which is highly selective, there is trade off between fidelity and clarity of the ECG trace when a filter is applied. The more filtering that is applied, the greater the possibility of removing ECG signal details.

The lower right corner of the printed ECG report includes a box that contains information about the filtering options used on the ECG.

NOTE **While all filters affect displayed and printed ECGs, the DXL Algorithm always receives, stores, and analyzes data at 0.05 to 150 Hz.**

Figure 1-10 Filter Information Box on the Printed ECG Report



Artifact Filter

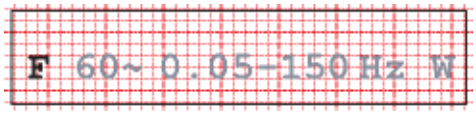
The Artifact filter removes skeletal muscle artifact. This noise source is the most difficult to eliminate because it possesses the same frequencies as legitimate ECG signals. While this filter eliminates skeletal muscle artifact, it also reduces all high frequency components of the ECG. This effect may make it impossible to detect pacemaker pulses, can cause visual underestimation of signal amplitudes, and can also render QRS notching invisible.

The filter removes up to 50 µV of signals in the 5 Hz to 150 Hz frequency range. This may affect P waves and the entire QRS-T complex.

Use the Artifact filter ***only*** for ECGs that would be unreadable due to significant levels of muscle artifact. Using the filter should provide at least rhythm information, although paced pulses may only be evident by looking at the markers that are produced on the ECG report.

When the Artifact filter is enabled, the **F** symbol is included in the filter information box at the lower right corner of the printed ECG report.

Figure 1-11 Artifact Filter Symbol on Printed ECG Report

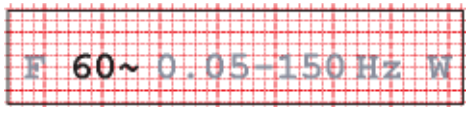


AC Filter

The AC filter removes interference created by the magnetic fields associated with electrical power interacting with the lead wires. The frequency of the AC interference is stable at 60 or 50 Hz. The AC filter removes the AC noise and leaves the ECG signal intact. The line frequency of 60 or 50 Hz is selected during the configuration of the acquisition device.

When the AC filter is used, the AC filter symbol is included in the filter information box at the lower right corner of the printed ECG report.

Figure 1-12 AC Filter Symbol on Printed ECG Report



Frequency Response Filters

These filters suppress frequencies at the high and low ends of the ECG signal spectrum. The available low frequency response filter settings are 40, 100, and 150 Hz.

In 1989, the American Heart Association recommended that frequencies up to 125 Hz be recorded for adult ECGs, and that frequencies up to 150 Hz be recorded for pediatric ECGs[3].

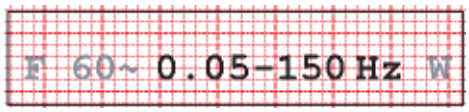
Changing the low-pass frequency filter to 40 or 100 Hz permits frequencies below these values to remain in the report and results in a smoother-looking ECG waveform, but eliminates some fine detail in the signal. Small deflections, notches, and slurs may be distorted or may disappear if one of these filters is applied.

The high-pass frequency response filter settings are 0.05, 0.15, and 0.5 Hz. Using this filter permits frequencies above the selected value to appear in the ECG report, and this filter suppresses frequencies below the selected value.

NOTE **When the baseline wander filter is enabled, the high-frequency response filter is automatically set to 0.5. It is recommended that the 0.15 high frequency response filter setting be used for all other ECGs. See “Baseline Wander Filter” on page 1-16 for more information.**

The frequency response of the ECG is included in the filter information box at the lower right corner of the printed ECG. The DXL Algorithm always uses 0.05 to 150 Hz bandwidth for maximum fidelity. The maximum fidelity waveform is always stored in the permanent record.

Figure 1-13 Frequency Response Filter on the ECG Report



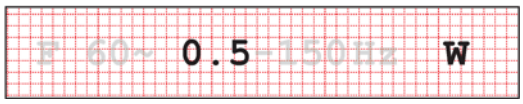
Baseline Wander Filter

Baseline wander is the slow (typically 0.1 – 0.2 Hz) drifting of the ECG baseline up or down during ECG recording. Baseline wander may result from patient respiration or from other sources. Severe baseline wander may make it difficult to determine the true wave shapes in the ECG.

Effective baseline wander suppression techniques do not distort the ST segment. While the highest frequency response limit of 0.05 Hz (recommended for normal use) eliminates baseline wander from most ECGs, additional suppression may be required. Enabling the baseline wander filter suppresses all frequencies below 0.5.

3. Bailey JJ, Berson AS, Garson A, Horan LG, Macfarlane PW, Mortara DW, Zywietz C. “Recommendations for Standardization and Specifications in Automated Electrocardiography: Bandwidth and Digital Signal Processing.” *Circulation* 81:730-739 (1990).

Figure 1-14 Baseline Wander Filter on the ECG Report

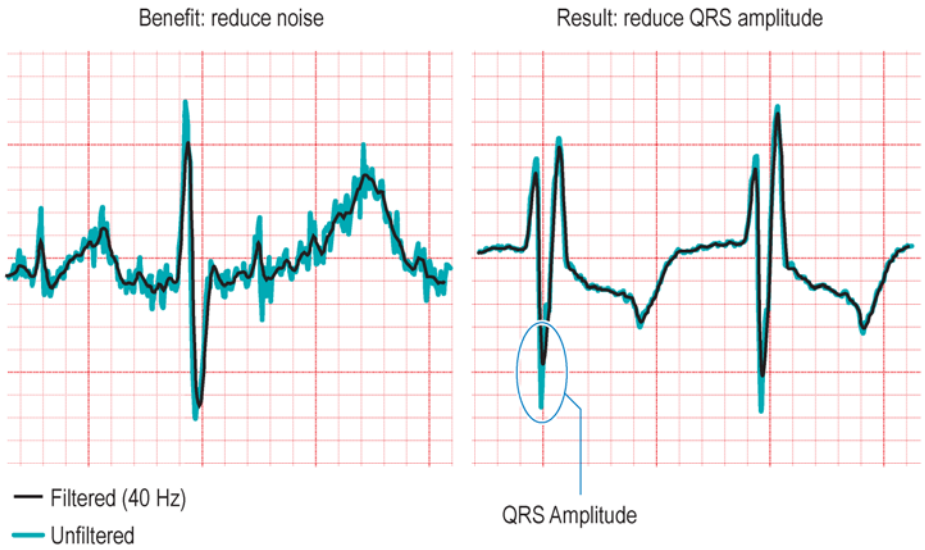


CAUTION A 0.5 Hz baseline wander filter that may distort the ST segment is used during continuous ECG recording in Rhythm mode. *Do not* attempt to interpret the contour aspects of rhythm ECGs at this setting. If contour analysis is important in Rhythm mode, use the 0.05 Hz Rhythm high-pass frequency response setting that minimizes the ST segment distortion. Rhythm characteristics of the ECG are accurately recorded, regardless of the low-pass frequency setting in Rhythm mode.

Negative Effects of Filtering on Waveforms

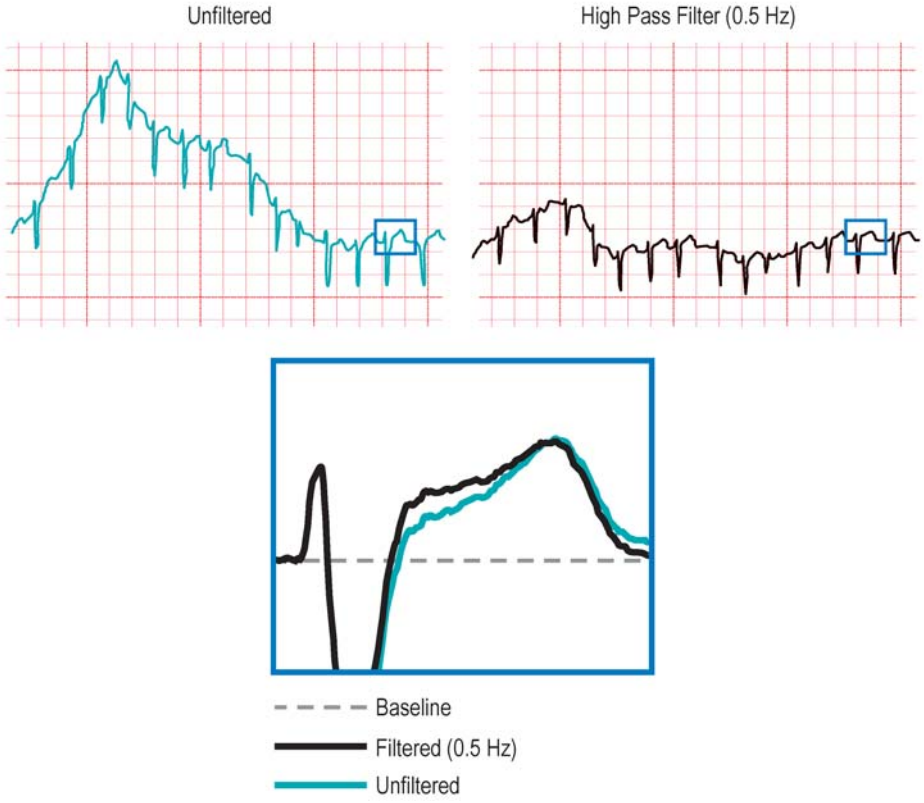
The following section describes how various filters described in the previous section can affect ECG traces. While low pass filters successfully reduce noise in ECG traces, they also reduce the QRS amplitude as shown in Figure 1-15 on page 1-17.

Figure 1-15 Effect of Low Pass Filtering on QRS Amplitude



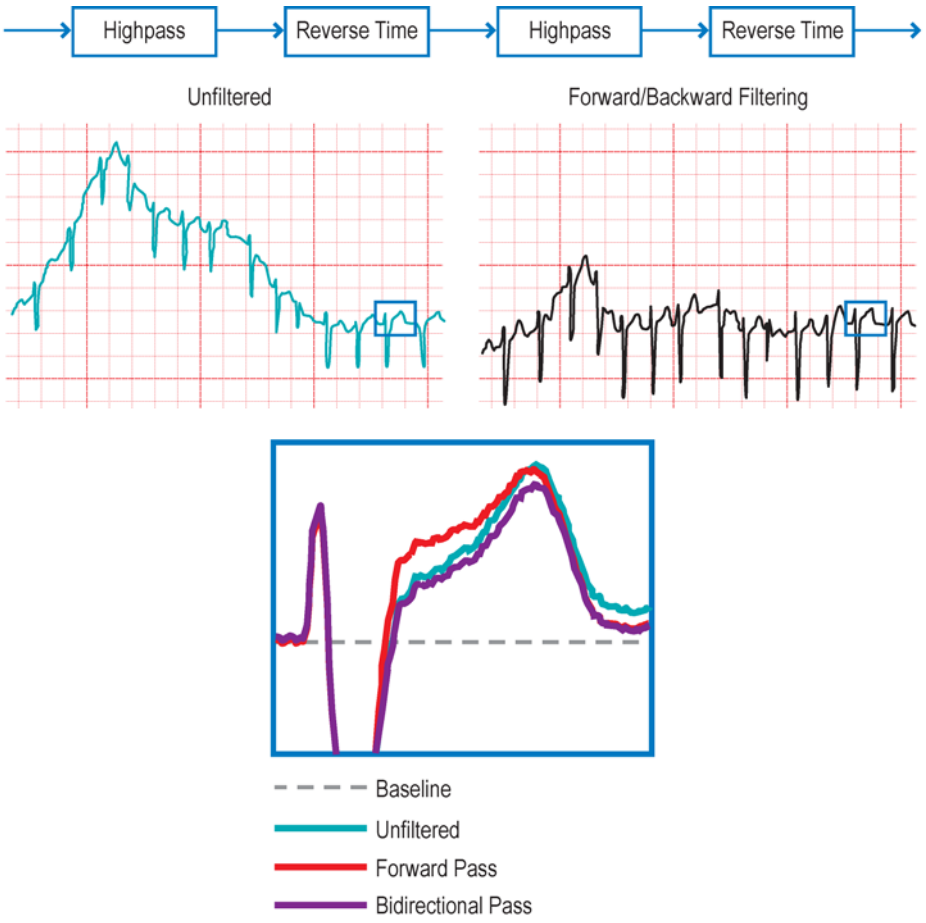
High-pass filters (0.5 Hz) reduce baseline wander, but also introduce ST distortion as shown in Figure 1-16 on page 1-18.

Figure 1-16 High Pass Filter Introduces ST Distortion



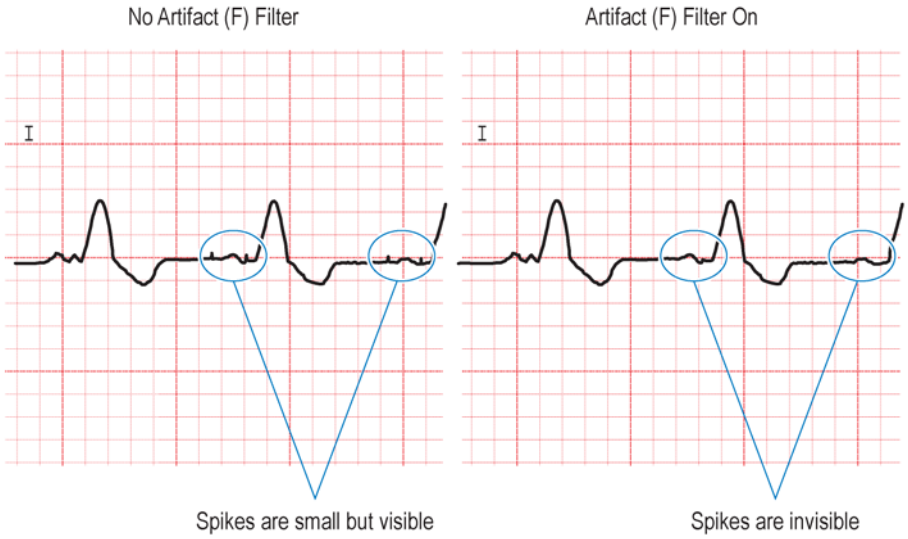
For previously captured data, a technique of forward/backward filtering removes most of the distortion, as seen in Figure 1-17 on page 1-19. However, this technique cannot be used on continuous real time data.

Figure 1-17 Effects of Forward and Backward Filtering on Stored ECG Data



Artifact filters remove many high frequencies, but this can result in the disappearance of pacemaker pulses from modern low-amplitude pacemakers. In Figure 1-18 on page 1-20, the patient has an AV sequential pacemaker. The pacer spikes are rendered invisible when using the Artifact filter.

Figure 1-18 Effects of Artifact Filter on Pacemaker Pulses



Avoiding Artifacts

Most artifacts are due to poor electrode contact with the patient's skin. The following techniques help to ensure good electrode contact in order to reduce the risk of introducing artifact into the ECG signal.

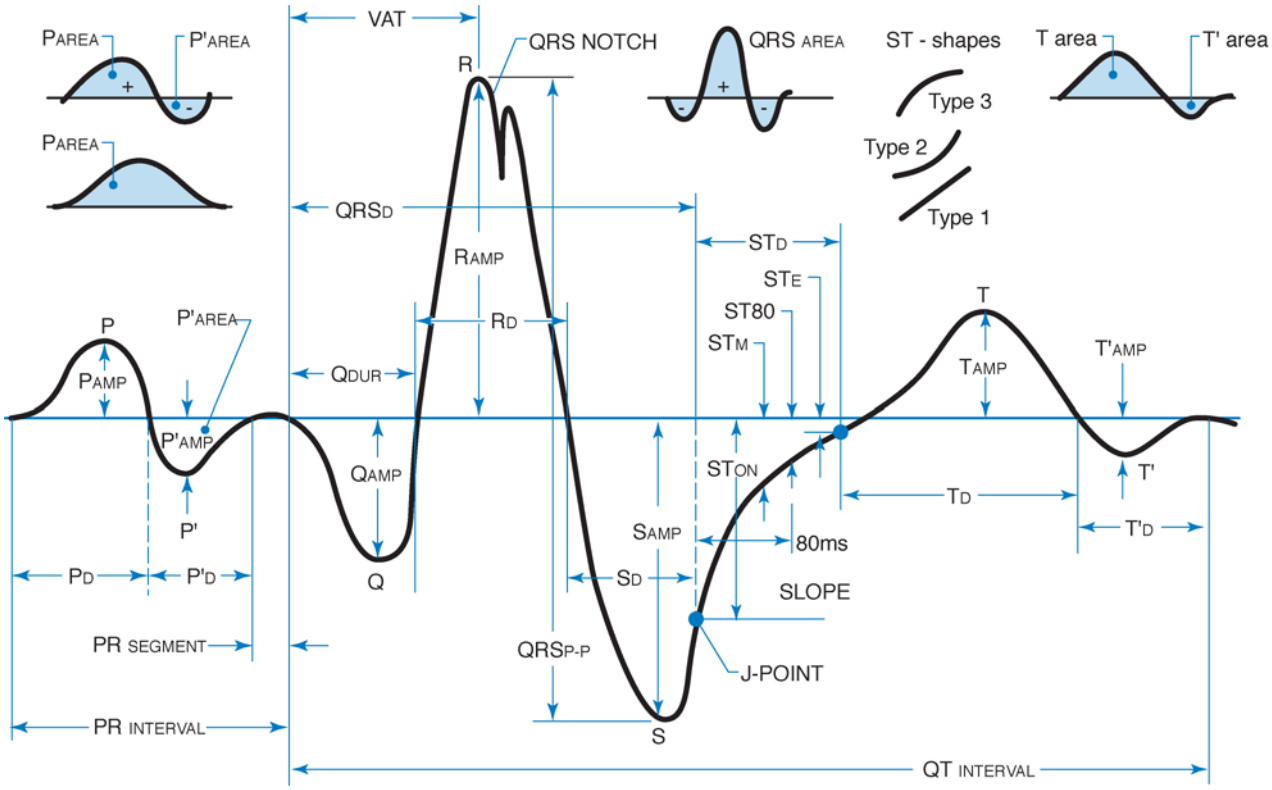
- Only use disposable electrodes that are fresh (not expired, not exposed to open air for an extended period of time).
- Do not mix different brands or types of electrodes.
- Clip hair from the patient's skin if necessary, and do not place electrodes directly on hair. Only place electrodes on the patient's bare skin.
- On adults only, use a dry gauze pad to rub the skin until slight redness appears, to both remove dead skin cells and to increase capillary blood flow.
- If the skin is oily, rub with alcohol, then water-moistened gauze. Alcohol alone dries out the skin and often causes poor electrode contact. Commercial sprays and electrode pads are available that also improve electrode contact and may be used as a substitute for rubbing the skin.
- Wait until the electrode contact stabilizes before initiating the ECG trace. Monitor the signal quality of the trace, ensuring that the acquisition device indicates a good signal quality for all leads.

Waveform Recognition and Measurements

The DXL Algorithm calculates measurements for all the waveforms on an ECG report. Every beat in each lead is measured individually, allowing the natural variation among beats to contribute to the representative measurements. In the algorithm, all of the representative group, lead, and global measurements are

calculated from the comprehensive set of measurements for each beat. The algorithm can use any combination of these three types of measurements (group, lead, global), thereby enhancing the flexibility and power of its interpretive capabilities.

Figure 1-19 ECG Morphology Measurements



Waveform Recognition

The initial step in the analysis process for the DXL Algorithm involves beat detection and waveform recognition.

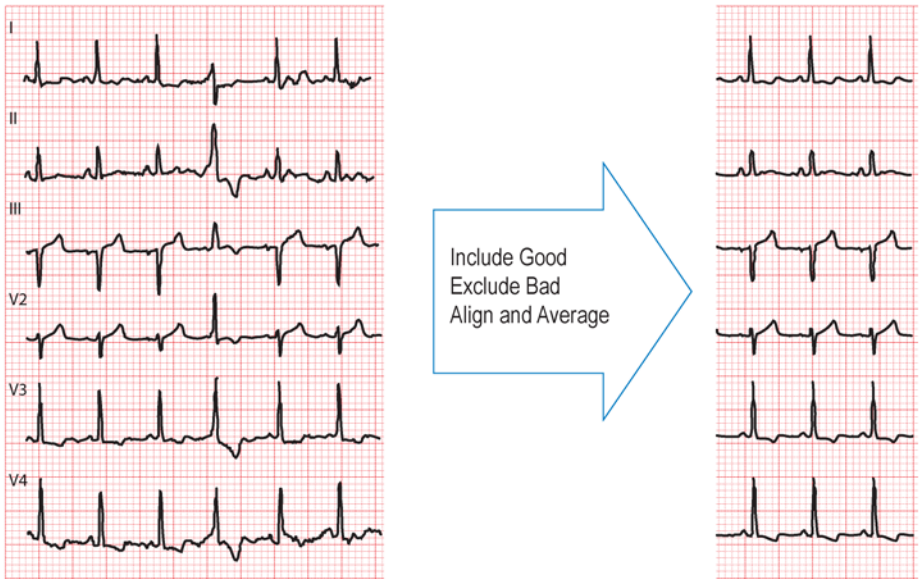
A pacing spike detector is run on all leads if the ECG pacemaker setting is configured for the **On** or **Unknown** setting. If the setting is specified as **Unknown**, spikes are declared if present in four or more leads. If the pacing detection setting is specified as **On**, fewer leads are required for the detection to be considered valid. Pacer spikes are removed and the resulting waves are analyzed with a boundary indicator derived from all leads over the ten-second analysis period.

After the approximate QRS complex and pacemaker spike locations are known, another boundary indicator waveform that enhances P and T wave detection is derived. Approximate P wave, QRS complex, and T wave regions are then determined for each beat in the ECG.

Comprehensive Measurements

After the approximate waveform locations are known, they are further refined to determine precise onsets and offsets for each waveform. Once determined, the amplitude, duration, area, and shape are calculated for every P wave, QRS complex, ST segment, and T wave in each lead. Waveform irregularities, such as notches, slurs, delta waves, and pacemaker spikes, are also noted for every beat.

Figure 1-20 Formation of Representative Beat



NOTE In Figure 1-20 on page 1-22, the fourth beat in series is excluded even though it is a near match in some leads.

Group Measurements

Each beat in the ECG is classified into one of five rhythm groups (described below) based on rate and morphology parameters. Each group has beats with similar R-R intervals, durations, and shapes. All ventricular paced beats are grouped together, regardless of other parameters.

- Group 1 measurements represent the type of beat that is predominant. The beats are matched to exclude outliers, and are then aligned and averaged before the final measurements are taken. This process removes residual noise and improves the precision of the measurements. Averaging after exclusion of outliers makes the most efficient use of the data in the beats. Figure 1-20 on page 1-22 shows how this process works.
- Groups 2 through 5 represent other beat types. Measurements of member beats are then averaged together.

The group into which each beat is classified is noted under the heading **RHYTHM GROUPING OF BEATS** in the Rhythm Analysis section of the Extended Measurements report. For more information, see “Extended Measurements Report” on page 5-26.

Lead Measurements

Measurements for each of the leads are calculated from the Group 1 beats. If *all* beats in the ECG are ventricular paced, then the measurements will be for paced beats. If an ECG contains both paced and non-paced beats, the measurements will be for *only* the non-paced beats.

The lead measurements are averaged representatives of the dominant waveform present in each lead and are reported in the Morphology Analysis section of the Extended Measurements report. For more information, see “Morphology Lead Measurements” on page 6-32.

Atrial Rhythm Analysis

Atrial rhythm is determined by examining leads I, II, III, V1 and V2. Using this method, the algorithm can determine the number of P waves per QRS complex. If the determination fails, no atrial rhythm parameters are calculated.

Global Measurements

The global measurements for the ECG (including the frontal plane axis measurements) are reported to the right of the lead measurements in the Morphology Analysis section of the Extended Measurements report. For more information, see “Extended Measurements Report” on page 6-31.

These interval, duration, and segment measurements are the measurements of the representative beat in each lead from Group 1. The global rate reported is the mean ventricular rate over the entire ECG, unless the algorithm determines that one of the group mean ventricular rates is more representative of the underlying rhythm. This can happen when a short run of premature beats occurs; in this case, they will be excluded before determining the rate.

Axis Measurements

Although it is convenient to use waveform amplitudes when making axis measurements manually, using the areas of the waveforms yields more accurate results. Philips equipment uses the waveform areas from the lead measurements in calculating the P, QRS, and T axes. The sum of the ST onset, and the middle and end amplitudes are used in calculating the ST axis.

The frontal plane axis measurements use the limb leads and nine lead pairs (all at least 60º apart) to estimate the axes. The horizontal plane axis measurements are calculated from leads V1–V6 in a similar manner.

The resulting estimates are examined to ensure that they converge to a single result. They are averaged to form the representative axis measurement.

QT Measurement

QT is measured individually and then combined into a global measurement. Both standard heart rate corrections (Bazett and Fridericia formulae) are applied.

Accuracy of Key Measurements

The measurement accuracy of the DXL Algorithm has been measured on the ECGs specified by the IEC 60601-2-51 standard for safety and performance of analyzing electrocardiographs.

Amplitude measurement accuracy is demonstrated on the set of analytic and calibration ECGs that are designed expressly for that purpose as part of the Conformance Testing initiative[4].

Interval measurement accuracy is demonstrated on a set of biological ECGs that have been annotated by a group of five cardiologists as part of the Common Standards for quantitative Electrocardiography (CSE) effort[5].

Appendix D, "Validation of the DXL ECG Algorithm," demonstrates the difference between the algorithm and the true measurement as a mean and standard deviation of the difference.

Accuracy of QT Measurement and QT Correction

Because of the recent appreciation of the connection between many medications and the development of Torsade de Pointes (polymorphic ventricular tachycardia), there is renewed interest in the accurate measurement of QT interval and various formulas for "correcting" QT interval for the effects of heart rate[6].

For more comprehensive information of the issues surrounding accurate measurements, consult the references listed in Table 1-2 on page 1-25.

4. Laguna P, Thakor NV, Caminal P, Jane R, Yoon HR, Bayes de Luna A, et al. "New algorithm for QT interval analysis in 24-hour Holter ECG: performance and applications." *Medical and Biological Engineering and Computing* 28:67-73, 1990.

5. Algra A, le Brun H, Zeelenberg C. "An algorithm for computer measurement of QT intervals in the 24 hour ECG." *Computers in Cardiology 1986*. Los Alamitos: IEEE Computer Society Press, 117-119 1987. Ahnve S. "Errors in the visual determination of corrected QT (QTc) interval during acute myocardial infarction." *Journal of the American College of Cardiology* 5:699-702, 1985. Savelieva I, Yi G, Guo X, Hnatkova K, Malik M. "Agreement and Reproducibility of Automatic Versus Manual Measurement of QT Interval and QT Dispersion." *American Journal of Cardiology*, 81:471-477, 1998.

6. DM Roden. "Drug-Induced Prolongation of the QT Interval." *New England Journal of Medicine* (2004) 350: 1013-22.

Table 1-2 Additional references related to measurement accuracy

Manual determination of QT interval varies among expert readers: Murray A, McLaughlin NB, Bourke JP, Doig JC, Furniss SS, Campbell RWF. "Errors in manual measurement of QT intervals." *British Heart Journal* 1994;71:386-90. ■ Found differences of 20 milliseconds in normals. Ahnve S. "Errors in the visual determination of corrected QT (QTc) interval during acute myocardial infarction." *Journal of the American College of Cardiology* 1985;5:699-702. ■ Found differences of 28 milliseconds (in myocardial infarction).
Automated methods also vary in the values they measure: McLaughlin, Neil B.; Campbell, Ronald W. F.; Murray, Alan. "Comparison of automatic QT measurement techniques in the normal 12 lead electrocardiogram." *British Heart Journal* July 1995; 74(7):84-89.
Automated methods may be more reproducible: I Savelieva, G Yi, X Guo, K Hnatkova, M Malik. "Agreement and Reproducibility of Automatic Versus Manual Measurement of QT Interval and QT Dispersion." *American Journal of Cardiology* Volume 81(4).February 15, 1998.471-477.
QT interval is different in different ECG leads, which makes "global" QT interval a matter of opinion. JM Glancy, PJ Weston, HK Bhullar et al. "Reproducibility and automatic measurement of QT dispersion." *European Heart Journal* (1996) 17, 1035-1039.
Heart Rate affects QT interval See "Challenges to Identifying the End of the T Wave" on page 1-25.

Challenges to Identifying the End of the T Wave

Finding the end of the T wave presents several technical challenges, including:

- T end is obscured by large U waves
- Biphasic T waves
- Notched T waves
- T waves with superimposed P waves
- Muscle noise
- Power line interference

- Baseline wander
- Very small amplitude T waves
- Very slow (flat) transition of T wave to baseline
- Distortion of the end of the T wave

The DXL Algorithm locates the nadir of the intersection of T and U, as do most manual readers. Biphasic and notched T waves are correctly detected. The effect of a superimposed P wave depends on its exact location. If it overlies the actual (presumed) end of the T wave, it will corrupt the measurement.

Filtering is used as appropriate to reduce certain types of noise. Formation of a representative beat removes muscle noise and allows a more precise determination. Low amplitude T waves are difficult with all techniques. The technique employed by the DXL Algorithm does not appear to be affected by terminal T wave distortion of the sort caused by Sotalol.

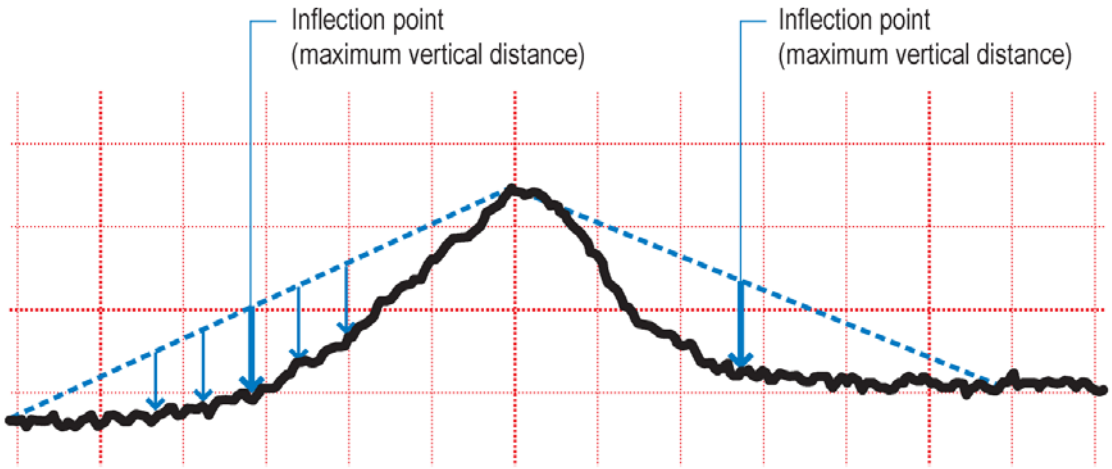
Identifying End of T Wave in the DXL Algorithm

To find the inflection point, whether onset or end, a line is drawn from the peak of the waveform to the start of the search region beyond which the inflection point is expected. The maximum vertical distance from that line to the signal (shown as blue arrows in Figure 1-21 on page 1-27) defines the inflection point.

To prevent errors, the following refinements are used:

- Only the distance between the line and the signal in the expected direction is used.
- The slope of the line is limited to prevent a very steep line, which would result in an inflection point found in a steep part of the waveform. This limitation is important in finding Q onsets if the waveform is a spatial vector magnitude. In practice, the “peak” is moved earlier in time to a point half the R magnitude.
- The slope of the line is kept to a minimum to prevent a nearly flat line, which results in an inflection point located at a maximum noise excursion.
- The maximum and minimum slope of the line depends on the baseline wander over the section of waveform in question; the baseline wander slope is subtracted from the max/min slopes used.

Figure 1-21 Determining End of the T Wave



Global QT Selection

For standard diagnostic use of the 16-lead electrocardiogram, a single value for interval measurements is desirable. Although in the past most of the normal limits were determined using only limb leads, it is common now to also consider precordial lead measurements.

This method is particularly problematic for the QT interval. Some leads may show a QRS onset later than other simultaneous leads, but the difference is usually small and isolated. However, the end of the T wave varies considerably from one lead to another (and tends to be later in the mid-precordial leads), so the question arises as to which lead should be used.

A common approach is to use the earliest Q onset in any lead to the latest T offset in any lead. This method is very subject to noise, however. It is clear from numerous studies that the end of the T wave is a very noisy measurement.

The earliest Q onset to latest T end measurement seems attractive from a theoretical point of view and was first suggested in the article, "The Measurement of the Q-T interval in the Electrocardiogram."[7]

This article also suggested the slope/intercept method, which, as we have seen, underestimates the true end of the T wave. When the article was published in 1952, there was no database to test against and so the recommendations were based on the theoretical understanding of the time.

More than 30 years later, an international cooperative project was established under the name "Common Standards for Quantitative Electrocardiography," now known as CSE[8].

7. E. Lepeshkin, B. Surawicz. "The Measurement of the QT interval in the Electrocardiogram." *Circulation* 1952; 6:378-388.

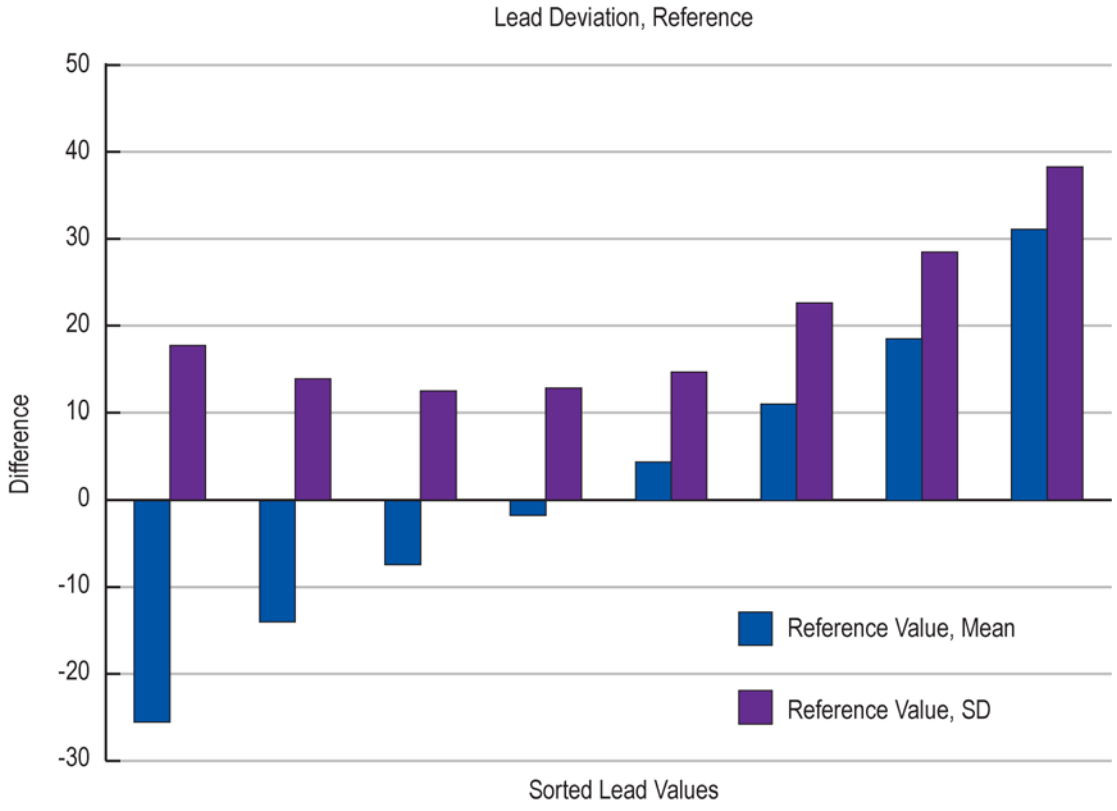
8. JL Willems, P Arnaud, JH van Bemmel, PJ Bourdillon, R Degani, B Denis, FM Harms, PW Macfarlane, G Mazzocca, J Meyer, et al. "Establishment of a reference library for evaluating computer ECG measurement programs." *Computers and Biomedical Research* 1985 Oct; 18(5):439-457.

The usual practice in dealing with noisy measurements is to use a “measure of central tendency,” either a median or a mean. The mean value can be easily distorted by outliers, measurements which are particularly unreliable.

The DXL Algorithm uses the median value in “reliable leads.” A lead is considered reliable if the beat-by-beat onset/offset determinations have a low variance. This helps to eliminate leads with small amplitudes and high respiratory variation, as well as leads with high noise content.

For guidance in determining global measures, consider the data presented in Figure 1-22 on page 1-28. Figure 1-22 shows the difference between QT measurements on each lead, and the five-cardiologist global reference value for QT on the set of biological ECGs. The QT differences are shown as mean difference (algorithm minus reference) and the standard deviation of the difference. For each ECG in the data set, the QT interval differences between the lead by lead QT interval values and the global QT reference are sorted from lowest to highest. The bars representing mean and SD shown are for, in order, the shortest set of QT differences, the second shortest set of QT differences and so on, up to the longest set of QT differences. It is clear that the minimum mean difference and the minimum SD occurs near the median and this is why the DXL Algorithm uses the median T end location in the calculation of QT interval. The data presented in this graph is the result of using the reference measurements, along with comparing the differences in individual lead measurements.

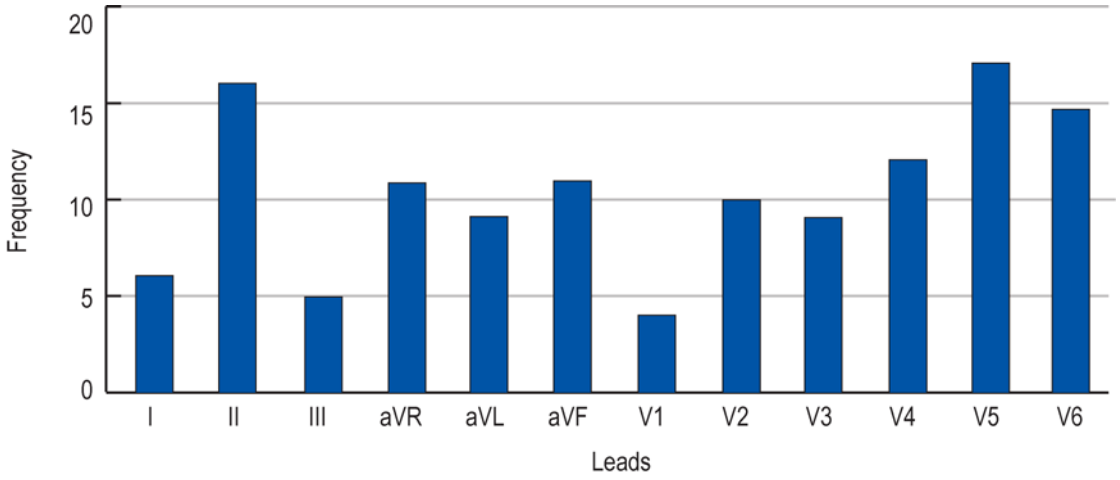
Figure 1-22 Lead Deviation Reference



The range of differences is about 60 milliseconds, which is what would be expected from a mixture of normal and abnormal patients. The standard deviation is minimal near the median, which is what one would expect from a basically noisy measurement.

It should also be mentioned that due to variation between patients, the “best” lead is often not predictable. For the same data set, see the results in Figure 1-23 on page 1-29. Figure 1-23 shows the frequency with which the QT interval measured on each lead is closest to the five-cardiologist QT interval reference. QT interval as measured by the DXL Algorithm on leads II and V5 is most often closest to the reference QT.

Figure 1-23 Lead of Median QT



Looking at superimposed leads, we usually see a clustering of the QT intervals, which most likely accounts for the distribution of the best lead. The traditional leads used in manual reading of drug toxicity studies are leads II and V5, the typical “best” leads.

QT Correction for Heart Rate

As heart rate increases, QT interval shortens. Many attempts have been made to convert values at any given heart rate to the expected values at 60 beats/minute.

Two formulas are commonly used[9] (although many others have been proposed): Bazett and Fridericia.

The Bazett[10] formula is more commonly used in the US, and the formula can be represented by:

$QTc = QT/(RR)^{0.5}$ (square root relation)

9. Molnar et al. “Evaluation of Five QT Correction Formulas Using a Software-Assisted Method of Continuous QT Measurement from 24-Hour Holter Recordings.” *American Journal of Cardiology* (1996); 78:920-26.

10. Bazett, H.C. “An Analysis Of The Time-Relations Of Electrocardiograms.” *Heart* (1920); 7:353-70.

The Fridericia[11] formula is more commonly used in Europe, and can be represented by:

$QTc = QT/(RR)^{0.33}$ (cube root relation)

Neither of these formulae is particularly good at "correcting" when the heart rate is very high or very low. There is considerable variability even when the heart rate is near 60 bpm.

The DXL Algorithm calculates both of these QT rate corrected values. The Philips acquisition device may be configured to output either Bazett or Fridericia, or both.

Generation of Interpretation Statements

Interpretations are divided into several categories, including, for example: hypertrophy, infarcts, ischemia, axis deviation, conduction defects, and others.

Each category is represented on the ECG report by a single statement if any criteria are met in the category. This statement is the last one encountered whose medical criteria were true based on the measurements, earlier decisions, and entered patient demographic information such as age or gender.

In each diagnostic category, more clinically significant findings override more benign ones. For example, in the category of **Ventricular Conduction Delay**, the statement **Left Bundle Branch Block (LBBB)** overrides **Borderline Intraventricular Conduction Delay** and **Incomplete Left Bundle Branch Block**. In addition, the presence of **LBBB** also suppresses a statement from a previous category, such as **Left Axis Deviation**, and bypasses tests for ventricular hypertrophy, most infarcts, ST deviations, and abnormal T waves. These suppression and bypass conditions are not generally addressed in the descriptions of the diagnostic categories that follow, but are clearly important in arriving at an appropriate interpretation.

It is convenient to consider diagnostic categories as rhythm- or morphology-based since rhythm considerations apply to both adult and pediatric patients. Morphologic differences are considerable in the two age groups.

Each diagnostic category includes a set of interpretive statements, with variations in severity and probability. Detailed cardiac rhythm criteria are described Chapter 2, "Adult and Pediatric Rhythm Analysis." Detailed morphology detection criteria are described in Chapter 3, "Adult Morphology Analysis" and Chapter 4, "Pediatric Morphology Analysis."

Overall Severity

Each interpretive statement selected for the ECG report has an associated severity. Severities that are more abnormal override lesser severities. The severities of all interpretive statements in a report are combined to determine

11. L. S. Fridericia. "Die Systolendauer im Elektrokardiogramm bei normalen Menschen und bei Herzkranken." *Acta Medica Scandinavica*, Stockholm, 1920, 57: 469-486.

the overall severity of the ECG. This severity is printed on each page of the ECG report.

Table 1-3 Overall ECG Severity

Severity	Code
No Severity	NS
Normal ECG	NO
Otherwise Normal ECG	ON
Borderline ECG	BO
Abnormal ECG	AB
Defective ECG	DE

About the Use of Extended Leads

The limb leads and chest leads are the traditional components of the standard twelve lead electrocardiogram, but it has long been recognized that additional right sided and posterior leads can provide information that is not well visualized on the standard 12 leads.

This section describes the available right sided and posterior extended leads. These extended leads have been incorporated into the DXL Algorithm when they provide additional information or certainty.

Although the DXL Algorithm can use up to six additional leads from V5R, V4R, V3R, V7, V8, and V9, the acquisition equipment that is used to record ECGs is limited to a total of four additional leads. Any combination of up to four additional leads may be recorded out of the six that are recognized by the algorithm. Some users may choose to add just a single additional lead, while other users may choose to record the maximum of four additional leads.

NOTE **Consult your product documentation for more information on configuring and using extended right side and posterior leads.**

Pediatric Lead Set (V3R, V4R, V7)

Many pediatric institutions add an additional three leads to the standard set. These common pediatric leads are V3R, V4R, and V7.

Use of these leads aids in the differentiation of right ventricular hypertrophy from right bundle conduction defects[12].

12. J Liebman. “What’s old, what’s new – in non-arrhythmia electrocardiography.” *Journal of Electrocardiology* 2004; 37:152-165.

Right-Sided Leads (V3R, V4R, V5R)

Right ventricular infarcts may accompany inferior myocardial infarction and have strong prognostic implications. The signs are subtle in the right-sided leads, and are generally invisible in the standard leads. The right-sided leads used are one or more of V3R, V4R and V5R.[13]

Posterior Leads (V7, V8, V9)

Posterior myocardial infarctions have traditionally been diagnosed from reciprocal changes in leads V1 and V2. Acute posterior infarcts have not yet produced a Q wave, so the tall R is missing in the right chest leads. Posterior ST elevation is expected to manifest as some degree of ST depression in these leads, but may not be sufficiently strong to entirely overcome the ST elevations often seen. Posterior leads are more sensitive and specific in this situation. The algorithm uses one or more of V7–V9.[14]

Myocardial Infarction and Culprit Coronary Arteries

It is possible to correlate the patterns of ST elevation in acute MIs (or of global ST depression) and make a suggestion as to which coronary artery is involved. This may have prognostic significance, as well as save time in treatment and help to disambiguate multiple abnormalities. More detail and examples are provided in the sections describing infarcts in Chapter 3, "Adult Morphology Analysis."

Evaluation of Algorithm Performance

See Appendix D, "Validation of the DXL ECG Algorithm," for a discussion of evaluation approaches and the results of evaluation on the current algorithm program.

13. Antman, Elliott M. et al. "ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction – Executive Summary." *Circulation* 2004;110:e82-e293.

14. RJ Zalenski, RJ Rydman, EP Sloan et al. "Value of Posterior and Right Ventricular Leads in Comparison to the Standard 12-Lead Electrocardiogram in Evaluation of ST-Segment Elevation in Suspected Acute Myocardial Infarction." *American Journal of Cardiology* 1997;79:1579-1585.

2

Adult and Pediatric Rhythm Analysis

ECG analysis begins with rhythm analysis, with the first interpretive statement describing the basic rhythm of the ECG, or the paced rhythm of the ECG.

A second interpretive statement may be appended to describe additional rhythm abnormalities, including premature complexes, pauses, atrioventricular conduction abnormalities, and miscellaneous arrhythmias.

Cardiac Rhythm Categories

Paced Rhythm

Paced rhythm interpretation concentrates on the apparent rhythm, not on the underlying pacemaker mode (which is often not apparent from the observed rhythm). Atrial, ventricular, dual AV sequential, and atrial-sensed ventricular-paced pacing rhythms may be described.

Statements are generated as follows:

- The term **PACED RHYTHM** is used when all beats fit a characteristic paced pattern.
- Paced complexes are described when pacing is intermittent and non-paced complexes are also detected. Such complexes may include ectopic atrial or ventricular premature complexes, or episodes of sinus rhythm. Intermittently paced rhythms are not further analyzed for rhythm patterns during the non-paced periods.
- Demand behavior with pulse inhibition in one or both chambers may be detected.
- Noise spikes in technically poor tracings may mimic pacer spikes. If these are suspected, a statement of pacemaker-like artifact is generated.
- When the ECG record is obtained with a magnet in place, the pacemaker spikes occur at a fixed rate and may be asynchronous with the underlying rhythm. This phenomenon is declared as a failure to sense and/or capture, and the presence of a magnet is questioned.
- An attempt is made to diagnose atrial fibrillation in the presence of ventricular pacing. No other atrial rhythm diagnosis is performed.

- QRS complexes that are not ventricular paced (non-paced or atrial paced complexes) and are not classified as ventricular ectopic beats are measured and used for further morphology interpretation. No further interpretation is considered for ECGs with continuous ventricular or AV dual pacing.

Basic Cardiac Rhythm

When no pacing spikes are found, one interpretive statement describes the basic cardiac rhythm and is based on the interrelationship of the atrial rate, ventricular rate, P wave axis, QRS duration, and other measurements.

Possible statements include those related to:

- Sinus, atrial, supraventricular, junctional, and ventricular rhythms
- Tachycardia, bradycardia, and varying rate
- Complete AV block
- AV dissociation
- Atrial fibrillation
- Atrial flutter

Statements are generated as follows:

- A normal P axis measurement (-30º to 120º in the frontal plane) is assumed to indicate a sinus origin of the P wave. An abnormal P axis signifies an atrial or a junctional origin.
- Tachycardia is generally defined as a rate of 100 bpm or higher in adults; bradycardia is slower than 50 bpm. This differs from the traditional value of 60 cited by many ECG texts. The operator may reset the default criteria from 50 bpm to 60 bpm (if available). Consult your Philips product documentation for more information.
- Pediatric heart rate limits vary considerably with age and are shown in Appendix A, “Normal Measurement Values.”
- An interpretive statement of complete AV block is generated when the ventricular rate is low (< 45 bpm) and the atrial rhythm is asynchronous with the ventricular rhythm. Additional categories of complete AV block include wide QRS complexes and atrial fibrillation.
- AV dissociation is detected by looking for a normal ventricular rate with considerable variation of the apparent PR intervals. While describing the ECG rhythm strip, the algorithm does not define the underlying rhythm (which may be complete heart block or a junctional rhythm). An attempt is made to diagnose the underlying rhythm, complete heart block or junctional rhythm, rather than AV dissociation.
- The detection of atrial fibrillation and atrial flutter is a complex combination of thresholds on atrial signal characteristics and RR interval variation. More

RR interval variation is expected for atrial fibrillation compared to atrial flutter. The atrial flutter waveform is expected to be more stable compared to atrial fibrillation. The atrial signal is isolated from the ECG signal by QRST subtraction and then the signal characteristics are measured from analysis of the repetitive nature of the atrial signal.

Ventricular Preexcitation

Ventricular preexcitation is recognized based on the occurrence of delta waves in multiple leads and a mean QRS duration greater than 100 ms.

Statements are generated as follows:

- A short PR (PR segment <55 ms or PR interval <120 ms) reduces the number of leads with delta waves required to detect this condition.
- Leftward or rightward initial QRS axis deviation criteria are added to determine whether a left or right accessory pathway is present. The rest of the algorithm program is bypassed if ventricular preexcitation criteria are met.

Premature Complexes

Premature complexes are recognized when the preceding R-R interval is shorter than the average R-R interval of a background ventricular rate that is basically regular. A reduction in R-R interval of 15% (typical) or greater is considered significant.

Premature complexes with normal QRS duration (QRSd) are considered to be atrial or junctional in origin, depending on the presence or absence of a P wave. Those with longer than normal QRSd are considered to be either ventricular in origin or to have aberrant conduction from a supraventricular origin.

Statements are generated as follows:

- Atrial premature complexes (APC, multiple APC) are generally recognized by their early appearance, normal QRS duration, and atypical P-wave morphology. More than one APC is diagnosed as multiple APCs. Two APCs in close succession are called a couplet.
- Ventricular premature complexes (VPC, multiple VPC) are generally recognized by an early appearance, wider than normal QRS duration, a compensatory pause, and a different polarity from normal beats.
  - Interpolated VPCs have ventricular morphologic characteristics without compensatory pauses.
  - Multiple VPCs are diagnosed when more than one VPC is detected.
  - Two adjacent VPCs are diagnosed as a pair. The characteristics are primarily morphological since compensatory pauses are not usually seen.

  - A sequence of three or more VPCs in close succession is called **Unsustained Ventricular Tachycardia**.

- Junctional premature contractions (JPC) have the same characteristics as APCs, but without a P-wave being detected. No attempt is made to detect retrograde P waves with JPCs.
- Ventricular or supraventricular bigeminy is declared when ventricular (V) or supraventricular (A) premature beats alternate with normal (N) beats. There must be at least two consecutive occurrences of the pattern (NV or NA) to generate an interpretive statement of bigeminy.
- Ventricular trigeminy is declared when two consecutive occurrences of the pattern NNV are detected.

Pauses

Long R-R intervals are significant if they are more than 140% (typical) of the average R-R in a background ventricular rate that is basically regular. They are considered to indicate either a sinus arrest or an intermittent AV block.

Statements are generated as follows:

- The presence or absence of a P wave, as well as the duration of the QRS, indicate the origin of an escape beat. Atrial and supraventricular escapes show a P wave and a normal QRS duration (QRSd). Junctional escapes show no P wave, but a normal QRSd. A prolonged QRSd indicates a ventricular origin of the escape beat, although aberration cannot be excluded.
- Different grades of second degree AV block are indicated on the basis of a greater number of P waves than QRS complexes.
- A statement indicating Mobitz I (Wenckebach) AV block depends on progressively longer PR intervals preceding the long R-R interval.

Miscellaneous Arrhythmias

This category includes arrhythmias that are not covered in the preceding sections.

Statements are generated as follows:

- Statements relating to interpolated beats depend on recognizing that consecutive R-R intervals are approximately one-half the average R-R of a background ventricular rate that is basically regular.
- Aberrant complexes are recognized when the R-R interval is only slightly decreased but the QRSd is prolonged, as if it were of ventricular origin.

Atrioventricular Conduction

Statements in this category are based on the measurement of a prolonged PR interval.

The PR interval varies slightly according to age and heart rate[1], as shown in the following table.

Table 2-1 Borderline and Abnormally Prolonged PR Intervals (ms)

	Heart Rate (bpm) Left Value = PR Interval Upper Limit (Borderline) Right Value = PR Interval Upper Limit (1st degree AV Block)			
Age (years)	less than 50	51-90	91-120	over 120
16-60	210-220	200-210	195-205	190-200
over 60	200-230	210-220	205-215	200-210

1. For example, see JW Mason, DJ Ramseth, DO Chanter et al. "Electrocardiographic reference ranges derived from 79,743 ambulatory subjects." *Journal of Electrocardiology* 2007;40:224-234.

3

Adult Morphology Analysis

The morphology interpretation starts by testing for dextrocardia. Morphology abnormalities are examined in anatomical order from right to left and from atria to ventricles.

This chapter describes the interpretive criteria (by diagnostic category).

Adult Morphology Categories

Dextrocardia

Dextrocardia is suggested if all of the following conditions are met:

- The P wave and the QRS axes are abnormal in the frontal plane (deviated rightward)
- The horizontal plane QRS is directed rightward
- Small QRS complexes are present in V5 and V6

The remainder of the morphology interpretation is bypassed if dextrocardia criteria are met.

Right Atrial Abnormality

Large P waves are considered suggestive of **Right Atrial Enlargement (RAE)**. The minimum duration considered significant is 60 ms. The minimum voltage considered significant is 0.24 mV (typical).

Greater than normal P wave duration and amplitude in limb leads produces a statement of **Consider Right Atrial Enlargement**.

Additional conditions such as a biphasic P wave in Lead V1 indicate probable RAE.

Larger P waves lead to more definitive interpretive statements regarding the likelihood of RAE.

Left Atrial Abnormality

Left Atrial Enlargement (LAE) is detected from large P waves on limb leads and a biphasic P in Lead V1, along with the durations and the amplitudes of the initial and terminal portions of a biphasic P wave.

A duration greater than 110 ms, combined with amplitudes over 0.10 mV in limb leads is considered significant, though not necessarily abnormal unless present in multiple leads. A notched P wave adds to the significance of the other values.

Lead V1 is specifically examined for duration, amplitude, and area of the negative component of the P wave.

Although duration of over 30 ms and amplitudes over 0.09 mV can be considered significant, the area of this negative component must be greater than 0.60 Ashman[1] units to be considered LAE.

Biatrial Abnormality

Biatrial Enlargement (BAE) combines right and left atrial enlargements.

Associated **Left Atrial Enlargement (LAE)** is diagnosed when a P amplitude greater than 0.1 mV in V1 co-exists with RAE.

Associated RAE is considered when LAE statements are combined with a significant P wave greater than 10 ms in duration and greater than 0.07 mV in amplitude, and an R wave greater than 1.0 mV in Lead V6.

Biatrial Abnormality (BAA) is considered if RAE and LAE statements with high severity were previously generated.

QRS Axis Deviation

Interpretive statements based on frontal QRS axis measurements describe left and right deviation as well as superior, horizontal, and vertical directions. Frontal QRS axis measurements counterclockwise from -30º are considered to be deviated to the left, and those clockwise from 90º are considered to be deviated to the right.

The mean QRS axis (mean vector of the electric force) is calculated in the frontal and horizontal planes. The normal frontal axis range varies with age and gender.

The frontal QRS axis in young male patients tends to the right.

The frontal QRS axis in older patients tends to the left.

A frontal QRS axis between -30º and 90º is considered normal, subject to modification by age and gender.

1. An Ashman unit is the area of 1 square millimeter at normal speed (25 mm/sec) and normal sensitivity (10 mm/mV). An Ashman unit equals 40 ms x 0.1 mV.

Ventricular Conduction Delays

A QRS duration (QRSd) greater than 100 ms is common to all of the interpretive statements in this category except for isolated **Left Anterior Fascicular Block (LAFB)** and **Left Posterior Fascicular Block (LPFB)**, which do not cause a prolonged QRS.

LAFB interpretations are associated with leftward deviation of the mean frontal QRS axis between -40º and 240º counterclockwise.

LPFB interpretations are associated with rightward deviation of the mean frontal QRS axis between 120º and 210º clockwise.

Other than the fascicular blocks, a definitive block interpretation requires that the QRSd exceed 120 ms. A QRSd between 110 and 120 ms is **Non-specific Intraventricular Conduction Delay**, and between 100 and 110ms is considered **Borderline Intraventricular Conduction Delay**.

Right Bundle Branch Block (RBBB) interpretations are always associated with the terminal portion of the QRS being directed to the right (dominant negative Q, S forces in Leads I, aVL, and V6, and positive forces in Lead V1). A QRSd between 110-120 ms is considered incomplete RBBB.

Left Bundle Branch Block (LBBB) interpretations are always associated with the terminal portion of the QRS being directed to the left dominant positive (R, R') forces in Leads I, aVL, and V6, and negative forces (Q, S) in Lead V1. A QRSd between 110-120 ms is considered incomplete LBBB.

Right Ventricular Hypertrophy

Right Ventricular Hypertrophy (RVH) is detected on the basis of several findings:

- Presence of a prominent R or R' in Lead V1
- Presence of a prominent Q, S, or S' in either Lead I or V6
- Right atrial enlargement
- Right axis deviation in the frontal plane
- Repolarization abnormalities typical of RVH

An R in V1 that is more than 75% the size of the Q or S is significant, and is considered to be prominent.

An R' larger than 20 ms and 0.30 mV in V1 is significant.

A QRS in V1 with a positive component larger than the negative component is highly significant.

Repolarization abnormalities typical of RVH are determined by an examination of Leads II, aVF, V1, V2, and V3 for the presence of depressed ST segments and inverted T waves as typical of the right ventricular strain pattern.

A Q, S, or S' larger than 40 ms and 0.20 mV in either Lead I or V6 is significant and is considered to be prominent. A QRS with a negative component larger than the positive component is highly significant.

The statements to be printed regarding RVH are determined by combinations of the above findings.

- One voltage criterion generates a **Consider RVH** statement.
- Two voltage criteria or one voltage plus repolarization abnormality generates a **Probable RVH** statement.
- **Definitive RVH** statements result when multiple findings are present.

Left Ventricular Hypertrophy

Echocardiography has become the standard for the diagnosis of left ventricular hypertrophy. But because many more ECGs than echoes are performed, ECG criteria remain important.

To maintain specificity, the criteria are rather insensitive. Criteria for electrocardiographic interpretation of left ventricular hypertrophy are quite varied due to the large number of criteria proposed, and the clinical spectrum that progresses from mild to severe.

The Philips DXL ECG Algorithm uses a cascade of findings to progress from the weakest to the strongest interpretation. This approach is similar to the Romhilt Estes method, but takes into account more recent criteria developed at Cornell.

This section describes the general process used for adults. Please note that pediatric interpretation requires quite different thresholds, see "Pediatric Morphology Analysis" on page 4-1 for more information.

NOTE **For readers using the TraceMaster ECG Management Systems, two *edit codes* are available for the overreader to use. These two edit codes have no associated criteria:**

- #LVHST NS "LVH WITH SECONDARY REPOLARIZATION CHANGES"
- #HVOLT NS "HIGH QRS VOLTAGE"

Left Ventricular Hypertrophy diagnoses are made on the basis of a point score derived from several findings, in order:

1 High voltage in QRS components (see page 3-5)

2 Left axis deviation in the frontal plane (see page 3-6)

3 Left atrial enlargement (see page 3-6)

4 ST-T changes characteristic of LVH (see page 3-6)

5 A prolonged QRS duration or ventricular activation time (see page 3-6)

Higher point scores result in more severe statements regarding the likelihood of LVH. Each of these steps is described in more detail in the following sections.

Assign point count to voltage

The first step is to assign a point count to the **LVH:Voltage** variable by comparing measurements with the following thresholds as shown in Table 3-1 on page 3-5.

NOTE All values in Table 3-1 are shown in millivolts (mV).

Table 3-1 Assigning Point Count to Voltage

Measurement	Meaning	Male Threshold (mV)	Female Threshold (mV)
RAVL:THRESH	R amplitude aVL	1.2	1.1
RISIII:THRESH	R in I + S in III	2.5	2.5
RV5V6:THRESH	Max R in V5-6	2.6	2.6
SV12RV56:THRESH	Sokolow-Lyon	3.5	3.25
LVHCNV:THRESH	Cornell Voltage	2.8	2.2
LVHCNP:THRESH	Cornell Product	280	300

Most of the measurements are self-explanatory.

LVHCNV is the Cornell Voltage: (RAVL + SV3).

LVHCNP is the Cornell Product: (RaVL+SV3) x QRSd

With these thresholds, we can progress through the following interpretations:

- **Code** is an abbreviation for the criteria assigned at a given stage.
- **Sev** is an abbreviation for the severity produced by assigning that specific code, and the ECG is assigned the highest severity.
- **Statement** is what prints on the final report.
- **Reasons** may appear on unconfirmed reports if the acquisition device is configured to display them. After confirmation on a TraceMaster ECG Management System, they are no longer shown. They generally indicate which evaluation criteria were met.

Table 3-2 Left Ventricular Hypertrophy Interpretations

Code	Sev	Statement	Reason
LVHV	BO	LVH BY VOLTAGE	R > RAVL:THRESH in aVL
LVHR56	BO	LVH BY VOLTAGE	R > RV5V6:THRESH mV in V5 or V6
LVHSR1	BO	LVH BY VOLTAGE	(R I+S III) > RISIII:THRESH mV

Table 3-2 Left Ventricular Hypertrophy Interpretations *(continued)*

Code	Sev	Statement	Reason
LVHSR	AB	CONSIDER LEFT VENTRICULAR HYPERTROPHY	(S V1/V2+R V5/V6) > SV12RV56:THRESH mV
LVHCNV	AB	CONSIDER LEFT VENTRICULAR HYPERTROPHY	(R aVL+S V3) >LVHCNV:THRESH mV

All of these codes are ignored if the patient is less than 35 years old. No further LVH tests are done in the presence of right axis.

- For a single code, **LVHVOLTAGE** gets one point.
- For a combination of two codes it gets two points.

Left Axis Deviation

The next test is for determining left axis deviation, which has thresholds dependent on age. No test is made if **Left Anterior Fascicular Block** has been previously diagnosed.

If positive, the flag **LAD:FOR:LVH** is set.

Further, any LVH diagnosis based on LAD is suppressed by the later diagnosis of an inferior infarct.

Left Atrial Enlargement

The next test is for Left Atrial Enlargement. No test is made if a clinical diagnosis of Mitral Valvular Disease is entered by the ECG technician or if atrial flutter or fibrillation has been previously found by the program.

If positive, the flag **LEFT:ATRIAL:ENLARGEMENT** is set.

ST-T Changes

The next test is for typical Anterolateral ST segment and T-wave changes (LV strain pattern).

If detected, the flag **ST:T: CHANGES:FOR:LVH** is set.

Prolonged QRS Duration Or Ventricular Activation Time

The final test is for widening of mean QRS duration or of Ventricular Activation Time in V5 or V6.

If detected, the flag **QRS:VAT:WIDE** is set.

No test is made if any Bundle Branch Block has been previously found.

With these findings and flags, we can proceed to the next set of interpretations listed in Table 3-3 on page 3-7.

Table 3-3 Left Ventricular Hypertrophy Interpretations

Code	Sev	Statement	Reason
LVHC	AB	CONSIDER LEFT VENTRICULAR HYPERTROPHY	R5/6/aVL, RISIII, S12R56, S3RaVL
LVHVP	AB	PROBABLE LEFT VENTRICULAR HYPERTROPHY	R56L/RISIII/S12R56/S3RL & LAA/LAD
LVHCNP	AB	PROBABLE LEFT VENTRICULAR HYPERTROPHY	(RaVL+SV3)xQRSd > LVHCNP:THRESH
LVHPRE	AB	PROBABLE LVH WITH SECONDARY REPOL ABNRM	R56L/RISIII/S12R56/S3RL & rep abn
LVH	AB	LEFT VENTRICULAR HYPERTROPHY	(SV1+RV5)>3.5/(RaVL+SV3)> LVHCNV:THRESH
LVH1	AB	LEFT VENTRICULAR HYPERTROPHY	R56L/RISIII/S12R56/S3RL & LAA/LAD
LVHREP	AB	LVH WITH SECONDARY REPOLARIZATION ABNORMALITY	R56L/RISIII/S12R56/S3RL & rep abn
LVHCO	AB	LVH WITH IVCD AND SECONDARY REPOL ABNRM	RISIII/S12R56, wQRSd, repol abnrm
LVHCOL		LVH WITH IVCD, LAD AND SECONDARY REPOL ABNRM	RISIII/S12R56, wQRS, LAD, rep abn

If any LVH diagnosis has been made, certain ST segment and T-wave criteria and non-diagnostic leftward axis criteria must be disabled. This happens later in the program.

LVH References

General Reference

Gertsch, M. “The ECG, A Two Step Approach to Diagnosis.” *Springer Berlin* 2004 ISBN 3-540-00869-1.

Cornell Criteria

Casale PN, Devereux RB, et al. “Improved sex-specific criteria of left ventricular hypertrophy for clinical and computer interpretation of electrocardiograms: Validation of autopsy findings.” *Circulation* 1987; 75: 565-572.

Sokolow-Lyon Criteria

Sokolow M, Lyon TP. “The ventricular complex in left ventricular hypertrophy as obtained by unipolar precordial and limb leads.” *American Heart Journal* 1949; 37: 161-186.

Romhilt-Estes Approach

Romhilt DW, Estes EH. “Point-score system for the ECG diagnosis of left ventricular hypertrophy.” *American Heart Journal* 1968; 75: 752.

Romhilt DW, Bove KE, et al. “A critical appraisal of the electrocardiographic criteria for the diagnosis of left ventricular hypertrophy.” *Circulation* 1969; 40: 185-195.

Low Voltage and Chronic Obstructive Pulmonary Disease Pattern

All leads are examined for QRS peak-to-peak voltage.

- Frontal leads — If no lead has a value exceeding 0.60 mV, the ECG is considered borderline low voltage. If no value exceeds 0.50 mV, the ECG is considered definite low voltage, an abnormal finding.
- Precordial leads — If no lead has a value exceeding 1.00 mV, the ECG is considered definite low voltage, an abnormal finding.

Combinations of low voltage statements, rightward deviation of the frontal P and QRS axes, and right atrial enlargement may generate statements suggesting the likelihood of chronic pulmonary disease.

Culprit Coronary Artery Concept

When the electrocardiogram clearly demonstrates signs of an acute infarct (the STEMI criteria are met), additional findings point to the probable anatomical site that is causing the functional ischemia. It is important to note that angiography is the gold standard for anatomical findings, but may be difficult to interpret or may even be misleading when there is multi-vessel disease. The ECG may help to direct therapy appropriately in these cases[2].

2. Kjell Nikus. “Cases Illustrating Use of the ECG for Decision Support after Determination of the Coronary Anatomy.” *International Journal of Bioelectromagnetism* Vol 5; 2003; page 8.

In addition to the traditional suggested localization of the infarct, the probably culprit artery is identified in parentheses.

Table 3-4 Involved Artery with Abbreviation

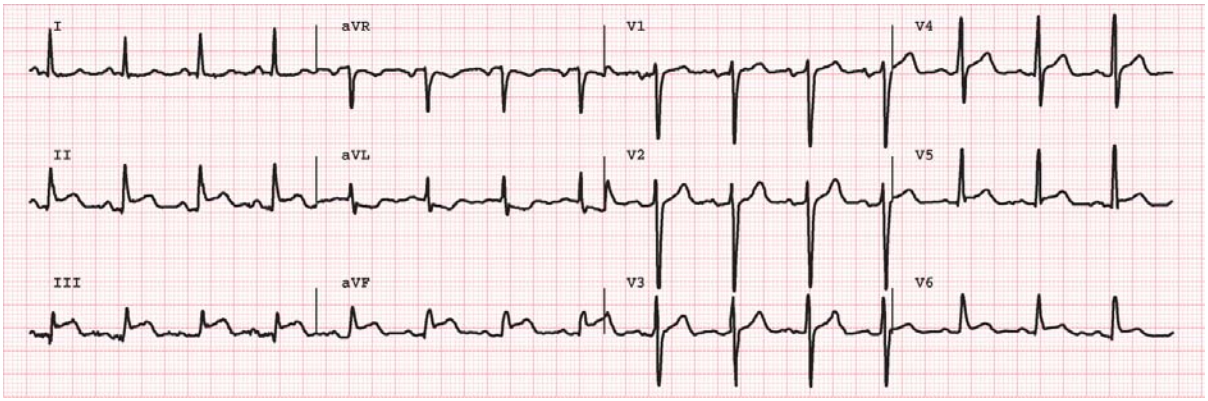
Involved artery	Abbreviation
Left Circumflex	LCx
Right Coronary	RCA
Left Anterior Descending	LAD
High Left Main/multi vessel disease	LM/MVD

NOTE **Strictly speaking, the LM/MVD case is not STEMI, but rather global ST depression, and this distinction should be made due to its extreme importance. In addition, a statement is generated to suggest recording of right-sided leads (V4R) in situations where right ventricular infarct is a possibility, since this has marked clinical significance.**

The following examples illustrate the core concepts.

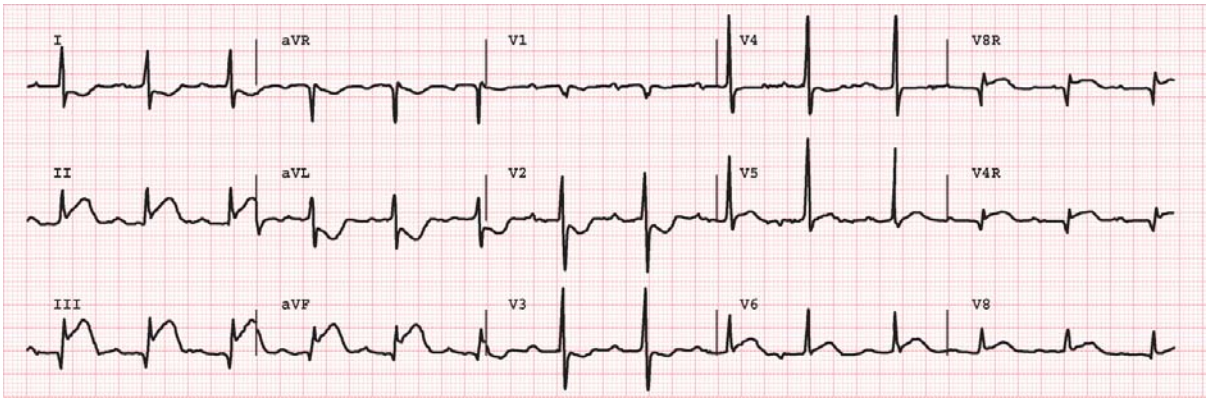
Figure 3-1 on page 3-9 shows an example of a classical inferior myocardial infarction. When these findings are present, the interpretive statement generated is **Acute IMI, suggest recording of right precordial leads**. The suggestion of recording additional leads is made to determine if a concomitant right ventricular infarct is also present, since it has an important effect on morbidity.

Figure 3-1 Inferior Myocardial Infarction, ECG Example



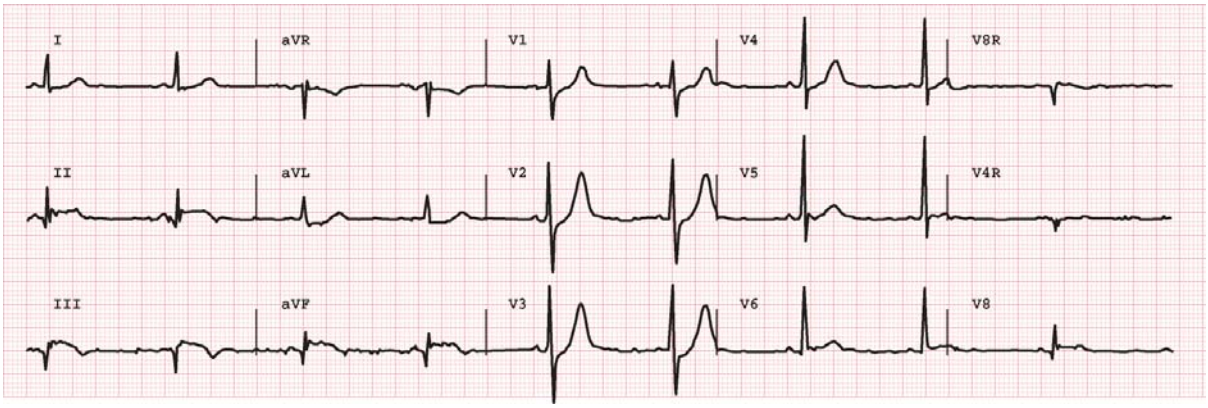
In Figure 3-2 on page 3-10, having the right-sided leads allows us to detect an acute inferoposterior infarct with right ventricular involvement due to a right coronary artery obstruction. Note the ST elevation in lead III is greater than in lead II, which generally indicates right coronary obstruction.

Figure 3-2 Acute Inferoposterior Infarct with Right Ventricular Involvement, ECG Example



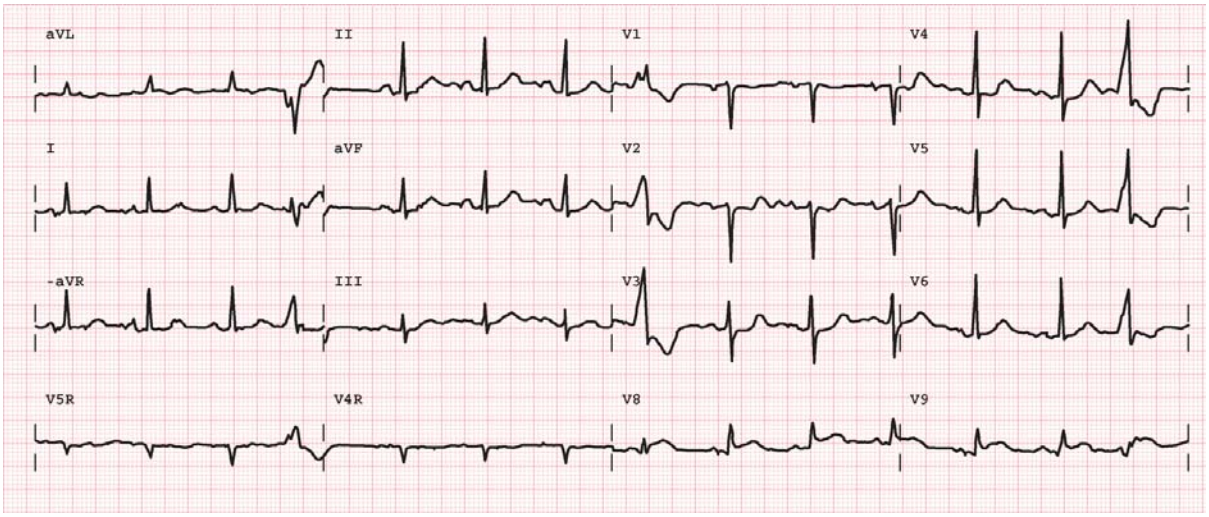
As shown in Figure 3-3 on page 3-10, ST elevation is usually greater in lead II than in lead III when the left circumflex artery is the site of obstruction, as in this case of an inferior myocardial infarct with posterior involvement.

Figure 3-3 Inferior Myocardial Infarction, ECG Example



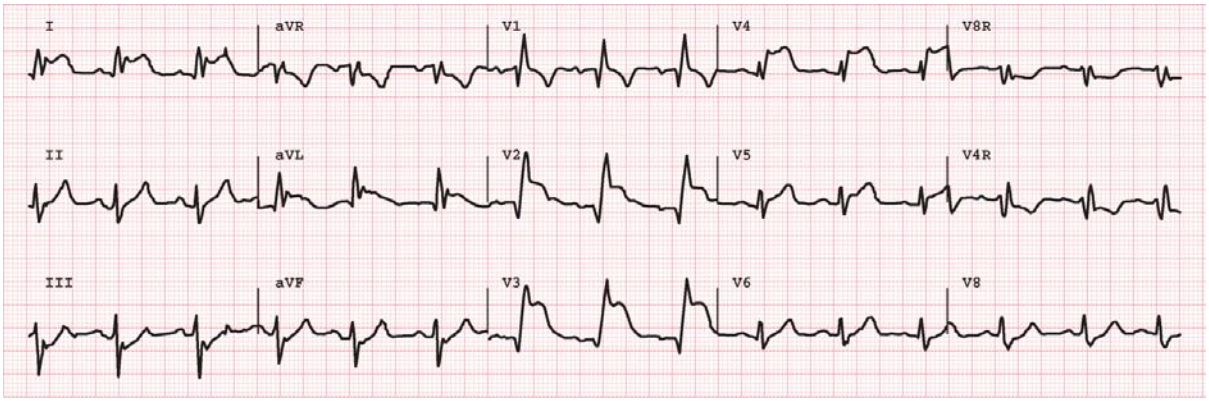
As shown in Figure 3-4 on page 3-11, posterior infarcts usually show ST depression in the right precordial leads, but the abnormality may be subtle and if often considered non-specific or subendocardial. The additional use of posterior leads demonstrates that this is a true posterior infarct, as shown in lead V8.

Figure 3-4 Acute Posterior Infarct, ECG Example



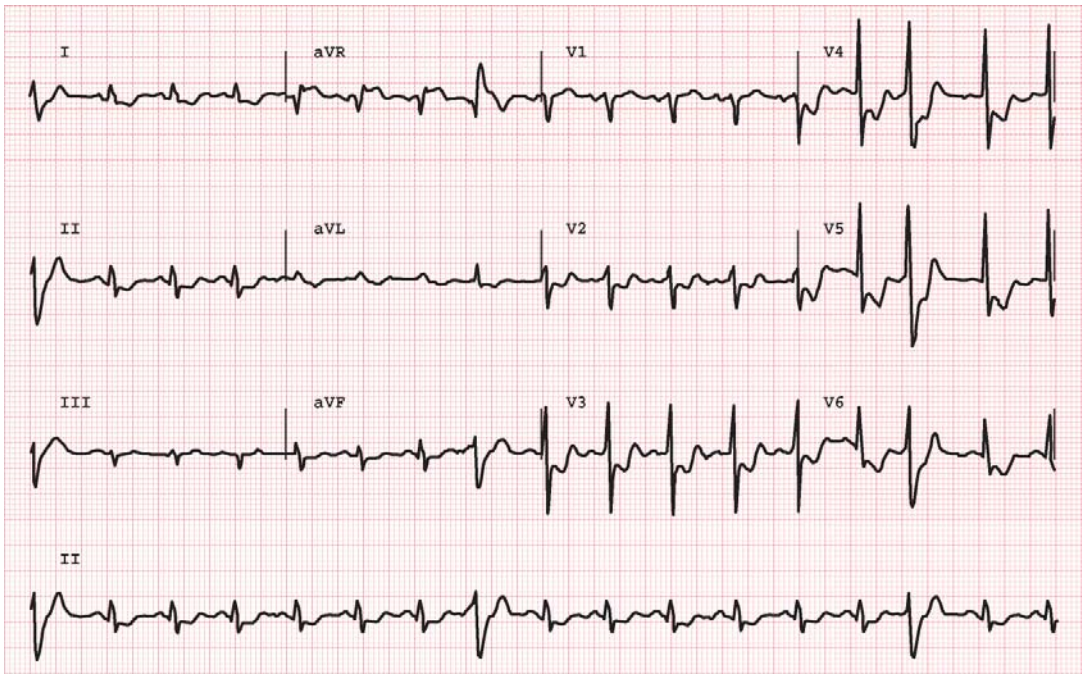
As shown in Figure 3-5 on page 3-11, classical ST segment elevation in anterior or anterolateral leads to an interpretation of acute anterolateral infarct due to LAD obstruction (which was in the proximal portion of the LAD in this patient).

Figure 3-5 Acute Anterolateral Infarct, ECG Example



As shown in Figure 3-6 on page 3-12, when there is widespread ST depression (six or more leads) accompanied by ST elevation in aVR, and sometimes in lead V1, the problem is most likely main left coronary artery obstruction, or severe multi-vessel disease.

Figure 3-6 ST Depression Accompanied by ST Elevation, ECG Example



Inferior Myocardial Infarction

Leads II, III, and aVF are examined for:

- Q wave presence and size
- Ratio of Q to R
- Presence of T wave changes (flattened or inverted)
- Presence of an elevated or depressed ST segment

As the Q waves become larger or appear in more leads, and the R waves become less prominent, the interpretive statements are more significant.

For inferior Q waves to be considered significant, at least one of them must be longer than 25 ms in duration and greater than one-sixth the amplitude of the associated R.

For any infarct statement to qualify, at least one Q wave must be longer than 35 ms and greater than one-fifth the amplitude of the R wave.

A leftward direction of the axis of the initial portion of the QRS adds to the likelihood of an inferior infarct statement.

T wave and ST changes are used to estimate the age of the infarct. Deeper T wave inversion and larger ST segment deviations generate statements indicating more recent infarction.

Gender and age influence the detection of inferior infarct. Males and younger patients are more likely to have normal Q waves in the inferior leads.

Following the 2004 ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction (6.2.3. Electrocardiogram):

> *"In patients with inferior STEMI, right-sided ECG leads should be obtained to screen for ST elevation suggestive of RV infarction."*

When inferior ST elevation criteria are met, right ventricular involvement is suggested, and recording of right chest leads is recommended.

The culprit artery will be suggested as an RCA origin when right-sided leads show ST elevation, or ST elevation is greater in lead III than in lead II.

Lead II STE > Lead III STE and V3 ST < V2 ST suggests a left circumflex origin.

Note that these statements are not made in the presence of several confounding conditions.

Lateral Myocardial Infarction

Leads I, aVL, V5, and V6 are examined for:

- Q wave presence and size
- Ratio of Q to R
- Presence of T wave changes (flattened or inverted)
- Presence of an elevated or depressed ST segment

For lateral Q waves to be considered significant, at least one must be longer than 35 ms and greater than 0.10 mV in amplitude. It must also have amplitude that is at least 20% as large as that of the R wave. As the Q waves become larger or show in more leads and the R waves become less prominent, the interpretive statements become more significant.

T wave and ST changes are used to estimate the age of the infarct. Deeper T wave inversion and larger ST segment deviations generate statements indicating more recent infarction.

Gender and age influence the detection of lateral infarct. Males and younger patients are more likely to have normal Q waves in the lateral leads.

A left anterior descending culprit artery is indicated when there is clear cut STEMI in the lateral leads.

Anteroseptal and Anterior Myocardial Infarction

Leads V1, V2, V3, and V4 are examined for:

- Presence of Q wave
- Q wave area
- Relative and absolute sizes of the R and S waves
- Whether the QRS area is negative or positive
- Presence of T wave changes (flattened or inverted)
- Presence of elevated or depressed ST segments.

Positive findings in V1 and V2 tend to be reported as anteroseptal infarcts, while abnormalities in V2, V3, and V4 tend to be reported as anterior infarcts.

For any anteroseptal or anterior Q wave to be considered significant, it must be longer than 30 ms in duration and over 0.07 mV in amplitude. As the Q waves become larger or show in more leads, and the QRS progression from negative to positive becomes shifted more laterally, the interpretive statements become more definitive for infarction in the anterior region.

Significant STEMI findings in anteroseptal or anterior leads produce an indication of left anterior descending culprit artery.

T wave and ST changes are used to estimate the age of the infarct. Deeper T wave inversion and greater ST elevations generate statements indicating more recent infarction.

Anterolateral and Extensive Anterior Myocardial Infarct

Leads V2, V3, V4, V5, and V6 are examined for:

- Q wave presence and size
- Relative and absolute sizes of the R and S
- Whether the QRS area in V3 is negative or positive
- Presence of T wave changes (flattened or inverted)
- Presence of elevated or depressed ST segments

For any anterolateral Q wave to be considered significant, it must be longer than 30 ms (typical) in duration and over 0.07 mV in amplitude. As the Q waves become larger or show in more leads, the interpretive statements become more definitive for infarction.

Positive findings in all six precordial leads generate statements describing extensive anterior infarction.

Significant STEMI findings in anterolateral or extensive anterior leads produce an indication of left anterior descending culprit artery.

Gender and age influence the detection of anterolateral infarct. Males and younger patients are more likely to have normal Q waves in the anterolateral leads.

Q, ST changes, and T wave are used to estimate the age of the infarct. Deeper T wave inversion and greater ST elevations generate statements indicating more recent infarction.

Posterior Myocardial Infarction

Leads V1, V2, and V3 are examined for:

- Relative and absolute sizes of the R and S waves
- Absent or insignificant Q wave
- ST depression
- Positive T wave

If the leads are available, V7, V8, and V9 are also measured for these characteristics and ST elevation is used to increase the sensitivity of the detection.

A prominent R, in the presence of an insignificant Q and an upright T may generate a statement suggesting the likelihood of a posterior infarct (PMI).

ST depression in V1-V3, and upward T or T' are detected for acute posterior infarct.

Combined inferior and posterior MI is called inferoposterior MI, and combined acute inferior MI and acute posterior MI is called acute inferoposterior MI.

Although most posterior infarcts are due to LCx blockage, the presence of ST elevation in right precordial leads indicates an RCA origin.

Indications of LVH or RVH decrease the likelihood of a PMI statement.

Gender and age influence the detection of a posterior infarct. Males and younger patients are more likely to have prominent R waves in V1 and V2.

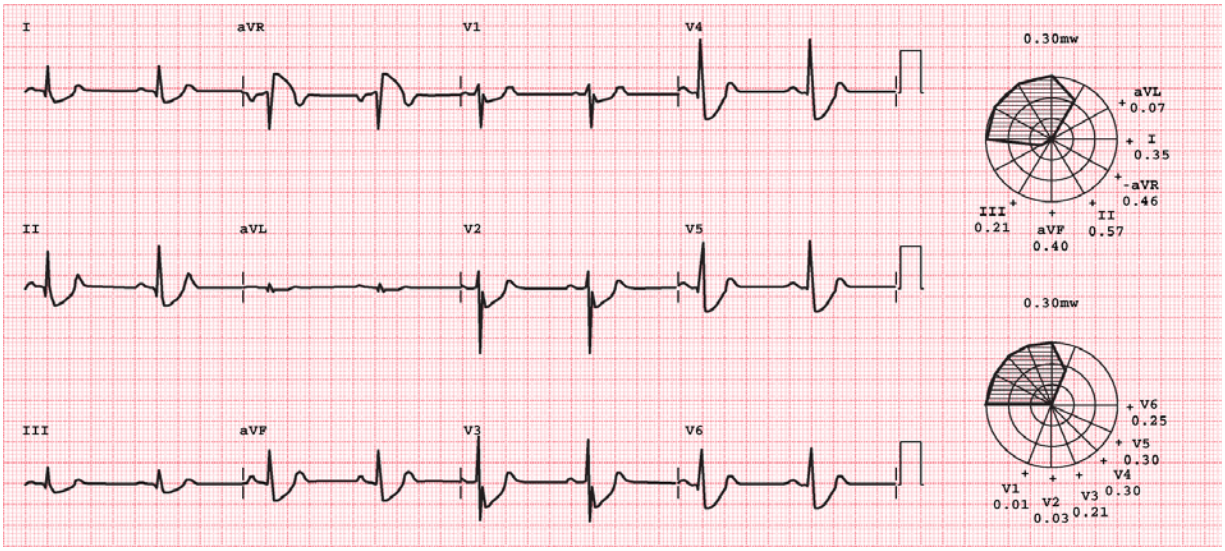
ST Abnormalities and ST Maps

ST elevation and depression are often of major clinical importance, and the anatomical distribution may give further information about the pathology behind the abnormality. Certain Philips acquisition devices provide the capability of displaying ST Maps. This section provides two examples of ST Map reports. For more information on the specific capabilities of your device to generate ST Maps, please consult the product documentation.

In Figure 3-7 on page 3-16, notice that the frontal leads are shown with widespread ST depression, and the precordial leads also show widespread ST depression that is extensive in leads V2-V6, a typical pattern of multivessel or left mainstream occlusion. An ST Map will not display a cross-hatched shaded

area if there are no abnormalities. With elevation, the hatched shaded area will appear prominently.

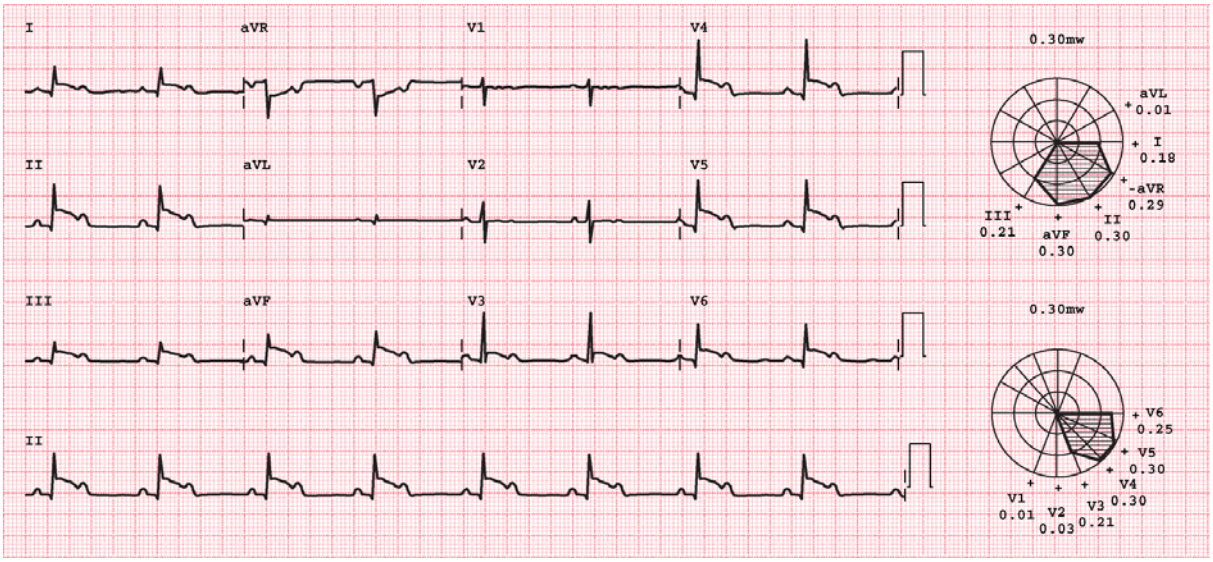
Figure 3-7 3x4 ST Map Report



In Figure 3-8 on page 3-16, notice that ST elevation is almost always directed down and to the right. The one exception is aVR, for which these locations signify a negative value. The ST Map is presented in Cabrera format, which is preferred when examining from a vector viewpoint.

This acute infarct displays abnormalities in the inferior leads with anterolateral involvement. Note that the ST Map has concentric circles for the degree of deviation that progresses from 1 mm to >= 3 mm.

Figure 3-8 3x4, 1R ST Map Report



ST Depression and Myocardial Ischemia

All leads are examined for negative values in the ST segment. The values examined include the following points in the ST segment:

- The onset of the ST segment (the J point)
- The point midway between the onset and the end of the ST segment
- 80 ms past the J point
- The end of the ST segment (the beginning of the T wave)

Besides negative values in the ST segment, other features are examined:

- The slope of the ST segment in degrees
- The shape of the ST segment (straight, concave up, or concave down). The smallest negative ST deflection that is considered significant is 0.03 mV.

As the negativity of the ST segment increases, more severe statements are generated. Minor depression of the segment produces statements with a severity code of **OTHERWISE NORMAL (ON)** or **NORMAL (NO)**. Increasing depression produces statements progressing through from **BORDERLINE** to **ABNORMAL**.

Whenever possible, the location of ST abnormalities is indicated as part of the interpretive statement. The localization generally fits the description listed in Table 3-5 on page 3-17.

Table 3-5 Location of Infarcts and Lead Group of ST-T Abnormalities

Lead Groups (Location)	I	II	III	aVR	aVL	aVF	V1	V2	V3	V4	V5	V6
Anterior							X	X	X	X		
Anterolateral	X			X	X			X	X	X	X	X
Lateral	X				X						X	X
Inferior		X	X			X						

ST depression is associated with rapid heart rate. A statement is generated indicating ST depression, probably rate related, if the mean heart rate is greater than 190 bpm minus the patient age in years.

A concurrent statement regarding RVH, LVH, LBBB, RBBB, any new infarct, or any statement associated with drug therapy or electrolyte imbalance impacts this category by tending to suppress ST depression statements. This is more likely for the less severe ST depression statements than for the more severe ones.

T Wave Abnormalities and Myocardial Ischemia

All leads are examined for:

- T wave amplitude
- Relative amplitude of the T and the QRS
- Whether the T is negative or positive

The frontal axis of the T wave and its relation to the frontal QRS axis is also measured.

Reduced T wave amplitude (both absolute and relative to the QRS), and negative T waves are considered to be abnormal findings. Minimal changes in one or a few leads produce less severe statements. As the changes become more prominent in magnitude, and the number of affected leads increases, the statements become more severe.

A frontal T axis that is not between -10º and 100º, or a QRS-T angle that is greater than 90º, may result in a statement indicating nonspecific T wave abnormalities. Whenever possible, the lead group of T wave abnormalities is indicated as part of the interpretive statement.

A concurrent statement regarding RVH, LVH, LBBB, RBBB, any infarct, or any statement associated with drug therapy or electrolyte imbalance impacts this category by tending to suppress T wave statements. This is more likely for the less severe T wave statements than for the more severe ones.

Repolarization Abnormalities and Myocardial Ischemia

This category includes statements indicating the presence of both ST segment and T wave abnormalities. None of these statements involves any new examination of measurements.

All statements in this category are determined by the combination of statements in the **T Wave Abnormalities** and **ST Depression** categories. The severity of the statements in this category depends on the severity of the qualifying ST and T wave abnormalities.

ST Elevation, Myocardial Injury, Pericarditis, and Early Repolarization

ST segment elevation is based on examination of all lead groups for positive values of the ST onset (J point), the deflection at 80 msec after onset, and the slope of the ST segment (in degrees).

The smallest positive ST displacement considered significant is 0.05 mV (0.5 mm). When ST elevation is small (0.05 mV to approximately 0.10 mV, that is, less than 1 mm), the statements are considered to be of **OTHERWISE NORMAL (ON)** or **BORDERLINE (BO)** severity. ST elevation greater than 1 mm is generally classified as **ABNORMAL (AB)**.

A specific lead group always follows a statement of borderline or abnormal ST elevation. Abnormal ST elevation in a specific lead group is described as **Consider**, **Probable**, or **Definite** myocardial injury. If ST elevation is widespread on all anterior, lateral, and inferior lead groups, either pericarditis or probable early repolarization is suggested.

Tall T Waves

All leads are examined for the presence of positive T waves with amplitudes that exceed 1.20 mV, *or* for positive T waves that exceed 0.50 mV *and* are also more than half the size of the peak-to-peak QRS voltage.

The presence of such T waves generates statements alerting to the possibility of metabolic, electrolyte, or ischemic abnormalities.

QT Abnormalities, Electrolyte Disturbance, and Drug Effects

Measurements of QT interval, as corrected for heart rate, and measurements associated with ST segment depression and T wave changes are examined for values characteristic of the effects of digitalis, and abnormal calcium and potassium levels.

A QT interval corrected for heart rate (QTc) that is shorter than 340 ms is considered to be a short QT interval with a severity code as **OTHERWISE NORMAL (ON)**.

QTc greater than 465 ms is considered as borderline prolonged QTc. An additional 20 ms qualifies the condition as prolonged QTc. Presence of RVH, LVH, and VCD suppresses statements of a prolonged QTc.

If the QTc is shorter than 310 ms, a statement of short QTc suggesting hypercalcemia is generated.

A significantly prolonged QTc interval greater than 520 ms is considered to be due to hypocalcemia.

A significantly prolonged QTc interval (> 520 ms), combined with ST segment depression and a positive T wave in multiple leads, is considered to be due to hypokalemia.

The presence of an Rx code indicating use of digitalis favors interpretive statements that the findings are compatible with the effects of this drug. A combination of a short QTc and repolarization abnormality is considered to be due to digitalis effect.

4

Pediatric Morphology Analysis

The pediatric Philips DXL 16-Lead ECG Algorithm is intended for use on ECGs of patients from birth up to 16 years of age. Age is an important factor in the pediatric algorithm since normal limits in heart rate, axis deviation, and waveform amplitudes are highly age dependent. Specification of age is highly recommended to improve overall ECG interpretation quality. If an age is not entered or is invalid, the interpretation is based on a default adult age, and a special statement noting this assumption is printed on the ECG report.

Specific age limits of ECG features are adopted in the pediatric algorithm[1].

This chapter describes the interpretive criteria (by diagnostic category).

Pediatric Morphology Categories

Dextrocardia

Dextrocardia is suggested if:

- The frontal P axis is between 90º and 180º
- Lead I or V6 has a negative P wave
- Leads I and V6 have a large S wave (> 0.6 mV)
- The P wave amplitude in Lead III is greater than in Lead II

The remainder of the algorithm is bypassed if dextrocardia criteria are met.

Right Atrial Abnormality

Large P waves are considered suggestive of **Right Atrial Enlargement (RAE)**. The minimum duration considered significant is 60 ms, the minimum voltage considered significant is 0.20 mV (typical).

1. Davignon A, Rautuharju P, Boiselle E, et al. “Normal ECG Standards for Infants and Children.” *Pediatric Cardiology* 1:123-131 (1979/80). For more information, see Appendix A, “Normal Measurement Values.”

Greater than normal P wave duration and amplitude in limb leads produce a statement of **Consider Right Atrial Enlargement**.

Additional conditions, such as a biphasic P wave in Lead V1, indicate **Probable RAE**.

Larger P waves lead to more definitive interpretive statements regarding the likelihood of RAE.

Left Atrial Abnormality

Left Atrial Enlargement (LAE) is detected from:

- Large P waves on limb leads
- Niphasic P in Lead V1
- Durations and the amplitudes of the initial and terminal portions of a biphasic P wave

A duration greater than 110 ms combined with amplitudes over 0.10 mV in limb leads is considered significant, though not necessarily abnormal unless present in multiple leads. A notched P wave adds to the significance of the other values.

Lead V1 is specifically examined for duration, amplitude, and area of the negative component of the P wave. Although duration of over 30 ms and amplitudes over 0.09 mV can be considered significant, the area of this negative component must be greater than 0.60 Ashman[2] units to be considered LAE.

Biatrial Abnormality

Biatrial Enlargement (BAE) combines right and left atrial abnormalities.

Associated LAE is considered when a P amplitude greater than 0.1 mV in V1 co-exists with RAE.

Associated RAE is considered when LAE statements are combined with a high amplitude P wave.

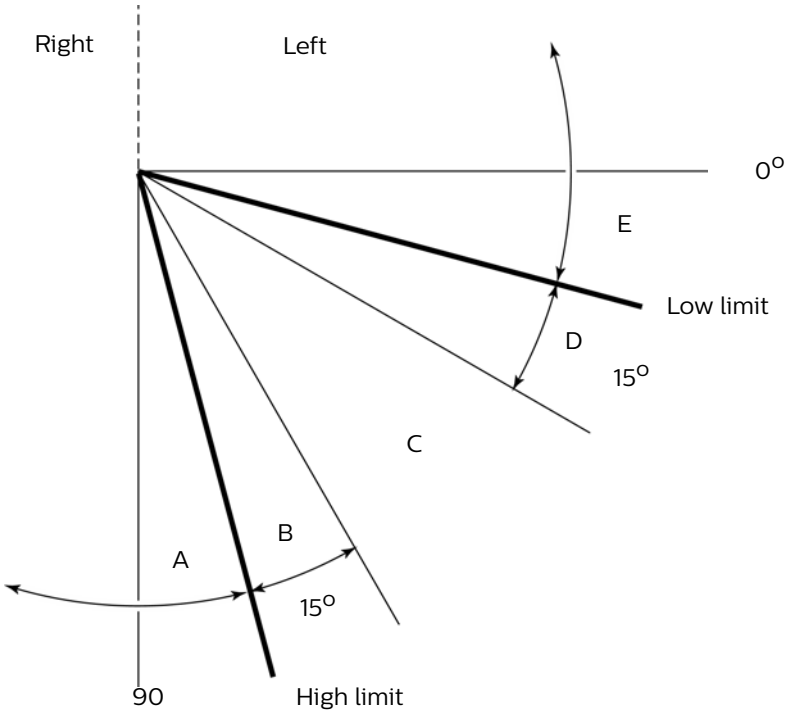
If RAE and LAE statements with high severity are generated from previous RAE and LAE categories, a statement of Biatrial Hypertrophy is generated.

QRS Axis Deviation

The frontal plane axis is examined for left axis deviation and right axis deviation. The normal limits of QRS axis are adjusted for age.

2. An Ashman unit is the area of 1 square millimeter at normal speed (25 mm/sec) and normal sensitivity (10 mm/mV). An Ashman unit equals 40 ms x 0.1 mV.

Figure 4-1 Limits for QRS Axis



A Right axis deviation

B Borderine right axis deviation

C Normal

D Borderline left axis deviation

E Left axis deviation

Figure 4-1 on page 4-3 illustrates the conditions for generating QRS axis deviation statements.

- **Left Axis Deviation (LAD)** statement is generated if the QRS axis is less than the low limit of normal. A borderline LAD statement is generated if the QRS axis in the frontal plane is within 15° of the low limit of normal.
- **Right Axis Deviation (RAD)** statement is generated if the QRS axis is greater than the high limit of normal. A borderline RAD deviation statement is generated if the QRS axis in the frontal plane is within 15° of the high limit of normal.
- Specific limits are listed in Table 4-1 on page 4-3 through Table 4-4 on page 4-5.

Table 4-1 Left Axis Deviation

Age	High Limit (°)	Low Limit (°)
0-23 hours	-90	54
1-3 days	-90	54

Table 4-1 Left Axis Deviation *(continued)*

Age	High Limit (°)	Low Limit (°)
4-6 days	-90	54
7-29 days	-90	54
1-2 months	-90	20
3-5 months	-90	-6
6-11 months	-90	-6
1-2 years	-90	-6
3-4 years	-90	-10
5-7 years	-90	-10
8-11 years	-90	-10
12-15 years	-90	-15

Table 4-2 Borderline Left Axis Deviation

Age	High Limit (°)	Low Limit (°)
0-23 hours	55	65
1-3 days	55	65
4-6 days	55	65
7-29 days	55	65
1-2 months	21	30
3-5 months	-5	1
6-11 months	-5	1
1-2 years	-5	1
3-4 years	-9	1
5-7 years	-9	1
8-11 years	-9	1
12-15 years	-14	1

Table 4-3 Right Axis Deviation

Age	High Limit (°)	Low Limit (°)
0-23 hours	216	269

Table 4-3 Right Axis Deviation *(continued)*

Age	High Limit (°)	Low Limit (°)
1-3 days	216	269
4-6 days	216	269
7-29 days	216	269
1-2 months	131	269
3-5 months	131	269
6-11 months	131	269
1-2 years	131	269
3-4 years	146	269
5-7 years	201	269
8-11 years	151	269
12-15 years	161	269

Table 4-4 Borderline Right Axis Deviation

Age	High Limit (°)	Low Limit (°)
0-23 hours	205	215
1-3 days	205	215
4-6 days	205	215
7-29 days	200	215
1-2 months	115	130
3-5 months	115	130
6-11 months	115	130
1-2 years	115	130
3-4 years	126	145
5-7 years	160	200
8-11 years	135	150
12-15 years	145	160

Ventricular Conduction Delays

The mean QRS duration normal limits are age dependent, and are listed in Table 4-5 on page 4-6.

A mean QRS duration that exceeds 110% of the normal limit is considered **Borderline Intraventricular Conduction Delay (IVCD)**.

A mean QRS duration that exceeds 120% of the normal limit generates a statement of **Nonspecific Intraventricular Conduction Delay (IVCD)**.

Table 4-5 Mean QRS Duration Normal Limits

Age	Normal Limit (ms)
12-15 years	100
8-11 years	88
5-7 years	88
3-4 years	88
1-2 years	78
6-11 months	84
3-5 months	84
1-2 months	84
7-29 days	70
4-6 days	70
1-3 days	70
0-23 hours	70

The presence of a ventricular conduction delay for age, and either an RSR' or no negative component at all (no Q or S) in V1 generates a **Right Bundle Branch Block (RBBB)** statement. For the RSR' to be significant, the R' must be at least 20 ms in duration and 0.15 mV in amplitude.

Incomplete Right Bundle Branch Block (IRBBB) requires a QRS complex similar to RBBB, RSR' or pure R, but with a narrower mean QRS duration, which is less than 120% of normal limit. In addition, synthesized vector measurements in the horizontal plane are applied to distinguish IRBBB from right ventricular hypertrophy.

A statement indicating **Left Bundle Branch Block (LBBB)** is generated in the presence of:

- Prolonged QRS duration for age
- A QRS axis for the terminal 40 ms between -90º and 90º (clockwise)

- A short (< 20 ms) or absent S in I, aVL, V5, V6, and a small or absent R wave in V1, V2, V3

In the absence of a statement regarding LBBB, a mean QRS axis between -60º and -90º generates a **Left Anterior Superior Fascicular Block (LAFB)** statement.

Right Ventricular Hypertrophy

This category is bypassed in the presence of RBBB. The detection of RVH is based on findings in RVH voltage, upright T, and **Right Axis Deviation (RAD)**.

Right Ventricular Hypertrophy (RVH) voltage is heavily age dependent. Six different age groups are established with appropriate voltage criteria for each group. A total of 24 different conditions form the criteria for significant RVH voltage in the varying age groups. Factors considered include:

- The absolute size of R and R' in V1 and V2
- The absolute size of S in V6
- The relative sizes of R and S in V1 and V6
- The presence of a QR pattern in V1

A statement indicating **Consider RVH** or **Probable RVH** is generated if the required voltage exceeds 98% of the normal distribution, as listed in Appendix A, "Normal Measurement Values."

Upward T wave criteria apply to newborns older than 48 hours and to children less than 9 years old. To qualify for RVH, an upward T in V1 without inverted T in V5 and V6 is required. RAD and borderline RAD also support the determination of RVH. The terminal angle of the horizontal plane synthesized vector measurement using an ECG also supports identifying mild RVH versus incomplete RBBB.[3]

Combinations of statements relating to these conditions generate statements varying in severity from **BORDERLINE (BO)** to **ABNORMAL (AB)**. The likelihood of RVH increases as the severity of the qualifying statements increases.

Left Septal Hypertrophy

A statement of **Left Septal Hypertrophy (LSH)** is generated if prominent R waves in V1 and Q waves in V5 and V6 are detected (R wave amplitude > 98% of the R wave amplitude for normal distribution).

LSH is considered if moderate R waves in V1 and Q waves in V5 and V6 are detected.

3. Zhou SH, Liebman J, Dubin AM, Gillette PC, et al. "Using 12-Lead ECG and Synthesized VCG in Detection of Right Ventricular Hypertrophy with Terminal Right Conduction Delay versus Partial Right Bundle Branch Block in the Pediatric Population." *Journal of Electrocardiography* 34 (supp):249-257 (2001).

Left Ventricular Hypertrophy

This category is bypassed in the presence of RBBB or LBBB.

The determination of **Left Ventricular Hypertrophy (LVH)** is based on the presence of qualifying statements in the LVH voltage criteria, **Left Axis Deviation (LAD)**, and an abnormal repolarization pattern typical for LVH. Various combinations of statements from these abnormalities produce statements of varying severity and certainty regarding the presence of LVH.

LVH voltage criteria applied in LVH classification are:

- R amplitude in I, II, aVL, aVF, V5 or V6
- S amplitude in V1 or V2
- R amplitude in V6 plus S amplitude in V1
- Prominent Q wave in V5, V6 or II, III, aVF

The LVH voltage criteria are age dependent. A measured value in voltage is considered abnormal only if it exceeds 98% limits in the normal distribution.[4]

A left atrial enlargement reflected by P wave and left axis deviation supports determination of LVH. Leads I, aVL, V4, V5, and V6 are examined for repolarization changes typical for LVH.

Two types of repolarizations are considered positive findings:

- Mid ST elevation, with a large positive T wave
- Slight mid ST depression that is upsloping, with a negative T wave

Pediatric LVH voltage criteria are highly age dependent. Appendix A, “Normal Measurement Values” includes the values that are considered significant for LVH voltages.

Biventricular Hypertrophy

The category of **Biventricular Hypertrophy (BVH)** combines findings of right and left ventricular hypertrophy.

Associated **Right Ventricular Hypertrophy (RVH)** is considered when an R amplitude greater than 1.0 mV in V1 exists with the presence of **Left Ventricular Hypertrophy (LVH)**.

Associated LVH is considered when RVH statements are combined with a Q wave greater than 10 ms in duration, greater than 0.07 mV in amplitude, and an R wave greater than 1.0 mV in lead V6.

BVH is also considered when the combined amplitudes of R and S exceed 6.0 mV in two of the following leads: V2, V3, or V4.

4. Davignon A, Rautuharju P, Boiselle E, et al. “Normal ECG Standards for Infants and Children.” *Pediatric Cardiology* 1:123-131 (1979/80). For more information, see Appendix A, “Normal Measurement Values.”

If RVH and LVH statements with high severity are generated from previous RVH and LVH categories, a statement of BVH is generated. The BVH statement suppresses individual RVH and LVH statements.

Low Voltage

All leads are examined for QRS peak-to-peak voltage.

- Frontal leads — If no lead has a value exceeding 0.60 mV, the ECG is considered **Borderline Low Voltage**. If no value exceeds 0.50 mV, the ECG is considered **Definite Low Voltage**, an abnormal finding.
- Precordial leads — If no lead has a value exceeding 1.00 mV, the ECG is considered **Definite Low Voltage**, an abnormal finding.

Combinations of low voltage statements, rightward deviation of the frontal P and QRS axes, and right atrial enlargement may generate statements suggesting the likelihood of chronic pulmonary disease.

Q Wave Abnormality and Myocardial Infarct

A statement of borderline Q wave abnormalities in an individual lead group is generated in the presence of large Q waves in two leads out of that group.

Q waves greater than one-fifth of the R wave amplitude generate a statement that the abnormal Q wave suggests infarct.

ST Depression

ST depression is determined in anterior, lateral, and inferior lead groups.

ST depression of more than 0.20 mV in one lead group produces a **Nonspecific ST Depression** statement.

If tachycardia is present, the statement of **ST depression, probably rate related**, is generated.

Any type of hypertrophy or ventricular conduction delay suppresses statements from this category.

T Wave Abnormality

Inverted T waves are sought in anterior, lateral, anterolateral, and inferior lead groups.

A **Tall T Wave Abnormality** statement is generated if the amplitude of the inverted T exceeds 1.0 mV in two or more leads in the particular lead group.

If RVH co-exists with inverted T waves in the anterior lead groups, the statement **Abnormal T**, **Probably Secondary to RVH**, **Anterior Leads** is generated.

The statement **Abnormal T**, **Probably Due to LVH**, **Anterolateral Leads** is generated if LVH co-exists with inverted waves in the anterolateral lead group.

Repolarization Abnormality

This category combines statements from the previous ST depression and inverted T wave categories to generate statements of repolarization abnormality. If ST depression and inverted T are found in the anterior lead group, a statement is generated to indicate **Repolarization Abnormality, Anterior Leads**.

ST Elevation, Pericarditis, and Early Repolarization

All leads are tested for ST elevation. ST elevation greater than 0.15 mV in these leads generates a statement suggesting a probable normal variation. Any hypertrophy and ventricular conduction delay suppresses statements from this category.

If ST elevation is seen on all anterior, lateral, and inferior lead groups, pericarditis is considered in children ages 5 to 15 years old.

For ECGs with nonspecific ST elevation and no T wave inversion, probable early repolarization is suggested in children ages 13 to 15 years old.

Tall T Waves

All leads are examined for the presence of T waves with amplitudes that exceed 1.20 mV, or that exceed 0.50 mV and are more than half the size of the peak-to-peak QRS voltage. The presence of such T waves may generate statements with the possibility of metabolic or electrolyte abnormalities.

QT Abnormality and Electrolyte Disturbance

A QT interval corrected for heart rate (QTc) shorter than 340 ms is considered to be **Borderline Short QT Interval** with a severity of **OTHERWISE NORMAL (ON)**.

A borderline prolonged QTc is greater than the following values, by age:

- 450 ms in children below 5 years
- 454 ms for children 5 to 12 years old
- 458 ms for boys 13 years and older
- 465 ms for females 13 years and older

An additional 20 ms qualifies as prolonged QT.[5]

- 470 ms in children below 5 years
- 474 ms for children 5 to 12 years old

5. Rautaharju PM, Zhou SH, Wong S, et al. “Sex differences in the evolution of the electrocardiographic QT interval with age.” *Canadian Journal of Cardiology* 8(7): 690-695 (1992).

- 478 ms for boys 13 years and older
- 485 ms for females 13 years and older

With RVH, LSH, LVH, BVH, or VCD present, the statement **Prolonged QTc Probably Secondary to Wide QRS Complex** is generated.

Hypercalcemia is suggested if the QTc is shorter than 310 ms.

Hypocalcemia is suggested by a significantly prolonged QTc interval (> 520 ms).

Hypokalemia is suggested by a significantly prolonged QTc interval (> 520 ms) combined with ST segment depression and a positive T wave in multiple leads.

Congenital Heart Defects

Various congenital cardiac conditions are suggested by varying combinations of atrial abnormalities, ventricular hypertrophy, ventricular conduction delays, QRS axis deviations, and QRS morphological features.

5

Critical Value Statements

Introduction

The Philips DXL ECG Algorithm makes it simple to align with the Joint Commission's National Patient Safety Goals to improve the timeliness of reporting critical test results and their receipt by a responsible licensed caregiver. The *Critical Values* feature is a configurable option that summarizes four critical values for ECG interpretation using simple terminology. When appropriate, the DXL Algorithm outputs an emphasized statement to advise caregivers of the need for urgent care, with the goal of reducing time from discovery of a critical cardiac event to intervention.

Thirty interpretive statements are summarized in the following four Critical Values:

- Acute Myocardial Infarction
- Acute Ischemia
- Complete Heart Block
- Very High Heart Rate

Table 5-1 through Table 5-4 on the following pages provide a list of all of the interpretive statements that will generate a Critical Value. Multiple interpretive statements can trigger an individual Critical Value, and this Critical Value statement appears in the interpretive statement block on the ECG report, and a second notation also appears at the bottom right of the ECG report for additional emphasis.

Acute Myocardial Infarction Critical Value Statements

If any of the interpretive statements listed in Table 5-1 result from the measurements generated by an ECG, the Critical Value statement **Acute MI** will appear on the ECG report.

Table 5-1 Acute Myocardial Infarct Critical Value Statements

Statement Code	Interpretive Statement
AMIA	Anterior infarct, acute.....ST >0.25mV, V2-V5

Table 5-1 Acute Myocardial Infarct Critical Value Statements *(continued)*

Statement Code	Interpretive Statement
AMIAP	Probable anterior infarct, acute.....ST >0.15mV, upright T, V2-V5
AMIPA	Anterior infarct, possibly acute.....ST >0.15mV, upright T, V2-V5
AMIAD	Anterior infarct, acute (LAD).....ST >0.25mV, V2-V5
IMIAP	Probable inferior infarct, acute.....ST>0.10mV, II III aVF
IMIPA	Inferior infarct, possibly acute.....Q >30mS, ST >0.10mV, II III aVF
IMIA	Inferior infarct, acute.....ST>0.10mV, T upright, II III aVF
IMIAR	Inferior infarct, acute (RCA).....ST>0.10mV in III > II
IMIAX	Inferior infarct, acute (LCx).....ST>0.10mV, II III aVF, STd V1-V3
PMIA	Posterior infarct, acute.....ST<-0.1 V1-V3 or ST>.05 V7-V9
PMIAP	Probable posterior infarct, acute.....ST<-.05 V1-V3 or >.05 V7-V9
PMIAX	Posterior infarct, acute (LCx).....ST<-0.1 V1-V3 or ST>.05 V7-V9
IPMIA	Inferoposterior infarct, acute.....ST>.1 inf, <-.1 V1-3 or >.05 V7-9
IPMIAR	Inferoposterior infarct, acute (RCA).....ST>.1 inf, <-.1 ant or>.05 V3R-5R
IPMIAX	Inferoposterior infarct, acute (LCx).....ST>.1 inf, <-.1 V1-3 or >.05 V7-9
LMIAP	Probable lateral infarct, acute.....Q >28mS, ST>0.10mV, I aVL V5 V6
LMIPA	Lateral infarct, possibly acute.....Q >28mS, ST >0.10mV, I aVL V5 V6
LMIA	Lateral infarct, acute.....ST >.10mV, I aVL V5 V6
LMIAD	Lateral infarct, acute (LAD).....ST >.10mV, I aVL V5 V6
ILMIA	Inferolateral infarct, acute.....ST>.10mV, inf-lat leads
ILMIAX	Inferolateral infarct, acute (LCx)....ST>.10mV, inf-lat leads
ILMIAR	Inferolateral infarct, acute (RCA)....ST>.10mV, inf-lat leads

Table 5-1 Acute Myocardial Infarct Critical Value Statements *(continued)*

Statement Code	Interpretive Statement
ASMIAP	Probable anteroseptal infarct, acute.....ST>0.15mV, T upright, V1-V3
ASMIPA	Anteroseptal infarct, possibly acute.....Q>30mS, ST>0.15mV, V1-V3
ASMIA	Anteroseptal infarct, acute.....ST >0.25mV, V1-V3
ASMIAD	Anteroseptal infarct, acute (LAD).....ST >0.25mV, V1-V3
EAMIA	Extensive anterior infarct, acute.....ST >0.15mV, V1-V6
EAMIAD	Extensive anterior infarct, acute (LAD)....ST >0.15mV, V1-V6
EAMIPA	Extensive anterior infarct, possibly acute.....Q >35mS, ST >0.15mV, V1-V6
ALIAP	Probable anterolateral infarct, acute.....ST >0.15mV, V2-V5
ALIPA	Anterolateral infarct, possibly acute.....Q >35mS, ST >0.15mV, V2-V6
ALIA	Anterolateral infarct, acute.....Q >35mS, ST >0.20mV, V2-V6
ALIAD	Anterolateral infarct, acute (LAD).....Q >35mS, ST >0.20mV, V1-V6
RMIAP	Probable right ventricular infarct, acute.....ST>.08, V3R-V5R, aVR & STd in lat
RMIA	Right ventricular infarct, acute.....ST>.10, V3R-V5R, aVR & STd in lat
RMIAR	Right ventricular infarct, acute (RCA).....ST>.08, aVR V3R-V5R & STd lat lds

Tachycardia Critical Value Statements

If any of the interpretive statements listed in Table 5-2 result from the measurements generated by an ECG, the Critical Value statement **Very High Heart Rate** will appear on the ECG report.

Table 5-2 Tachycardia Critical Value Statements

Statement Code	Interpretive Statement
ETACH	Extreme tachycardia.....V-rate >(220-age)
TACHW	Wide-QRS tachycardia.....V-rate> ** , QRSd> **
VTACH	Extreme tachycardia with wide complex, no further rhythm analysis attempted

Complete Heart Block Critical Value Statements

If any of the interpretive statements listed in Table 5-3 result from the measurements generated by an ECG, the Critical Value statement **Complete Heart Block** will appear on the ECG report.

Table 5-3 Complete Heart Block Critical Value Statements

Statement Code	Interpretive Statement
3AVB	AV block, complete (third-degree)....V-rate<45, AV dissociation
3AVBIR	Complete AV block with wide QRS complex.....V-rate< ** , QRSd> ** , AV dissoc
3AVBFF	A-flutter/fibrillation w/ complete AV block...A-rate>220, V-rate< ** , AV dissoc

Acute Ischemia Critical Value Statements

If any of the interpretive statements listed in Table 5-4 result from the measurements generated by an ECG, the Critical Value statement **Acute Ischemia** will appear on the ECG report.

Table 5-4 Acute Ischemia Critical Value Statements

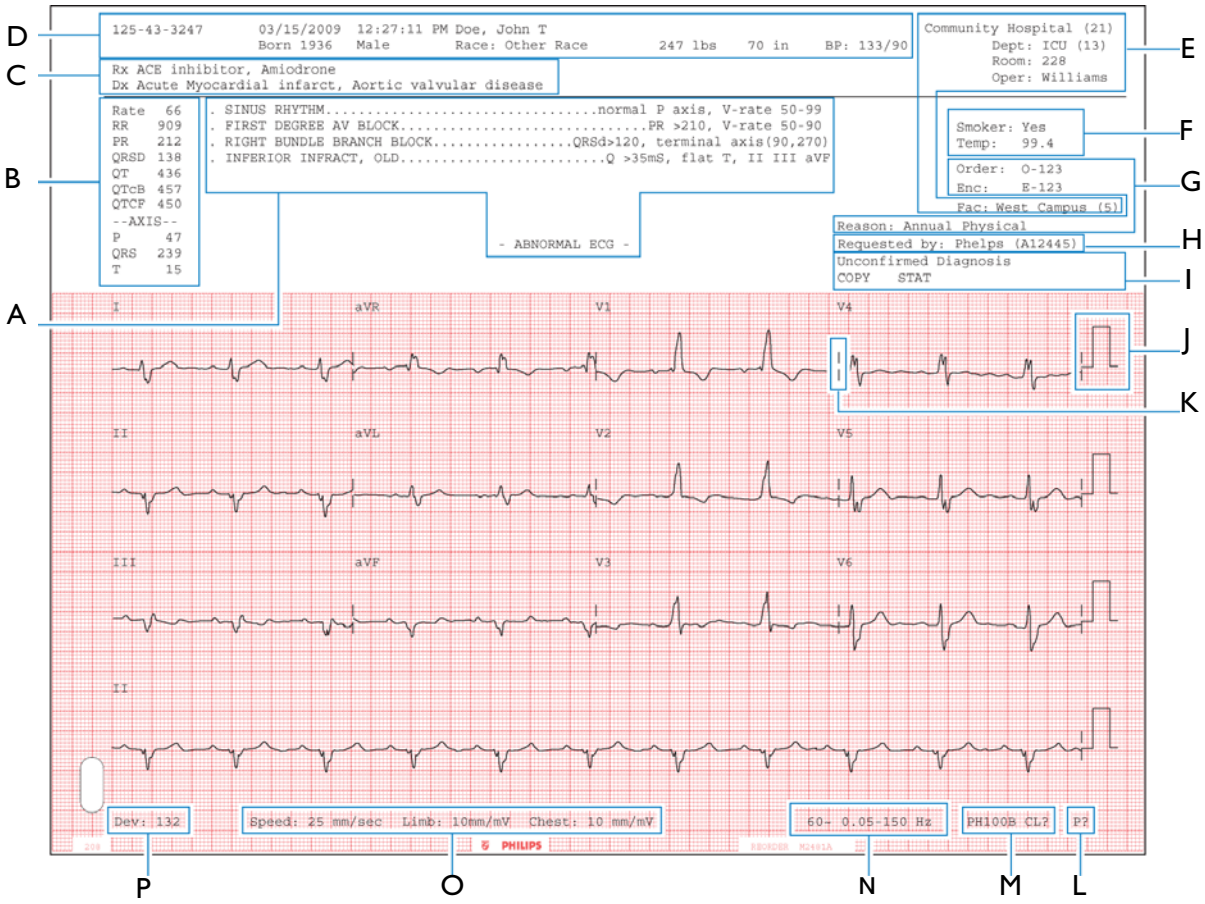
Statement Code	Interpretive Statement
LMMVD	Repolarization abnormality, severe global ischemia (LM/3VD)

6

Reading the Printed ECG Report

The following ECG report formats may be generated by Philips Medical Systems equipment. For more information on available printed report formats, see your product documentation.

Figure 6-1 A 12-Lead 3x4, 1R Report (page one)



A Interpretive, Reason, and Severity Statements (see page 6-3)

B Basic Measurements (see page 6-5)

C Patient ID Clinical Information (see page 6-7)

D Patient ID Information (see page 6-9)

I Report Information (see page 6-13)

J Calibration Information (see page 6-14)

K Time Separator (see page 6-16)

L Pacing Detection Setting (see page 6-17)

E Institution Information (see page 6-10)

F Configurable Clinical Information (see page 6-11)

G ECG Order Information (see page 6-12)

H Physician Information (see page 6-13)

M Algorithm Version (see page 6-18)

N Filter Settings (see page 6-19)

O Speed and Sensitivity Settings (see page 6-19)

P Device Identification Number (page 6-20)

Additional patient information fields may appear on the top of a second page of the ECG report if more than two clinical fields (Rx, Dx, Sx, Hx) are entered with the Patient ID information.

Additional configurable clinical information fields may also appear on the top of a second page of the ECG report if more than four fields are configured.

Figure 6-2 A 12-Lead 3x4, 1R Report (page two)

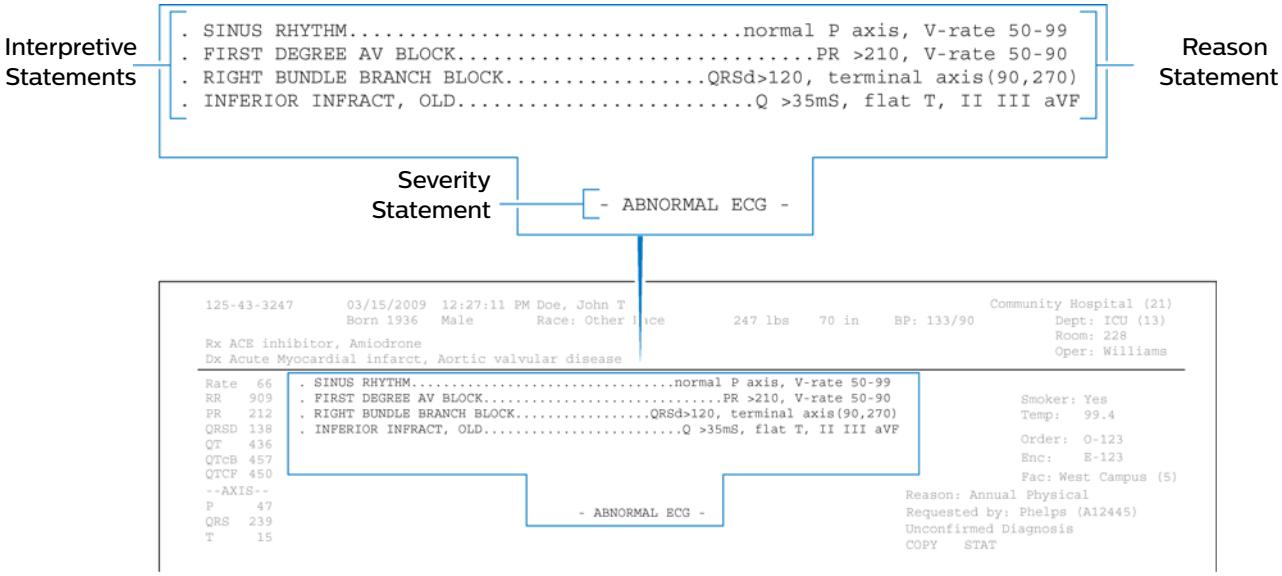
Q Additional Configurable Clinical Information (see page 6-11)

R Additional Patient Clinical Information Fields (see page 6-7)

Interpretive, Reason, and Severity Statements

This area of the report contains the interpretive, reason, and severity statements generated by the Philips DXL ECG Algorithm.

Figure 6-3 Interpretive, Reason, and Severity Statements on the ECG Report



Interpretive statements may include a reason statement that summarizes the criteria that generated the interpretive statement.

NOTE The interpretive statements may include quality statements that describe a signal quality problem that occurred during recording, such as **ARTIFACT IN LEAD(S) I, III, aVL**.

Severity Statement

Each interpretive statement included on the ECG report has an associated severity. Severities that are more abnormal override lesser severities. The severities of all selected interpretive statements are combined to determine the overall severity of the ECG. This severity code is printed on the front page of the ECG report.

Table 6-1 Overall ECG Severity with Code

Severity	Code
No Severity	NS
Normal ECG	NO
Otherwise Normal ECG	ON
Borderline ECG	BO
Abnormal ECG	AB

Table 6-1 Overall ECG Severity with Code *(continued)*

Severity	Code
Defective ECG	DE

Critical Values

When the **Critical Values** setting is enabled on the acquisition equipment, statements may appear on the ECG report if specific interpretive statements are generated by the Philips DXL ECG Algorithm. These statements are intended to alert caregivers of an ongoing or imminent cardiac event, such as a silent MI, that require immediate clinical treatment. This feature is provided in part to help satisfy Section 2C of Goal 2 of the 2009 National Patient Safety Goals of the United States of America, as defined by the Joint Commission on Accreditation of Healthcare Organizations.

There are four Critical Value statements that may appear on the ECG report. These statements are shown in Figure 6-4 through Figure 6-7.

About the Extreme Tachycardia Statement

The extreme tachycardia statement **Very High Heart Rate** is generated by the following formula: the measured heart rate in beats per minute, minus the patient age in years. If this value is 150 bpm or higher, the measurement will generate the extreme tachycardia statement. If no patient age is specified for the ECG, the default patient age specified on the acquisition device is used.

Figure 6-4 Acute Myocardial Infarction Statement on ECG Report

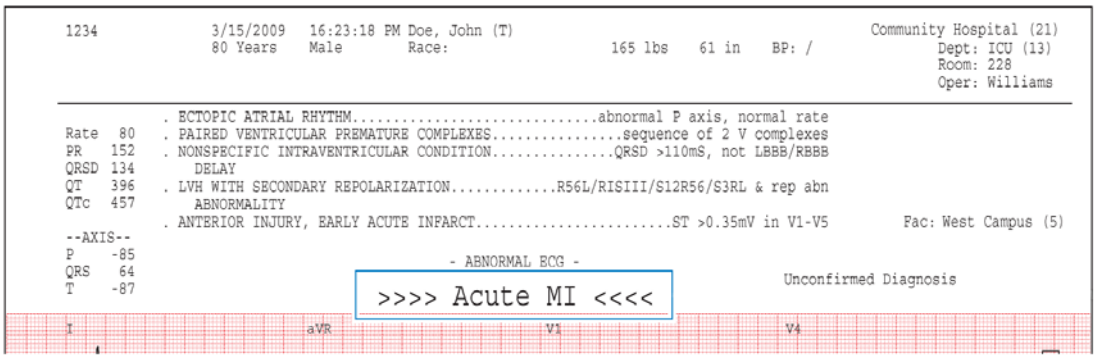


Figure 6-5 Extreme Tachycardia Statement on ECG Report

Figure 6-6 Complete Heart Block Statement on ECG Report

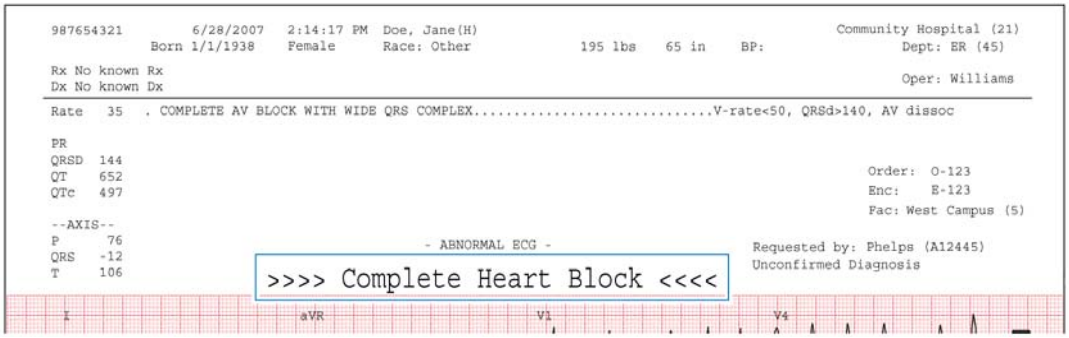


Figure 6-7 Acute Ischemia Statement on ECG Report

Basic Measurements

These measurements provide standard interval and duration measurements in milliseconds, and limb lead axis measurements in degrees. These are the values measured from the representative beat pattern in the ECG.

About Rate-Corrected QT Interval Settings

The default QT rate correction formula available on the acquisition device is Bazett's formula. The Fridericia, Hodges, and Framingham rate-corrected QT interval settings can be enabled on the acquisition device.

The QT rate correction formulas are listed below:

Bazett's

$QTc = QT / \sqrt{RR}$

Fridericia

$QTc = QT / \sqrt[3]{RR}$

Hodges

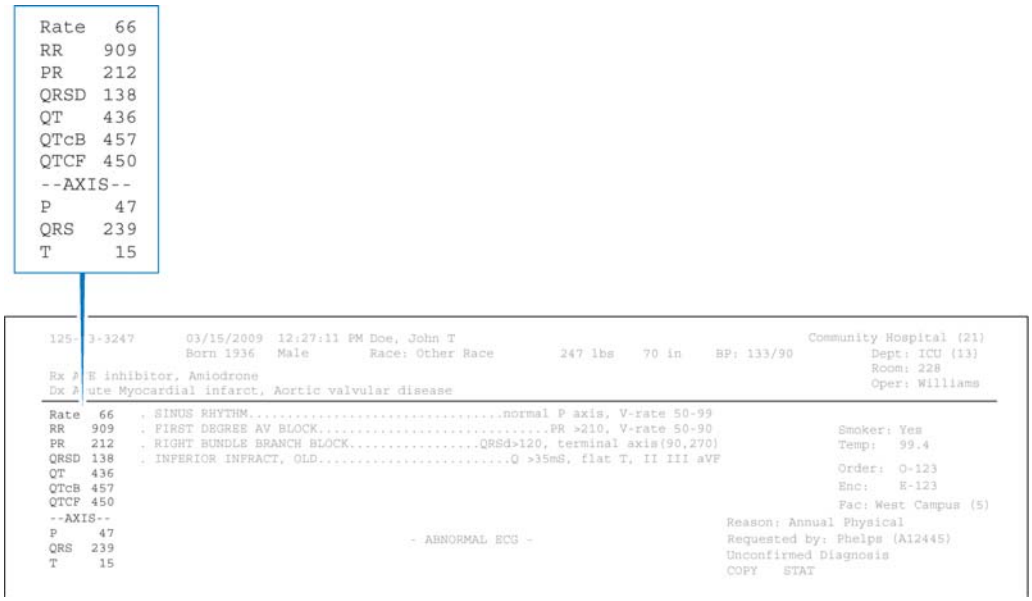
$QTc = QT + 1.75(HR - 60)$

Framingham

$QTc = QT + 0.154(1 - RR)$

For certain clinical situations, the Fridericia, Hodges, or Framingham corrected QT intervals may be preferable over Bazett's, and these additional measurements may be configured to appear in the measurements section of the printed ECG report.

Figure 6-8 The Bazett's (QTcB) and Fredericia (QTcF) Rate-Corrected QT interval on the printed ECG report



NOTE Some reports do not include the heart rate (RATE) in Basic Measurements, but do include a heart rate above the interpretive statements. This rate may be edited.

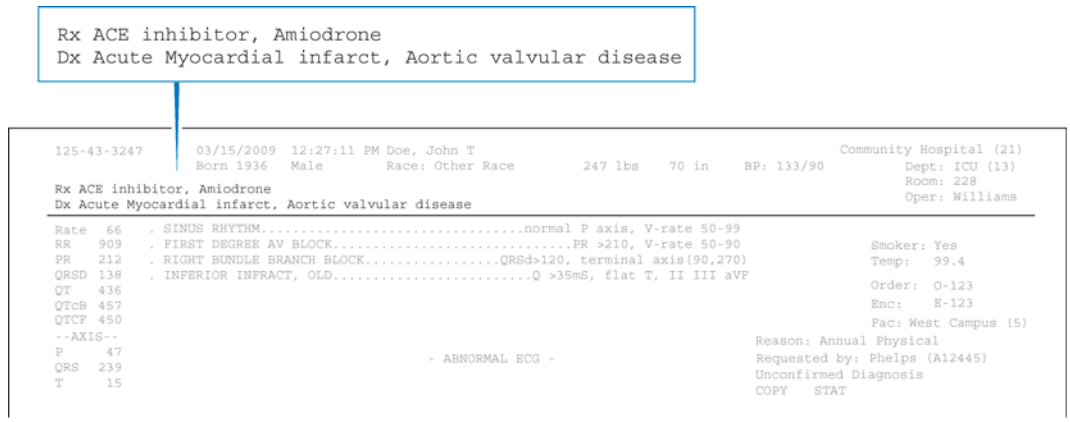
Table 6-2 Basic Measurements

Label	Description	Units
RATE	Heart rate	beats per minute
RR	RR interval	milliseconds
PR	PR interval	milliseconds
QRSD	QRS duration	milliseconds
QT	QT interval	milliseconds
QTcB	Bazett's Rate-Corrected QT interval	milliseconds
QTcF	Fridericia Rate-Corrected QT interval	milliseconds
QTcH	Hodges Rate-Corrected QT interval	milliseconds
QTcFm	Framingham Rate-Corrected QT interval	milliseconds
P	Frontal P axis	degrees
QRS	Frontal QRS axis	degrees
T	Frontal T axis	degrees

Patient ID Clinical Information

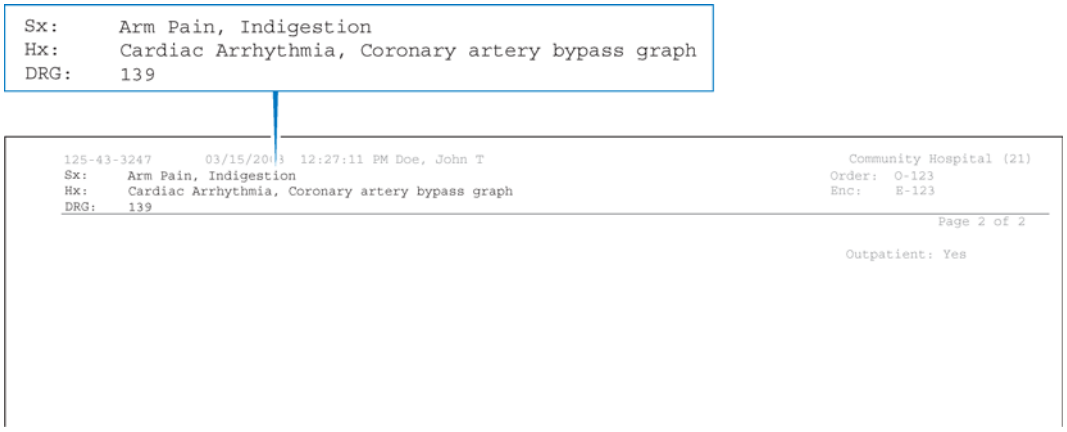
This area of page one or page two of the ECG report contains clinical patient information that is entered on the patient information entry screen, or that is contained in the order associated with the ECG. This includes information about the patient's Medications (Rx), Diagnoses (Dx), Symptoms (Sx), History (Hx), and a Diagnosis Related Group (DRG) code. The example below is for informational purposes only.

Figure 6-9 Patient ID Clinical Information on the ECG Report (page one)



If more than two Patient ID Clinical Information fields are entered, the third and subsequent fields appear at the top of a second page of the report.

Figure 6-10 Patient ID Clinical Information on the ECG Report (page two)



Patient ID Information

This section contains patient identification information. The example below is for informational purposes only.

Figure 6-11 Patient ID Information on the ECG Report

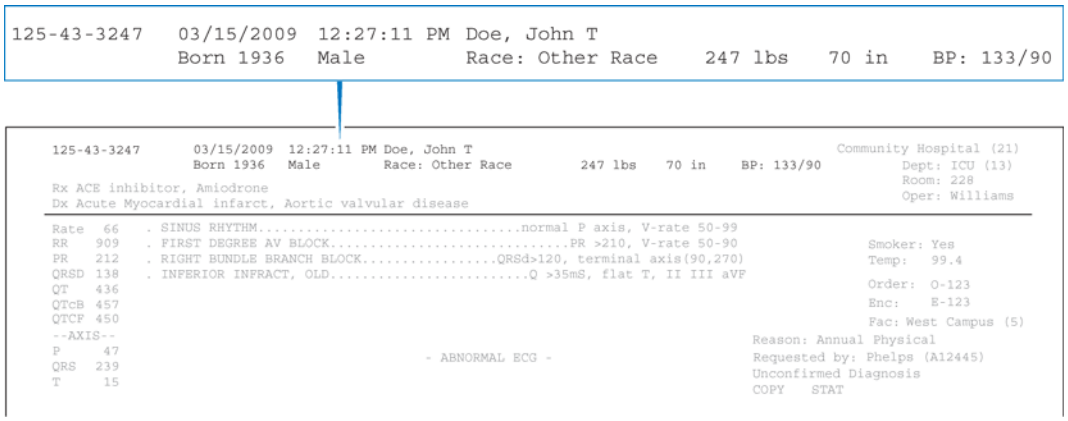


Table 6-3 Patient ID Information

Label	Description
123456789	Patient identification number
09/06/2009; 12:27:11 PM	▪ Date and time of ECG acquisition ▪ Cannot be edited
Doe, John T.	Patient name
70 Years	Patient age (may be configured to display date of birth)
Male	Patient gender
Race	Patient ethnicity
247 lbs, 70 in.	Patient weight and height
BP: 133/90	Patient blood pressure (mm/Hg)

Institution Information

This block of identification information is optional and is fully configurable. The example below is for informational purposes only.

Figure 6-12 Institution Information on the ECG Report

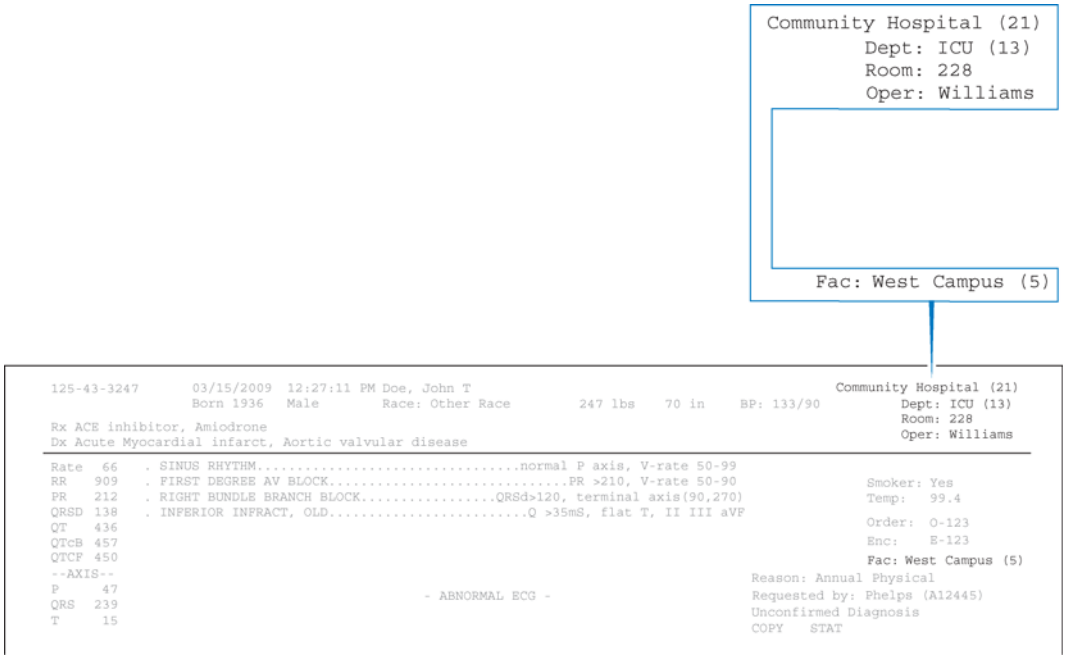


Table 6-4 Institution Information

Label	Description
Community Hospital (21)	Name and ID number of institution
Dept: ICU (13)	Name and ID number of department
Room: 228	Room number of patient or room number where ECG was acquired
Oper: Williams	Operator identification
Fac: West Campus (5)	Name and ID number of facility or other unit within an institution

Configurable Clinical Information

This information is configurable to fit specific clinical needs. Up to eight configurable clinical information fields may be available for use on the acquisition device.

The first four clinical fields appear on page one of the ECG report. The fifth and subsequent fields appear on page two of the ECG report. The examples below are for informational purposes only.

Figure 6-13 Configurable Clinical Information on the ECG Report (page one)

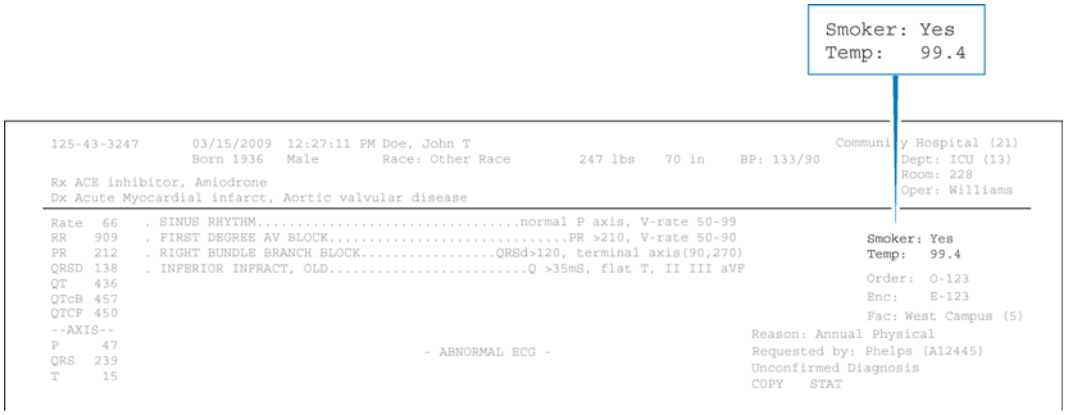


Figure 6-14 Configurable Clinical Information on the ECG Report (page two)

ECG Order Information

This area of the ECG report is optional and fully configurable, and is intended to meet the requirements of an order management system.

Figure 6-15 ECG Order Information on the ECG Report

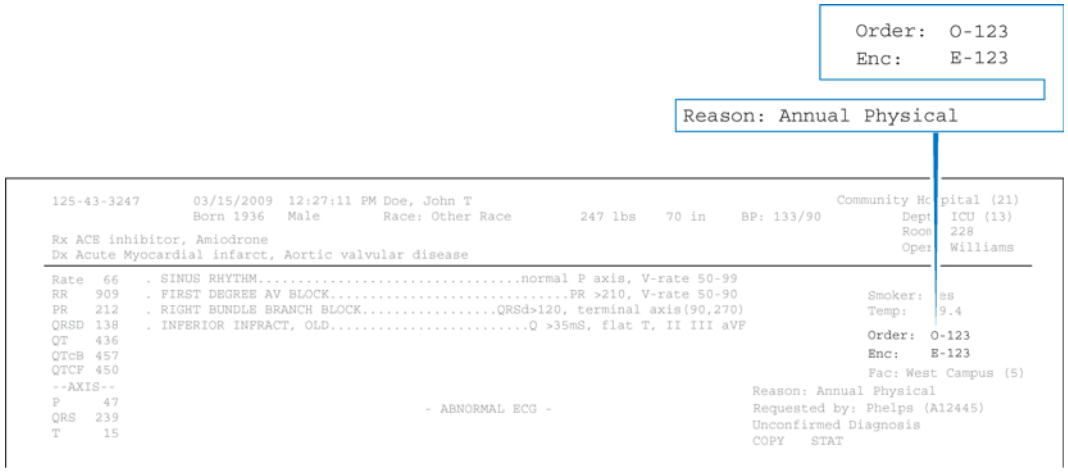


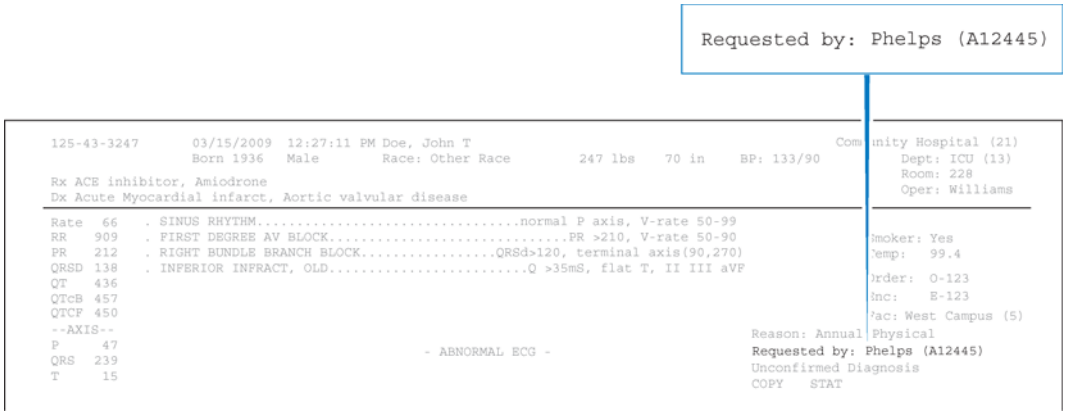
Table 6-5 ECG Order Information

Label	Description
Order: 0-123	Institution-defined order number, part of order management system.
Enc: E-123	Institution-defined encounter number, part of order management system.
Reason: Annual Physical	The reason for acquiring the ECG, may be part of an order management system.

Physician Information

This information block is optional, and contains physician identification information, including the name of the ordering physician, and may include the NPI (National Provider Identifier) number in parenthesis. The NPI is only applicable to providers inside the United States.

Figure 6-16 Physician Information on the ECG report



Report Information

Information about the status of the ECG report is included in this section, and may include a statement indicating that the ECG report has not yet been overread by a qualified physician.

Figure 6-17 Report Information on the ECG Report

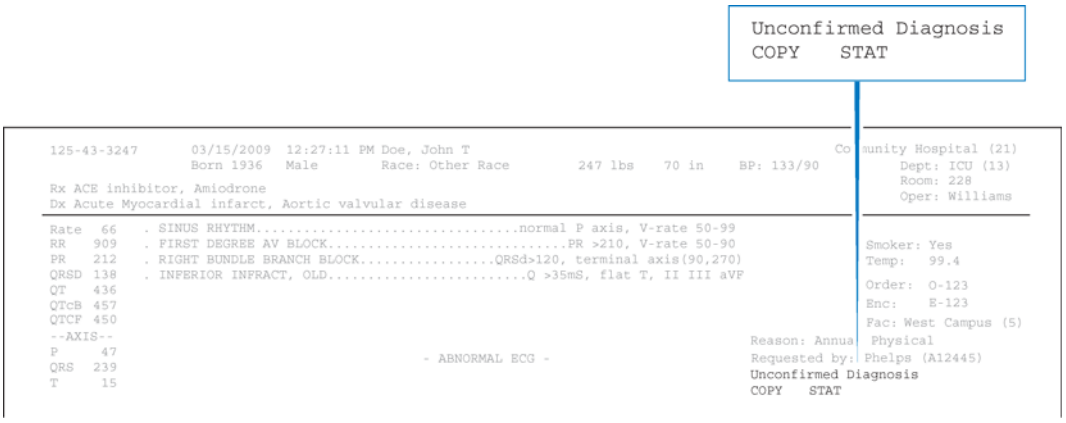


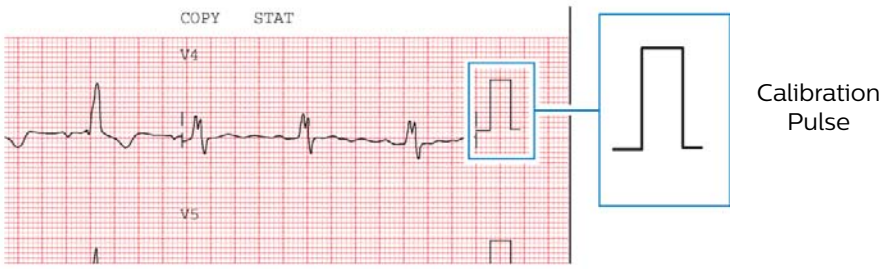
Table 6-6 Report Information

Label	Description
Unconfirmed Diagnosis	■ Indicates that the ECG report has not been overread by a qualified physician. ■ This statement may be customized by an institution.
COPY	The ECG report is a printed copy of an original.
STAT	The ECG report is designated as STAT.
Non-standard lead gains	■ The limb leads or precordial leads were recorded at a gain other than the standard 10mm/mV. ■ see “Calibration Information” on page 6-14..

Calibration Information

The calibration pulse is the rectangular waveform shown in each line of ECG trace. It shows the hypothetical deflection of the trace in response to a 1 mV calibration pulse applied to the acquisition circuitry.

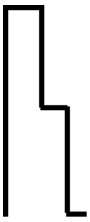
Figure 6-18 Calibration Pulse on the ECG Report



The shape of the calibration pulse reflects the scaling of the trace.

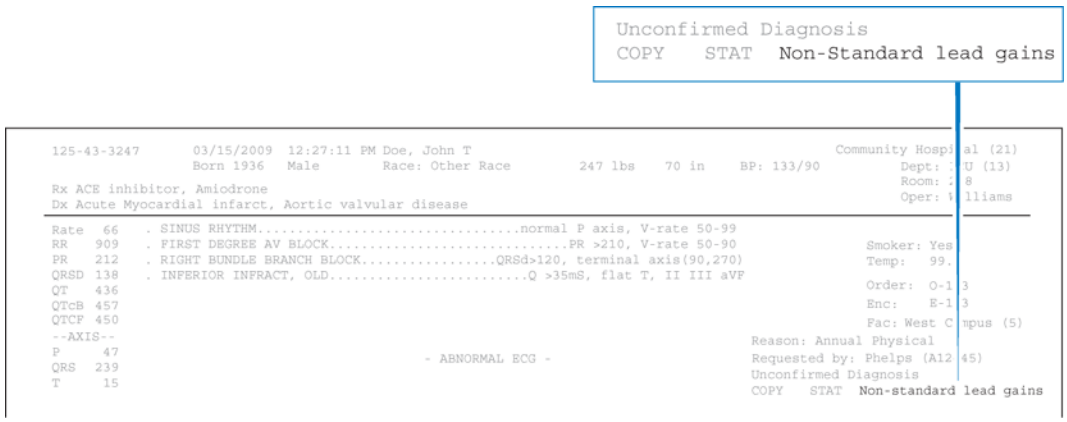
- If the calibration pulse is square ⌐¬ the precordial leads and limb leads were recorded at the same scale.
- If the calibration pulse is stepped ⌐┐ the precordial leads were recorded at half the scale of the limb leads.

Table 6-7 Calibration Pulse Shapes

Calibration Pulse Shape	Limb (mm/mV)	Precordial (mm/mV)
	5	5
	5	2.5
	10	10
	10	5
	20	20
	20	10

NOTE For ECG recordings where the precordial leads or limb leads were recorded at a gain other than 10mm/mV, the statement **Non-standard lead gains** appears in the Report Information section on the printed report.

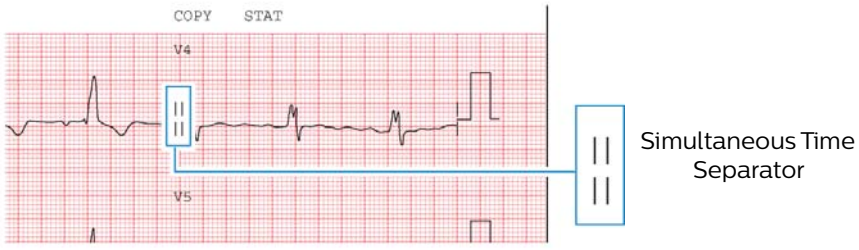
Figure 6-19 Calibration information on the ECG report



Time Separator

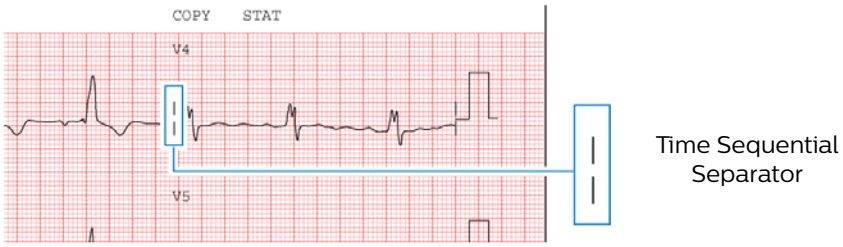
The time separator marks indicate whether the ECG data is displayed on the printed ECG report simultaneously or time-sequentially. The data for each lead is always acquired simultaneously.

Figure 6-20 Simultaneous time separator on ECG report



The double line indicates that the ECG data for each lead is displayed simultaneously. The starting point of each lead is the same time even though they may appear to start at different times on the printed ECG report.

Figure 6-21 Time sequential separator on ECG report



The single line indicates that the ECG data for each lead is displayed over a continuous period of time. For example, on a 3x4 grid all signals start at 0 in the

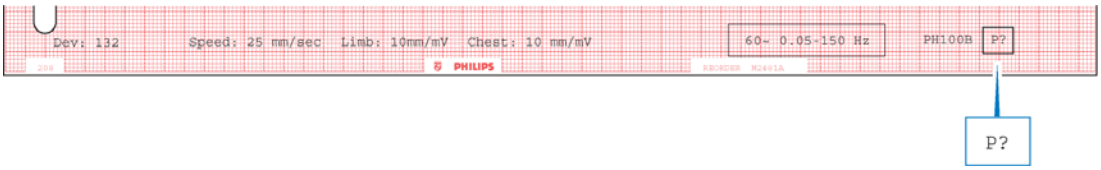
first column, 2.5 seconds in the second column, 5.0 seconds in the third column, and 7.5 seconds in the fourth column.

Pacing Detection Settings

This area of the report contains information about the pacing detection settings that were selected when the ECG report was printed.

Pacemaker pulses that are detected by the acquisition device are marked on the ECG report with small vertical tick marks. These marks enable the overreader to identify false pacemaker pulse detections, or if true pulses are not being detected.

Figure 6-22 Pacing Detection Setting on the ECG report



The table below describes the possible Pacing Detection Settings available on the acquisition device, along with the setting code that appears on the printed ECG report.

Table 6-8 Pacing Detection Settings

Setting	Description	ECG Report Code
Paced Unknown	▪ This is the default setting and normally is used for both paced and non-paced patients. ▪ Pacemaker pulse detection is on and is at normal sensitivity. ▪ Occasional false pacemaker pulse detections may occur in ECGs with excessive noise. ▪ False detections may result in an incorrect interpretive statement appearing on the report. ▪ Small amplitude pacemaker pulses may not be detected using this setting.	**P?**

Table 6-8 Pacing Detection Settings *(continued)*

Setting	Description	ECG Report Code
Non-paced	▪ Pacemaker pulse detection is off. ▪ Use this setting if there are false pacemaker pulse detections from noise, or if incorrect interpretive statements or inappropriate paced ECG complexes appear on the report.	No code appears on the ECG report if the Non-paced setting is selected.
Paced	▪ Pacemaker pulse detection is on and is set at a higher sensitivity. ▪ Use this setting if small amplitude pacemaker pulses are not being detected at the default (**Not Known if Paced**) setting. ▪ False pacemaker pulse detections may occur if the ECG is noisy.	**P**
Paced (magnet)	▪ Use this setting if the ECG is acquired with an active pacemaker magnet or programmer in place. ▪ Pacemaker pulse detection is on and is at a higher sensitivity. ▪ Magnets or programmers often put the pacemaker in a fixed-rate, non-sensing mode. ▪ The statement **ECG ACQUIRED WITH MAGNET IN PLACE** is printed on the ECG report. This statement notifies the overreader that a magnet or programmer was used and would explain the fixed rate behavior of the pacer.	**PM**

Algorithm Version Number and Options

The version number of the Philips DXL ECG Algorithm is printed at the bottom of the ECG Report, along with notations for enabled algorithm options. The algorithm version number appears as **PH100B**. A Critical Values (**C**) lead reversal detection symbol (**L?**) can also appear in this area of the ECG report if these optional features are is enabled.

Figure 6-23 Algorithm Version Number, and Lead Reversal Detection Symbol on ECG report

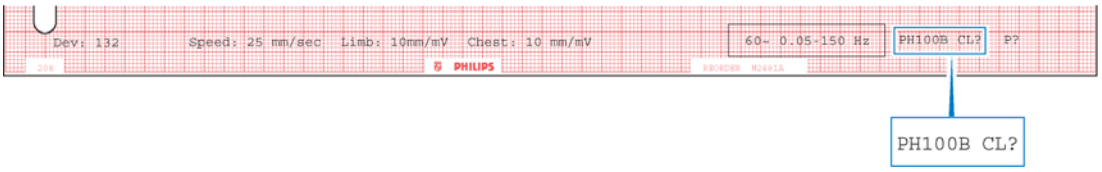


Table 6-9 Algorithm Version Number and Lead Revesal Detection Symbol

Label	Description
PH100B PH110C	▪ PH refers to Philips ▪ **10** or **11** refers to the version of the measurement program ▪ **0B** or **0C** refers to the criteria version installed on the cardiograph
C	This symbol appears on the report if the optional Critical Values feature is enabled on the acquisition device
L?	▪ This symbol may appear with the algorithm version number ▪ If this symbol appears, it designates that the optional lead reversal detection feature is enabled on the acquisition device, and that the device detected a lead reversal that the operator overrode when printing the ECG

Speed and Sensitivity Settings

This area contains information about the speed and sensitivity settings that were used for the ECG recording.

Figure 6-24 Speed and Sensitivity Settings on the ECG Report

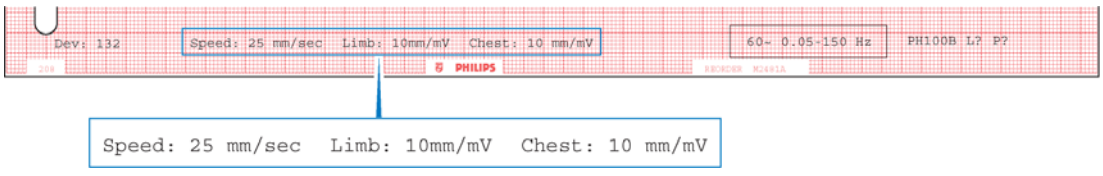


Table 6-10 Speed and Sensitivity Settings

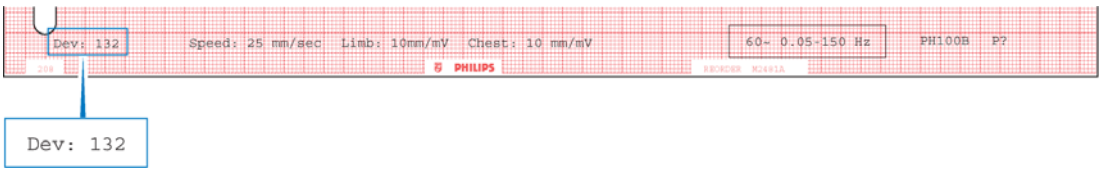
Label	Description
Speed	■ The speed at which the ECG was printed ■ Available settings: – 25mm/sec – 50 mm/sec
Limb	■ The limb lead sensitivity setting ■ Available settings: – 5, 10, or 20 mm/mV
Chest	■ Precordial lead sensitivity setting ■ Available settings: – 2.5, 5, 10, or 20 mm/mV

NOTE For ECG recordings where the precordial leads or limb leads were recorded at a gain other than 10mm/mV, the statement **Non-standard lead gains** appears in the Report Information section on the printed report.

Device Identification Number

This identification number may be entered at the acquisition device. This number is used to identify the individual device that acquired the ECG.

Figure 6-25 Device ID on the ECG Report



12-Lead ECG Report Examples

The following section includes examples of other 12-lead ECG formats.

- 3x4, 3R report with Standard Leads
- 3x4, 1R report with Cabrera Leads
- 6x2 report (5-second waveform segments) with Cabrera Leads

- 12x1 report with Cabrera Leads. The 12x1 report shows 10 seconds of continuous waveform data for 12 leads and includes a second page with interpretive, reason, and severity statements (if configured).
- Panoramic (Pan-12) report with Cabrera Leads. The Pan-12 report shows a one-second representative complex for each Cabrera Lead and three pre-selected rhythm strips at the bottom (aVF, V2, V5).

Figure 6-26 3x4, 3R Report with Standard Leads

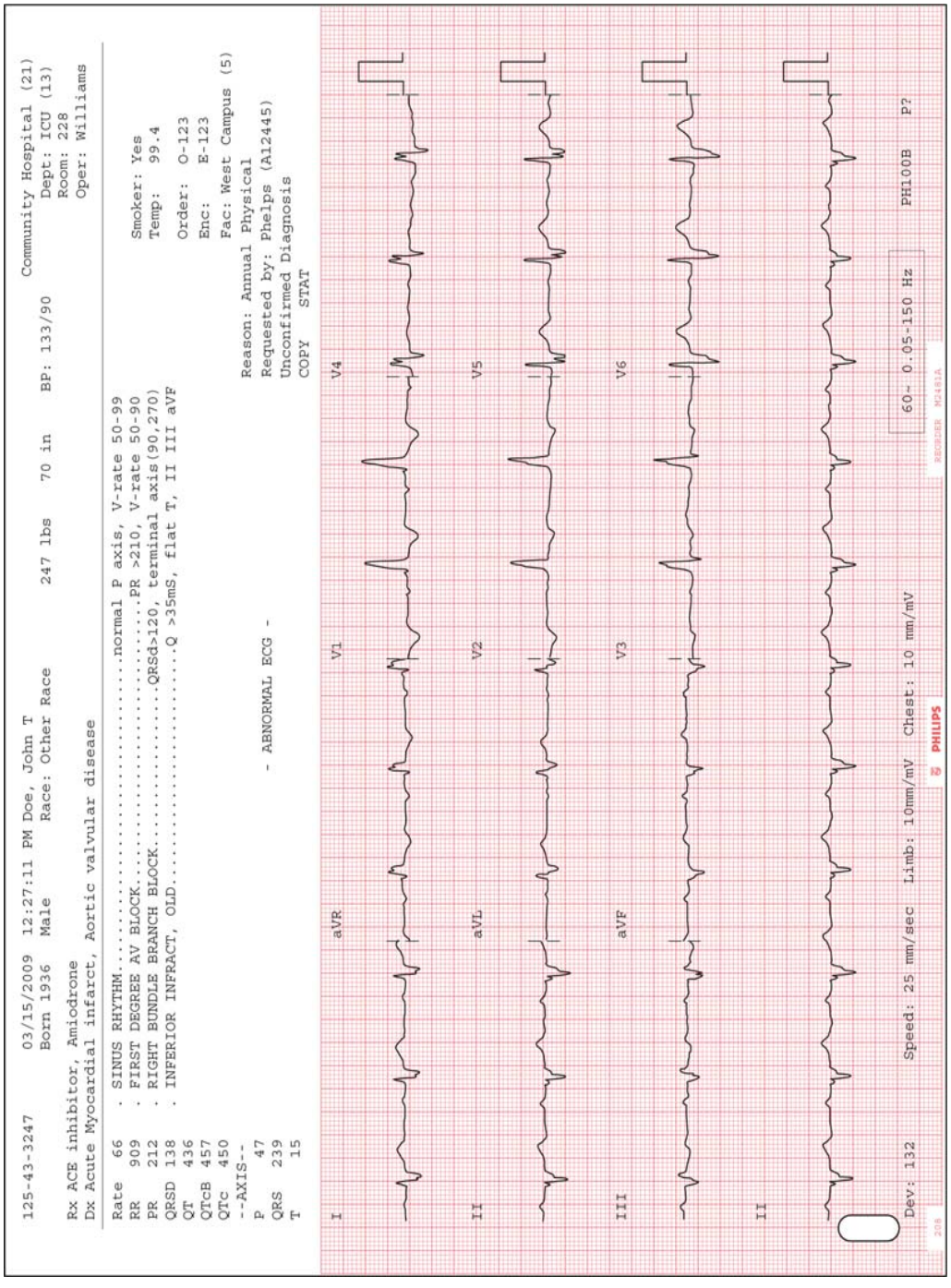


Figure 6-27 3x4, 1R Report with Cabrera Leads and Simultaneous Acquisition

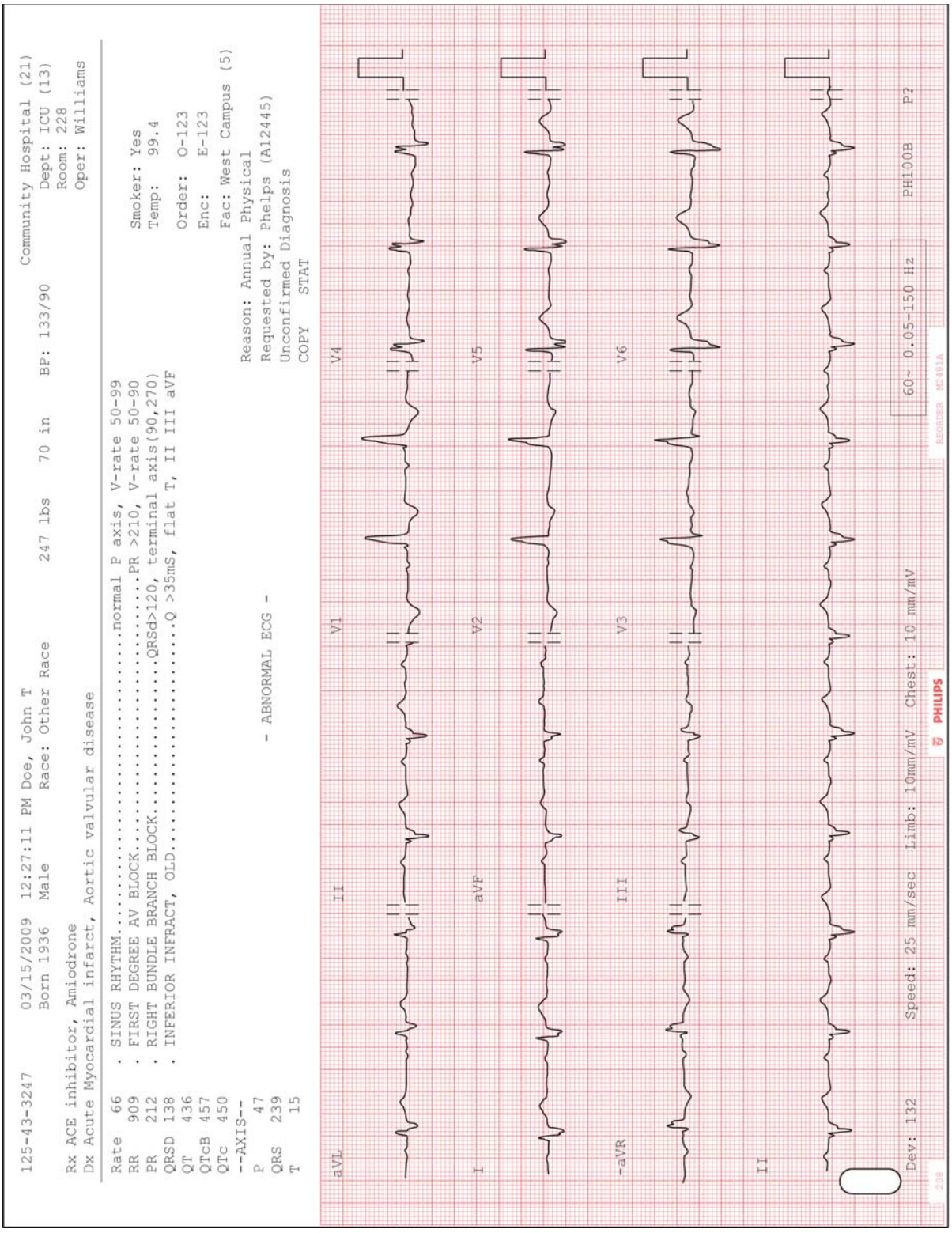


Figure 6-28 6x2 Report with Cabrera Leads

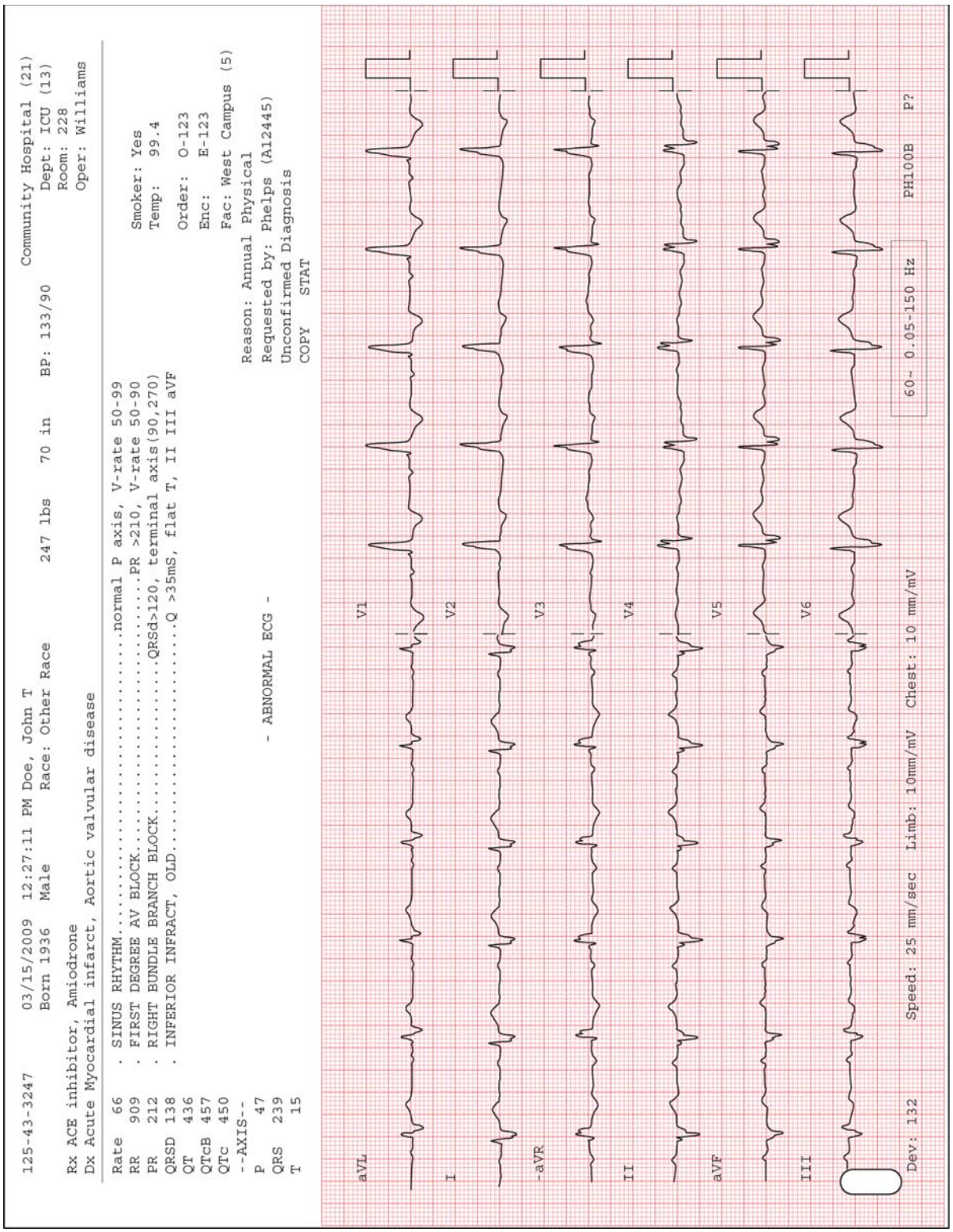


Figure 6-29 12x1 Report with Cabrera Leads (page one)

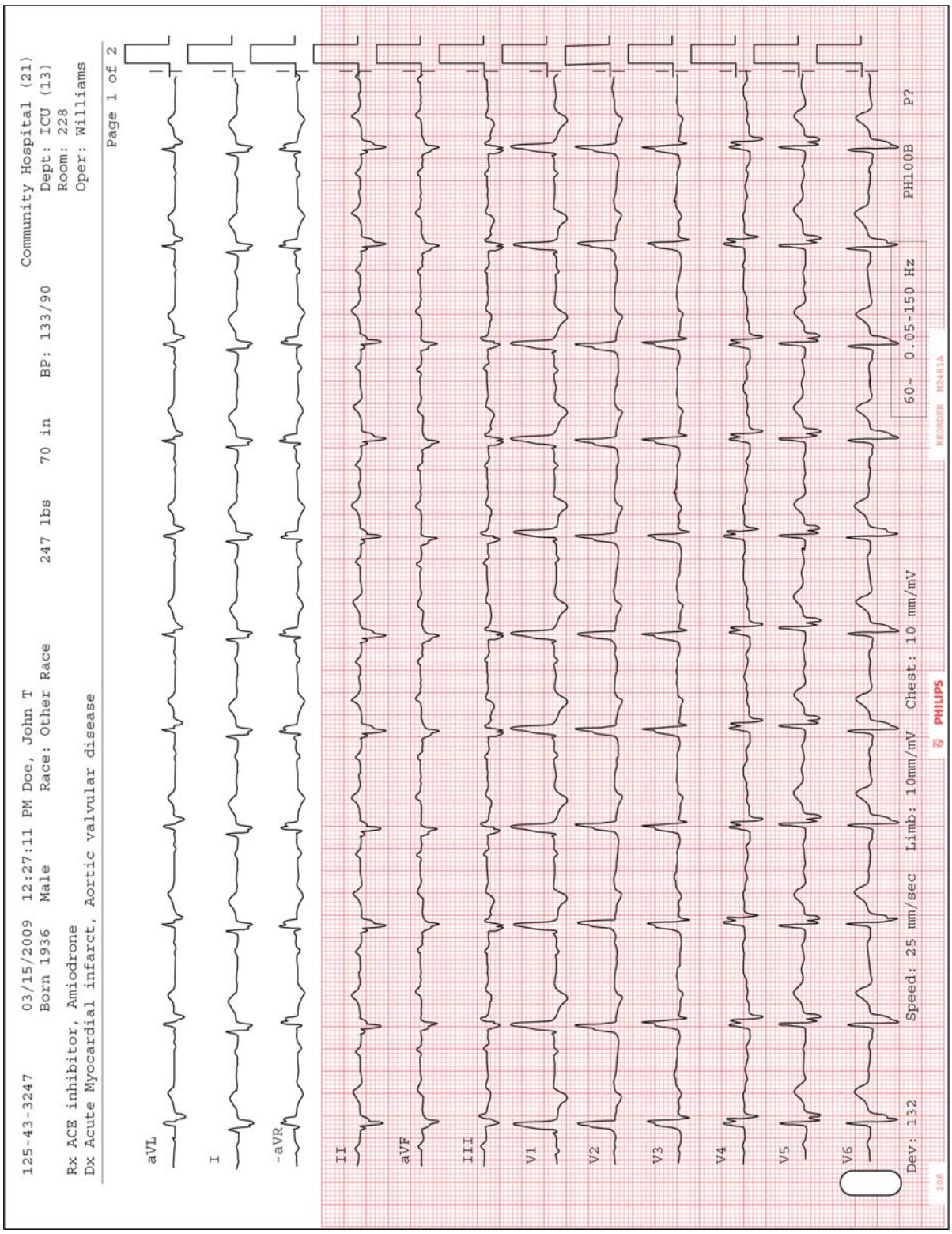
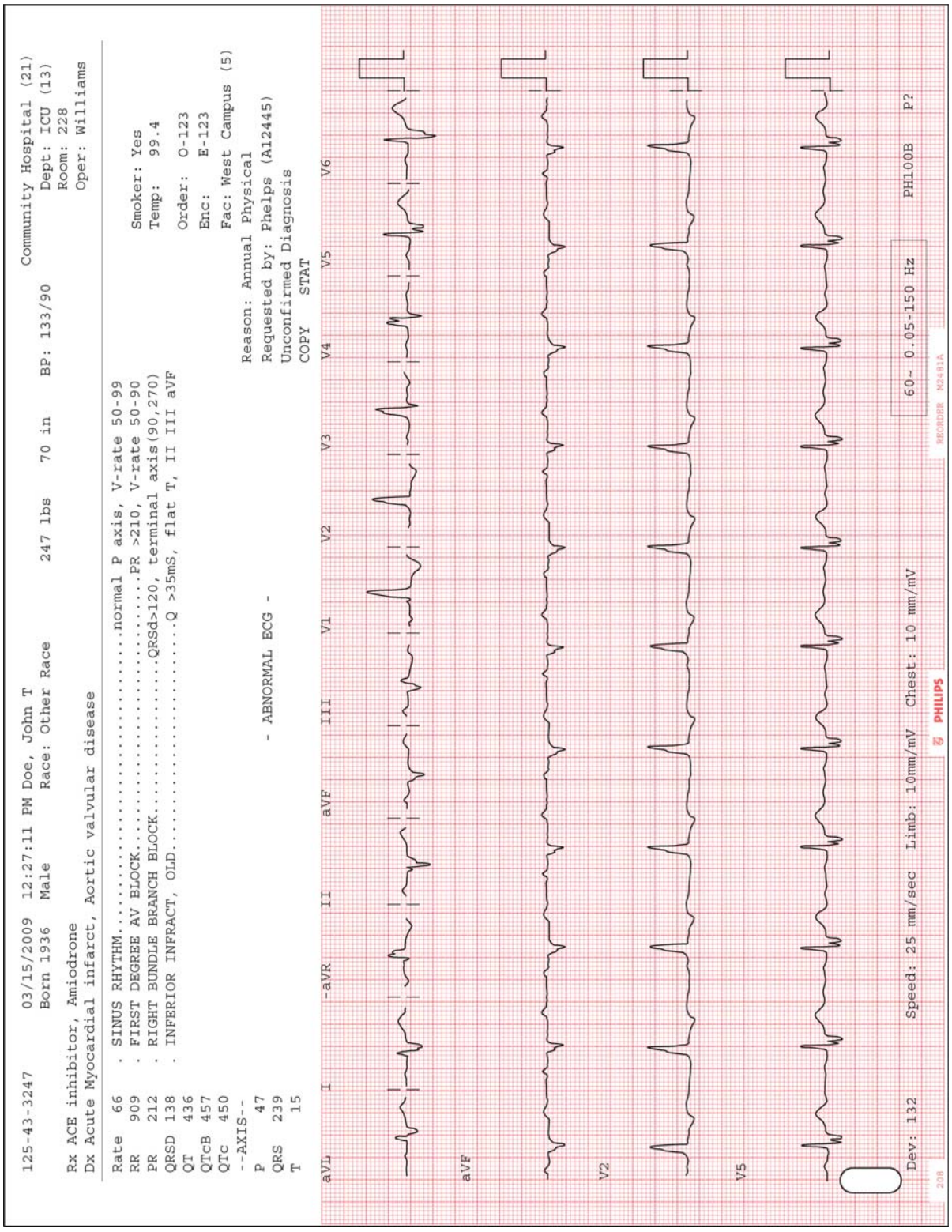


Figure 6-30 12x1 Report with Cabrera Leads (page two)

Figure 6-31 Panoramic (Pan-12) Report



NOTE Leads are displayed in Cabrera sequence on the Panoramic (Pan-12) Report regardless of the selected lead standard on the acquisition device.

15 and 16-Lead ECG Report Examples

The following section provides examples of 15 and 16-lead format ECG report formats that may be available on the acquisition device including:

- Pediatric 3x5, 1R report with Standard Leads
- Pediatric 3x5, 3R report with Standard Leads
- Balanced 4x4, 1R report with Standard Leads
- Adult Posterior 3x5 3R report with Standard Leads

Figure 6-32 Pediatric 3x5, 1R Report with Standard Leads

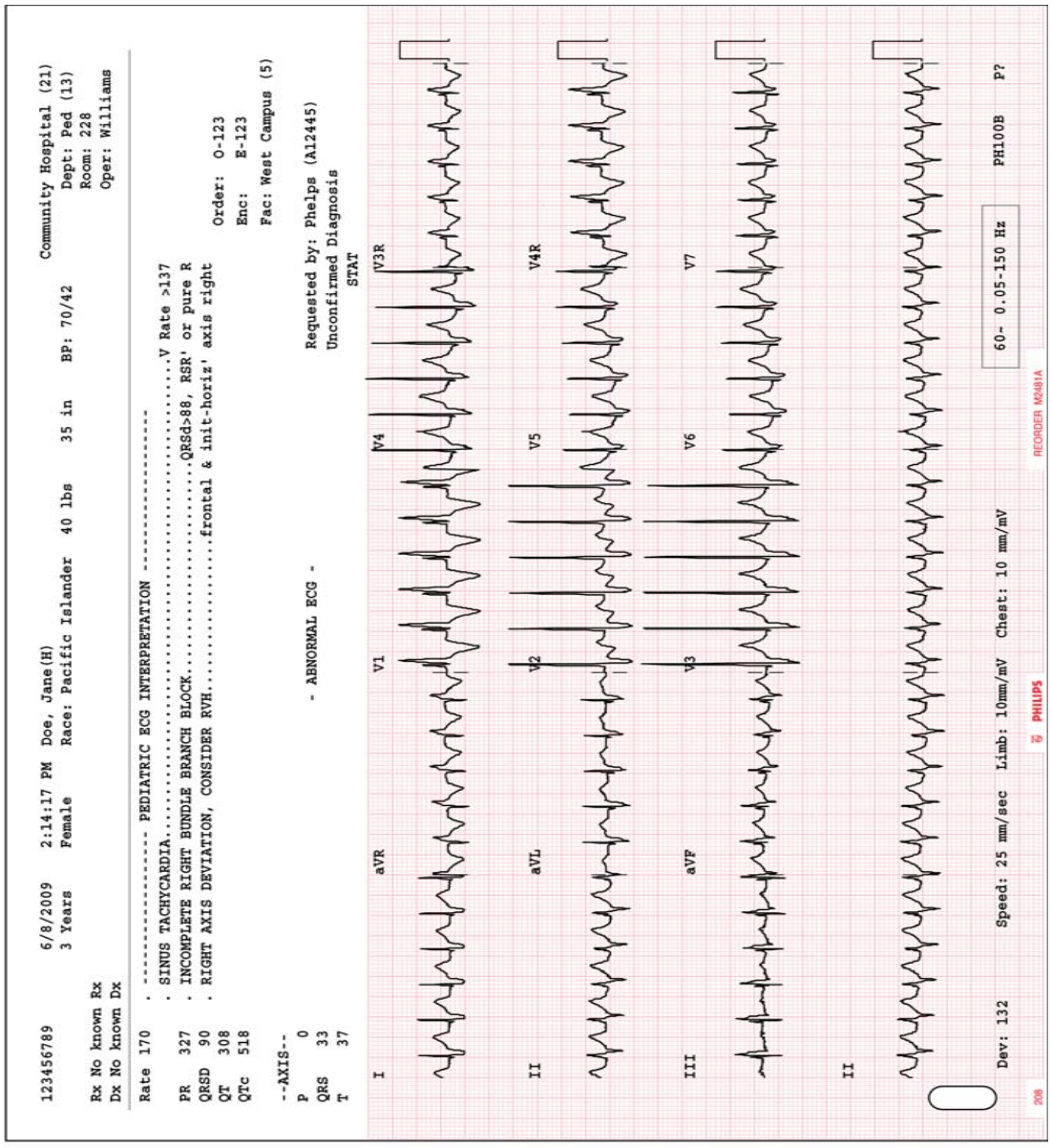


Figure 6-33 Pediatric 3x5, 3R Report with Standard Leads

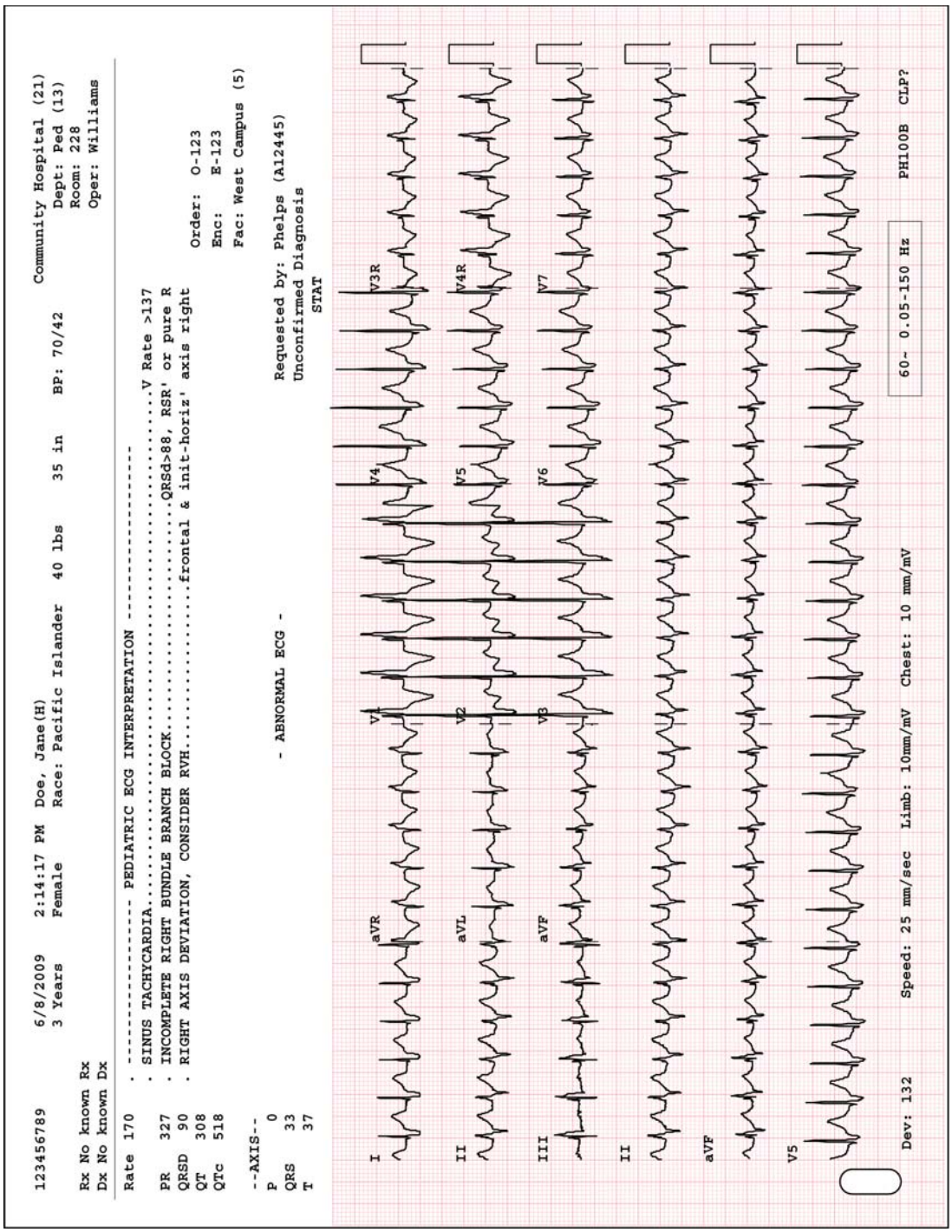


Figure 6-34 Adult Balanced 4x4, 1R with Standard Leads

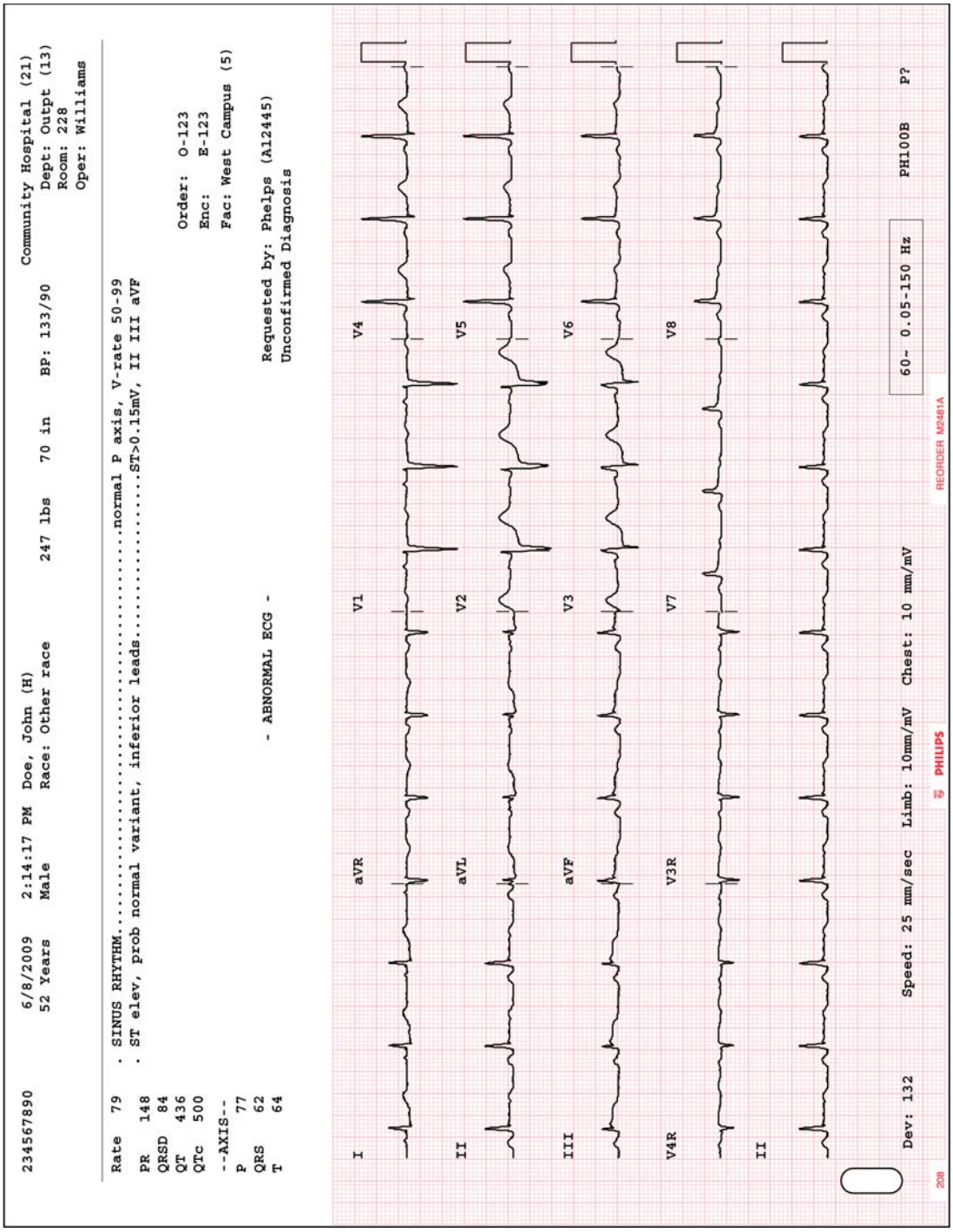
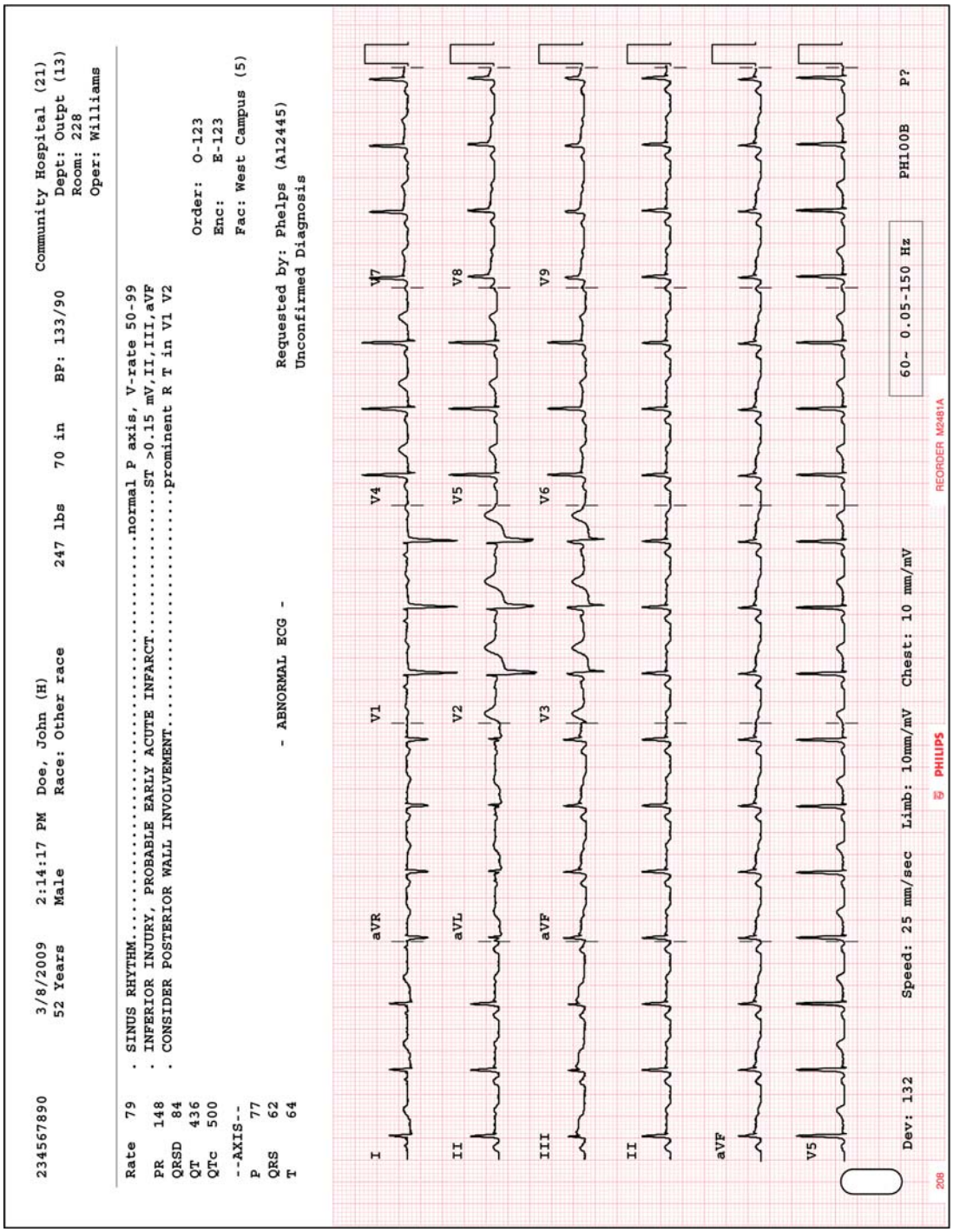


Figure 6-35 Adult Posterior 3x5, 3R Report with Standard Leads



Extended Measurements Report

The Extended Measurements report summarizes the output of the Philips DXL ECG Algorithm. The report includes the morphology characteristics for the individual leads, and the rhythm characteristics for the rhythm groups. The algorithm uses this measurement information to generate interpretive statements. The Extended Measurements report is especially useful if you want to examine the measurements used to generate an interpretation.

Morphology Analysis

Figure 6-36 Morphology Analysis page of the Extended Measurements Report

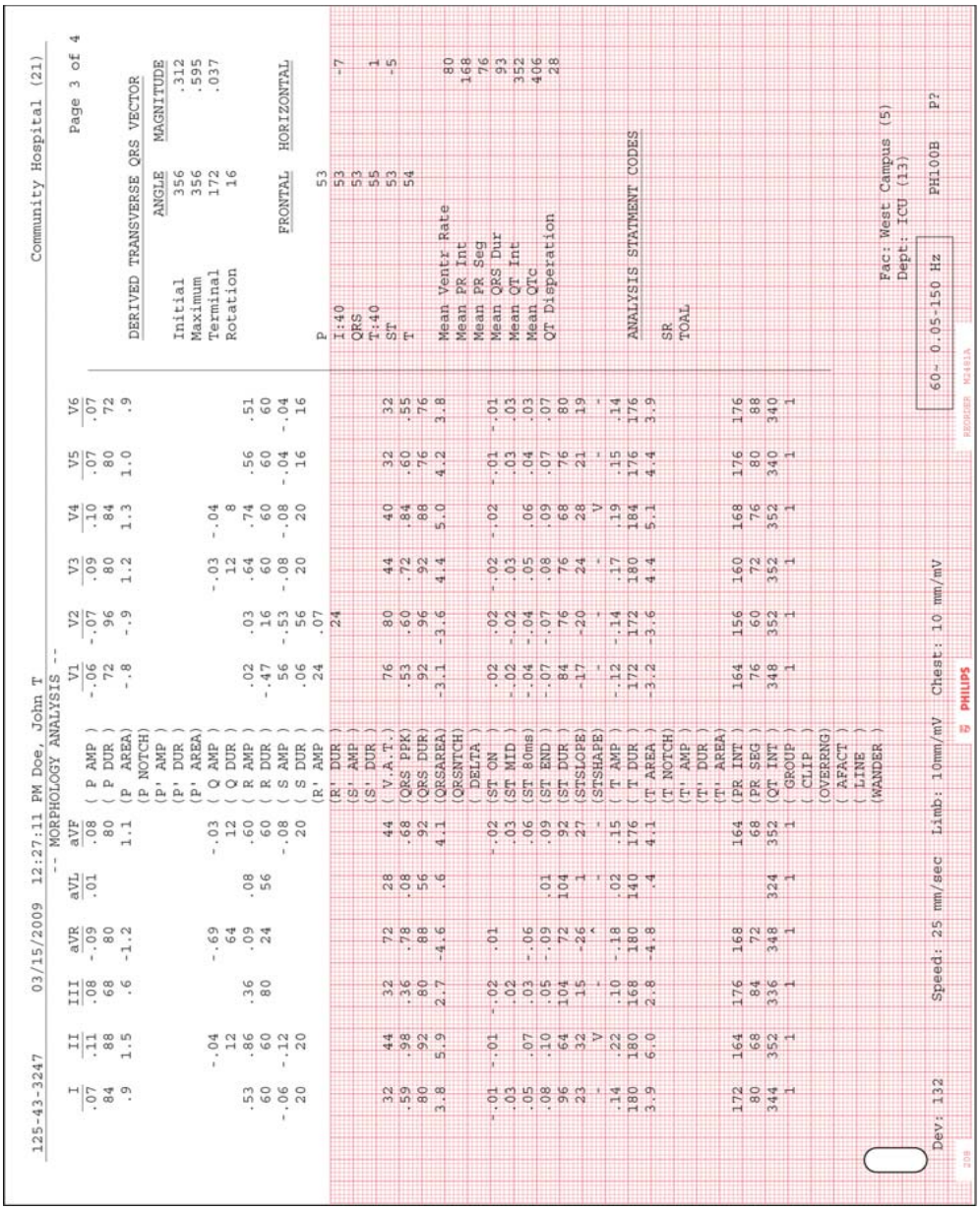


Table 6-11 on page 6-32 through Table 6-14 on page 6-37 define the parameters in the order that they appear on the Morphology Analysis page of the Extended Measurements report.

Morphology Lead Measurements

The parameter measurements are shown in the illustration below. Table 6-11 on page 6-32 describes every representative measurement in each lead.

Figure 6-37 ECG Morphology Measurements

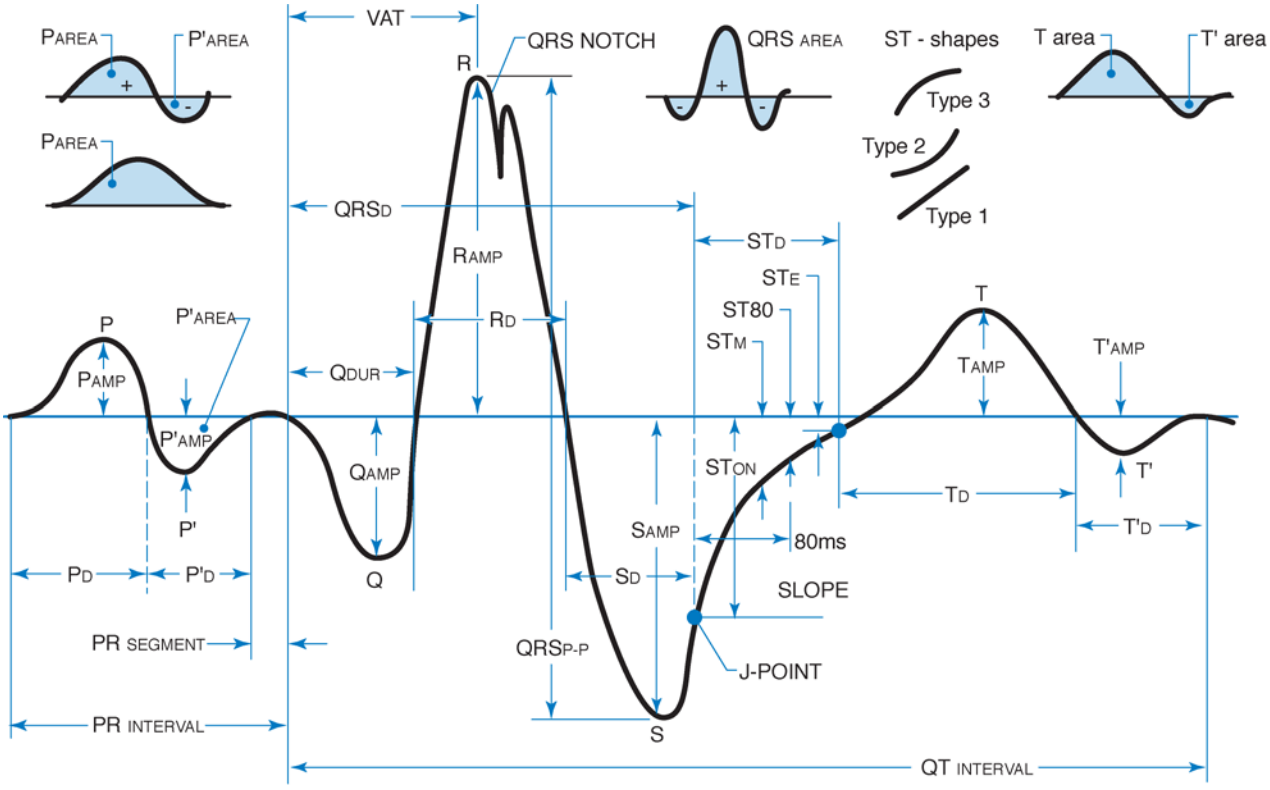


Table 6-11 Morphology Lead Measurements

Parameter	Units or Value	Description
P AMP	millivolts	P wave amplitude
P DUR	milliseconds	P wave duration
P AREA	Ashman units[a] (40 ms x 0.1 mV)	P wave area for monophasic P waves or the area of the initial portion of a biphasic P wave

[a] An Ashman unit is the area of 1 square millimeter at normal speed (25 mm/sec) and normal sensitivity (10 mm/mV). An Ashman unit equals 40 ms x 0.1 mV.

[b] All isoelectric segments including I and K waves are excluded from the Q, R, S, R' and S' waves.

Table 6-11 Morphology Lead Measurements *(continued)*

Parameter	Units or Value	Description
P NOTCH	Yes or No	Indicates the presence or absence of a notch in the P wave
P′ AMP	millivolts	P′ wave amplitude
P′ DUR	milliseconds	P′ wave duration
P′ AREA	Ashman units[a] (40 ms x 0.1 mV)	Area of the terminal portion of a biphasic P wave
Q AMP	millivolts	Q wave amplitude
Q DUR[b]	milliseconds	Q wave duration
R AMP	millivolts	R wave amplitude
R DUR[b]	milliseconds	R wave duration
S AMP	millivolts	S wave amplitude
S DUR[b]	milliseconds	S wave duration
R′ AMP	millivolts	R′ wave amplitude
R′ DUR[b]	milliseconds	R′ wave duration
S′ AMP	millivolts	S′ wave amplitude
S′ DUR[b]	milliseconds	S′ wave duration
V.A.T.	milliseconds	Ventricular Activation Time is the interval from the onset of the QRS complex to the latest positive peak in the complex, or the latest substantial notch on the latest peak (whichever is later)
QRS PPK	millivolts	Peak-to-peak QRS complex amplitude
QRS DUR	milliseconds	QRS complex duration, measured from its onset to the ST segment onset (J point)
QRSAREA	Ashman units[a] (40 ms x 0.1 mV)	The area of the QRS complex

[a] An Ashman unit is the area of 1 square millimeter at normal speed (25 mm/sec) and normal sensitivity (10 mm/mV). An Ashman unit equals 40 ms x 0.1 mV.

[b] All isoelectric segments including I and K waves are excluded from the Q, R, S, R’ and S’ waves.

Table 6-11 Morphology Lead Measurements *(continued)*

Parameter	Units or Value	Description
QRSNTCH	**+** or **-**	■ Indicates a notch in the QRS complex ■ + indicates a notch or slur in the R or R′ wave ■ - indicates a notch or slur in the Q, S, or S′ wave
DELTA	Yes or No	Indicates the presence or absence of pronounced delta waves preceding QRS complexes
ST ON	millivolts	Elevation or depression at the onset (J point) of the ST segment
ST MID	millivolts	Elevation or depression at the midpoint of the ST segment
ST 80ms	millivolts	Elevation or depression of the ST segment 80 ms after the end of the QRS complex (J point)
ST END	millivolts	Elevation or depression at the end of the ST segment
ST DUR	milliseconds	ST segment duration
STSLOPE	degrees	ST segment slope. Slope is measured in degrees for 25 mm/sec, 1mV/cm scaling, and can range from -90 to +90 degrees.
STSHAPE	**-**, **V**, or **?**	The ST segment shape: **-** = Straight **V** = Concave upward **?** = Concave downward
T AMP	millivolts	T wave amplitude
T DUR	milliseconds	T wave duration
T AREA	Ashman units[a] (40 ms x 0.1 mV)	T wave area for monophasic T waves or the area of the initial portion of a biphasic T wave
T NOTCH	Yes or No	Indicates the presence or absence of a notch in the T wave
T′ AMP	millivolts	T′ wave amplitude

[a] An Ashman unit is the area of 1 square millimeter at normal speed (25 mm/sec) and normal sensitivity (10 mm/mV). An Ashman unit equals 40 ms x 0.1 mV.

[b] All isoelectric segments including I and K waves are excluded from the Q, R, S, R’ and S’ waves.

Table 6-11 Morphology Lead Measurements *(continued)*

Parameter	Units or Value	Description
T' DUR	milliseconds	T' wave duration
T' AREA	Ashman units[a] (40 ms x 0.1 mV)	Area of the terminal portion of a biphasic T wave
PR INT	milliseconds	Interval from the onset of the P wave to the onset of the QRS complex
PR SEG	milliseconds	Interval from the end of the P wave to the onset of the QRS complex
QT INT	milliseconds	Interval from the onset of the QRS complex to the end of the T wave
GROUP	1 (or 2-5)	Indicates the rhythm group used to derive the representative beat waveform, from which measurements are calculated. Will be Group 1 unless no Group 1 beats were detected during the analysis interval for this lead.
CLIP	Y = Yes	Indicates clipping of QRS complexes
OVERRNG	Y = Yes	Indicates that the ECG signal is outside the measurement parameters of the instrument
AFACT	MOD = Moderate artifact MARK = Significant artifact SEV = Severe artifact	Artifact (most likely muscle tremor) is present when more than 16 up-and-down strokes exceeding 1mm in amplitude are detected within 1 second
LINE	MOD = Moderate noise MARK = Significant noise SEV = Severe noise	AC (power line) noise is present
WANDER	MOD = Moderate wander MARK = Significant wander SEV = Severe wander	A steady baseline wander exceeding 10mm/sec is present

[a] An Ashman unit is the area of 1 square millimeter at normal speed (25 mm/sec) and normal sensitivity (10 mm/mV). An Ashman unit equals 40 ms x 0.1 mV.

[b] All isoelectric segments including I and K waves are excluded from the Q, R, S, R' and S' waves.

Derived Transverse QRS Vector

The derived transverse QRS vector is a three-dimensional signal made up of X, Y, and Z (Frank leads) signals projected onto a transverse plane. The values are derived by estimating the X, Y, and Z signals from a standard 12-lead. Table 6-12 on page 6-36 lists the derived transverse QRS vector parameters.

Table 6-12 Derived QRS Vector Parameters

Parameter	Units or Value	Description
Initial	■ vector angle in degrees ■ vector magnitude in mV	The vector for the initial (first 40 ms) transverse QRS signal
Maximum	■ vector angle in degrees ■ vector magnitude in mV	The maximum transverse QRS vector
Terminal	■ vector angle in degrees ■ vector magnitude in mV	The vector from the terminal (last 40 ms) or last part of the transverse QRS signal
Rotation	100 to -100	■ The direction of the vector rotation over the entire QRS complex – A positive rotation value indicates a clockwise vector rotation – A negative rotation value indicates a counterclockwise vector rotation ■ A larger magnitude indicates a higher confidence in the rotation estimate

Frontal/Horizontal Plane Axis Parameters

Table 6-13 on page 6-36 lists frontal and horizontal plane axis parameters.

Table 6-13 Frontal/Horizontal Plane Axis Parameters

Parameter	Units or Value	Description
P	degrees or ind (indeterminate)	Mean P wave axis
I:40	degrees or ind (indeterminate)	Initial 40 ms QRS complex axis
QRS	degrees or ind (indeterminate)	Mean QRS complex axis

Table 6-13 Frontal/Horizontal Plane Axis Parameters *(continued)*

Parameter	Units or Value	Description
T:40	degrees or ind (indeterminate)	Terminal 40 ms QRS complex axis
ST	degrees or ind (indeterminate)	Mean ST wave axis
T	degrees or ind (indeterminate)	Mean T wave axis

Global Measurements

Table 6-14 on page 6-37 lists the global measurements representative of the entire ECG.

Table 6-14 Global Measurement Parameters

Parameter	Units or Value	Description
Mean Ventr Rate	beats per minute	Representative ventricular rate for the entire ECG
Mean PR Int	milliseconds	Representative PR interval for the entire ECG
Mean PR Seg	milliseconds	Representative PR segment for the entire ECG
Mean QRS Dur	milliseconds	Representative QRS duration for the entire ECG
Mean QT Int	milliseconds	Representative QT interval for the entire ECG
Mean QTc	milliseconds	Representative QT interval adjusted for heart rate
QT Dispersion	milliseconds	Difference between the longest and shortest QT interval for the entire ECG

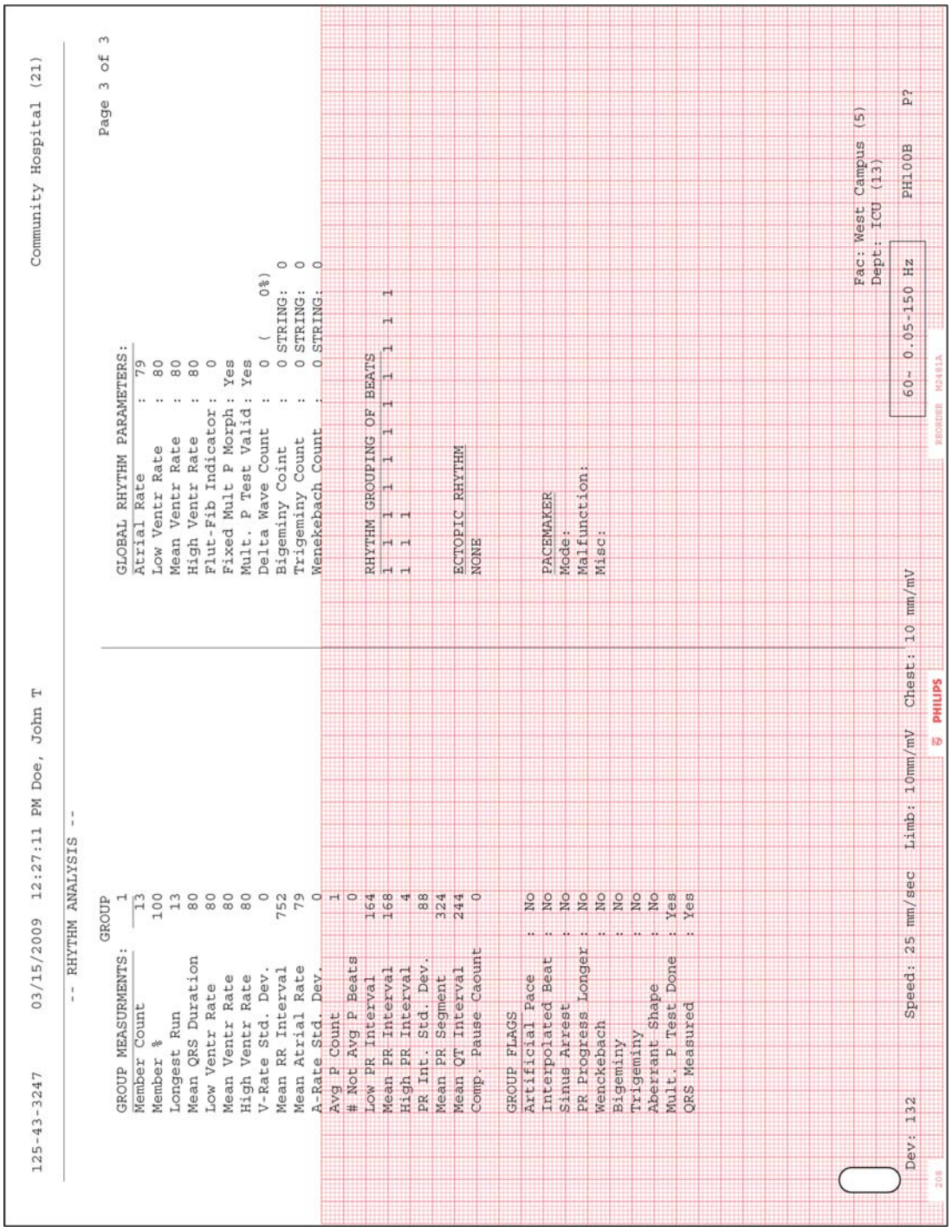
Analysis Statement Codes

These statement codes are the abbreviated criteria codes for the interpretive statements. These statement codes are used when editing reports with a Philips TraceMasterVue ECG Management System.

For lists of codes and statements, see “Interpretive Statements, by Category” on page B-1.

Rhythm Analysis

Figure 6-38 Rhythm Analysis Section of the Extended Measurements Report



The following parameters are given for each rhythm group detected by the cardiograph during the analysis interval.

Group Measurements

The group measurements are listed in the table below.

Table 6-15 Group Measurements

Parameter	Units or Value	Description
Member Count	not applicable	Number of beats in the rhythm group
Member %	percentage	Percentage of the total number of beats represented by the rhythm group
Longest Run	not applicable	Longest contiguous run of beats in the rhythm group
Mean QRS Duration	milliseconds	Average QRS duration in the rhythm group
Low Ventr Rate	beats per minute	Lowest ventricular rate in the rhythm group
Mean Ventr Rate	beats per minute	Average ventricular rate in the rhythm group
High Ventr Rate	beats per minute	Highest ventricular rate in the rhythm group
V-Rate Std Dev	same units as the associated measurement	Standard deviation of the ventricular rate in the rhythm group
Mean RR Interval	milliseconds	Average interval between R waves in the rhythm group
Mean Atrial Rate	beats per minute	Average atrial rate in the rhythm group
A-Rate Std Dev	same units as the associated measurement	Standard deviation of the atrial rate in the rhythm group
Avg P Count	not applicable	Average number of P waves per QRS complex in the rhythm group
# Not Avg P Beats	not applicable	Number of QRS complexes in the rhythm group that do not have the average number of P waves per QRS complex

Table 6-15 Group Measurements *(continued)*

Parameter	Units or Value	Description
Low PR Interval	milliseconds	Shortest PR interval in the rhythm group
Mean PR Interval	milliseconds	Average PR interval in the rhythm group
High PR Interval	milliseconds	Longest PR interval in the rhythm group
PR Int Std Dev	same units as the associated measurement	Standard deviation of the PR interval in the rhythm group
Mean PR Segment	milliseconds	Average PR segment in the rhythm group
Mean QT Interval	milliseconds	Average QT interval in the rhythm group
Comp. Pause Count	not applicable	Number of beats followed by a compensatory pause in the rhythm group

Group Flags

The parameters in this part of the rhythm analysis indicate the presence or absence of various rhythm-related conditions in the rhythm groups identified.

Table 6-16 Group Flags

Parameter	Units or Value	Description
Atrial Pace	Yes or No	Beats in the rhythm group are atrial paced
Ventricular Pace	Yes or No	Indicates that beats in the rhythm group are paced. All paced beats are grouped together unless the pacing is a mixture of atrial and ventricular/dual chamber paced beats. In this case, the atrial paced beats fall together in a separate group.
Interpolated Beat	Yes or No	Indicates the rhythm group contains only interpolated beats
Sinus Arrest	Yes or No	Indicates a prolonged R-to-R interval. Set for the sinus arrest resumption group.

Table 6-16 Group Flags *(continued)*

Parameter	Units or Value	Description
PR Progress Longer	Yes or No	Indicates the PR interval is getting progressively longer in the rhythm group
Wenckebach	Yes or No	Indicates presence of the Wenckebach phenomenon in the rhythm group
Bigeminy	Yes or No	Indicates presence of a bigeminy rhythm. Set for the group consisting of ectopic beats.
Trigeminy	Yes or No	Indicates presence of a trigeminy rhythm. Set for the group consisting of ectopic beats.
Aberrant Shape	Yes or No	Indicates that beats in the rhythm group are in the minority, and are either wider or of a different polarity from other beats in the same lead(s)
Multifocal	Yes or No	Indicates that beats in the rhythm group have different foci or origin
Mult. P Test Done	Yes or No	Indicates that beats in the rhythm group were tested for multiple P waves
QRS Measured	Yes or No	Indicates that QRS-related parameters were measured in the rhythm group

Global Rhythm Parameters

The following parameters provide global information for beats in the ECG.

Table 6-17 Global Rhythm Parameters

Parameter	Units or Value	Description
Atrial Rate	beats per minute	The representative atrial rate for the analysis interval. This is not a simple arithmetic average.
Low Ventr Rate	beats per minute	The lowest ventricular rate during the analysis interval
Mean Ventr Rate	beats per minute	The average ventricular rate during the analysis interval
High Ventr Rate	beats per minute	The highest ventricular rate during the analysis interval

Table 6-17 Global Rhythm Parameters *(continued)*

Parameter	Units or Value	Description
Flut-Fib Indicator	not applicable	Indicates approximate number of flutter-like or coarse fibrillatory waves per lead
Fixed Mult P Morph	Yes or No	Indicates that all P waves are of consistent morphology
Mult P Test Valid	Yes or No	Indicates that the tests performed to detect multiple P waves produced consistent results
Paced Beats Measrd	Yes or No	Indicates that a dual or ventricular paced beat group was used for the representative beat (no non-paced or atrial paced beats were measured)
Delta Wave Count	not applicable	Number of QRS complexes with pronounced delta waves
Delta Wave %	percentage	Percent of total beats with pronounced delta waves
Bigeminy Count	not applicable	Total number of beats in a bigeminy pattern, whether or not they are contiguous
Bigeminy String	not applicable	Total number of beats in the longest continuous bigeminy pattern
Trigeminy Count	not applicable	Total number of beats in a trigeminy pattern, whether or not they are contiguous
Trigeminy String	not applicable	Total number of beats in the longest continuous trigeminy pattern
Wenckebach Count	not applicable	Total number of Wenckebach cycles. A Wenckebach cycle is a series of beats whose PR intervals grow progressively longer, culminating in an unusually long RR interval (a dropped beat).
Wenckebach String	not applicable	The number of beats preceding the dropped beat

Rhythm Grouping of Beats

The Rhythm Grouping of Beats is a number sequence that shows the rhythm group number for each beat as determined by the rhythm analysis portion of the algorithm.

Table 6-18 Rhythm Grouping of Beats

Number	Description
1, 2, 3, 4, or 5	Rhythm group number
0	Beat unclassifiable by program

Ectopic Rhythm

The parameters in this section indicate the type of ectopic beats detected including their underlying rhythm.

NOTE If more than one ectopic rhythm code is generated for the report, only the highest severity rhythm code is printed in this section.

Table 6-19 Ectopic Rhythm Parameters

Parameter	Description
NONE	No ectopic beats detected
APC	Atrial Premature Complex
JPC	Junctional Premature Complex
APCs	Atrial Premature Complexes
JPCs	Junctional Premature Complexes
ABIG	Supraventricular Bigeminy
VPC	Ventricular Premature Complex
VPCs	Ventricular Premature Complexes
APC & VPC	Ectopic beats of Supraventricular and Ventricular origin
VTRIG	Ventricular Trigeminy
VBIG	Ventricular Bigeminy
MFPVCs	Multiform Premature Ventricular Complexes
PAIR	One or more pairs of Ventricular Complexes
MFPAIR	One or more pairs with Multiform Ventricular Complexes (not necessarily in the same pair)
RUN	Runs of three or more Ventricular Complexes

Table 6-19 Ectopic Rhythm Parameters *(continued)*

Parameter	Description
MFRUN	Runs with Multiform Ventricular Complexes (not necessarily in the same run)

Pacemaker

The parameters in this section indicate the type of paced rhythm detected. There are three types of pacemaker information included: Mode, Malfunction, and Miscellaneous.

The Mode information indicates the type of pacing identified.

Table 6-20 Pacemaker Mode Parameters

Parameter	Description
APACE	Continuous Atrial Paced
VPACE	Continuous Ventricular Paced
ASVPR	Continuous Atrial-Sensed Ventricular Paced (with P-wave tracking)
AVDPR	A-V Dual Paced
MIXPR	Mixed pacing type with inhibition of at least one chamber
IAPACE	Intermittent Atrial Paced
IVPACE	Intermittent Ventricular Paced
IASVRP	Intermittent Atrial-Sensed Ventricular Paced
IAVDPR	Intermittent A-V Dual Paced
IVPACD	Intermittent Ventricular Paced (On Demand)
IAPACD	Intermittent Atrial Paced (On Demand)
IMIXPR	Intermittent Paced Beats with inhibition of at least one chamber detected in the paced beats
UNKPR	Unrecognized Pacemaker Rhythm where pacer spikes or artifact are present

The Malfunction information identifies any detected pacing system malfunctions.

Table 6-21 Pacing Malfunction Parameters

Parameter	Description
PACENC	Pacer Non-Capture
PACENS	Pacer Non-Sense
PACNCNS	Pacer Non-Capture and Non-Sense
PACERA	■ Runaway Pacer (asynchronous pacing, for example fixed rate pacing with no sensing) ■ A pacemaker magnet may be present

The Miscellaneous information section contains pacing information not included in any other section.

Table 6-22 Miscellaneous Pacing Information

Parameter	Description
PACART	Miscellaneous pacing artifact was detected
MAGNET	The ECG was specified as being acquired with a pacemaker magnet or interrogator in place

7

New Features in the DXL ECG Algorithm, vPH110C

This chapter describes the updates made to the DXL ECG Algorithm, version PH110C.

Selecting the Algorithm Version

The algorithm selection option on the cardiograph now also includes the PH110C algorithm. For details, see the cardiograph Instructions for Use.

Changes to Global Measurements of QT Interval

Measurement of the QT interval presents several challenges. For extensive information on these issues, see pages 1-25 through 1-29.

The PH110C algorithm uses a criterion of T-wave amplitude greater than 150 uV to guarantee “reliability” for a particular lead.

QT Correction for Heart Rate

Increases in heart rate shorten the QT interval and decreases in heart rate lengthen the interval. The standard heart rate used to establish normal limits for QT interval is 60 beats/min. Many formulae have been used to account for, or “correct”, the shortening or lengthening[1].

The PH110C algorithm supports the following correction methods:

- The square and cube root relations, *Bazett* and *Fridericia*; sometimes called power relations due to the exponents involved.
- The *Hodges* and *Framingham* formulae, which are part of a different approach using linear relationships.

The formula for each method is provided on page 7-2.

1. Molnar et al. “Evaluation of Five QT Correction Formulas Using a Software-Assisted Method of Continuous QT Measurement from 24-Hour Holter Recordings.” *American Journal of Cardiology* (1996); 78:920-26.

Ventricular conduction defects (VCD) prolong the QRS duration and widen the QT interval due to that prolonged depolarization. To account for the added secondary prolongation of the QT interval when correction for heart rate, QT correction formulae were developed for VCD[2] and recommended for use[3]. Use of these formulae for wide QRS will prevent the false appearance of long QT intervals just due to the secondary effect of wide QRS. The formula for wide QRS QT correction is:

$$QTc(ms) = QT(ms) - 155\left(\frac{RR(ms)}{1000} - 1.0\right) - 0.93(QRSd(ms) - 139) - k$$

where k = 34 for women and 22 for men.

When QT correction for wide QRS is enabled, a QRS duration of 120 ms or greater will change the QT correction formula from the configured primary QT correction formula to the formula for wide QRS.

None of these formulae is particularly good at heart rate correction of QT when the heart rate is very high or very low. Individual patient variability and the effect of medications causes variation even near a heart rate of 60.

The DXL Algorithm calculates all four QT rate corrections. The Philips acquisition device can be configured to output any of Bazett, Fridericia, Hodges, or Framingham values. The configured correction formula is used during waveform analysis to trigger statements describing abnormally long or short corrected QT intervals.

Bazett

The Bazett correction[4] is favored in the United States and is calculated as the following *square root* relation:

$$QTc = \frac{QT}{\sqrt{RR}}$$

Fridericia

The Fridericia correction[5] is more commonly used in Europe and can be calculated as the following *cube root* relation:

$$QTc = \frac{QT}{\sqrt[3]{RR}}$$

2. Rautaharju, P.M, Zhang, Z.M., Prineas, R., Heiss, G. “Assessment of prolonged QT and JT intervals in ventricular conduction defects.” American Journal of Cardiology, 2004; 93:1017-1021.

3. Rautaharju, P., Surawicz, B., Gettes, L. “AHA/ACCF/HRS recommendations for the standardization interpretation of the electrocardiogram: part IV: The ST segment T, U waves, and the QT interval.” American College of Cardiology, 2009; 53:982-991,

4. Bazett, H.C. “An Analysis Of The Time-Relations Of Electrocardiograms.” *Heart* (1920); 7:353-70.

5. L. S. Fridericia. “Die Systolendauer im Elektrokardiogramm bei normalen Menschen und bei Herzkranken.” *Acta Medica Scandinavica*, Stockholm, 1920, 57: 469-486.

Hodges

The Hodges corrected QT interval[6] is calculated as:

$$QTc = QT + 1.75(HR - 60)$$

Framingham

The Framingham corrected QT interval[7] is calculated as:

$$QTc = QT + 0.154(1 - RR)$$

Paced Rhythm Analysis

The PH110C algorithm handles the case where both right and left ventricles have pacer leads with different impulse times, which is common in Cardiac Resynchronization therapy (CRT). The DXL algorithm recognizes closely spaced pace spikes as biventricular pacing and reports it as part of the paced rhythm analysis.

For additional information on how the DXL Algorithm analyzes pacer information, see Chapters 1 and 2 of this guide.

Culprit Artery Categories

For additional information, see “Culprit Coronary Artery Concept” on page 3-8.

The algorithm supports a new category: Proximal left anterior descending (proxLAD). The involved artery table is updated as follows:

Table 7-1 Involved Artery with Abbreviation

Involved artery	Abbreviation
Left Circumflex	LCx
Right Coronary	RCA
Left Anterior Descending	LAD
Proximal Left Anterior Descending	proxLAD
High Left Main/multi vessel disease	LM/MVD

6. Hodges M, Salerno Q, Erlien D: Bazett’s QT correction reviewed. Evidence that a linear QT correction for heart rate is better. *JACC* 1983;1:694.

7. Sagie, Larson, Goldberg, Bengtson, Levy: An improved method for adjusting the QT interval for heart rate (the Framingham Heart Study). *Am J Cardiol* 1992;70:797-80.

Repolarization Abnormalities and Myocardial Ischemia

The findings related to repolarization abnormalities are updated. The algorithm adds the finding of deeply inverted T-waves in leads V2, V3, and V4. This ECG pattern is found in some patients with cerebrovascular accidents (CVA) and more often with severe stenosis of the proximal left anterior descending artery[8].

For additional information, see “Repolarization Abnormalities and Myocardial Ischemia” on page 3-18.

Changes to Critical Value Statements

The following changes apply to the critical value statements generated by the algorithm.

Myocardial Infarction Critical Value Statements

In addition to the statements listed in Table 5-1 on page 5-1, the following new statements can also trigger the Acute MI warning on the ECG report.

Table 7-2 New Acute Myocardial Infarct Critical Value Statements

Statement Code	Interpretive Statement
AMIAPD	Anterior infarct, acute (proxLAD).....ST >0.2 mV, V2-V5
LBBMIP	Probable acute infarct and LBBB .....concordant ST

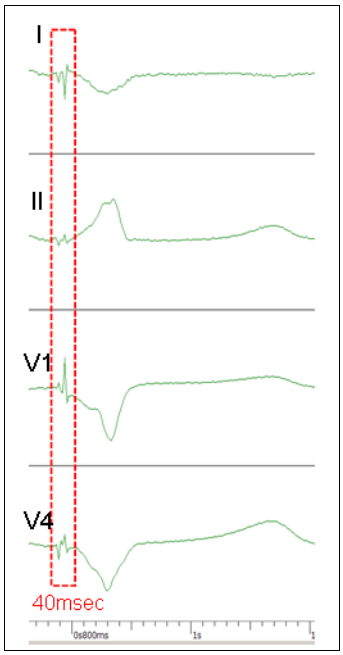
Complete Heart Block Critical Value Statements

Table 5-3 on page 5-4 has changed.

The *3AVBFF* entry for *A-flutter/fibrillation w/ complete AV block* has been removed because atrial fibrillation or atrial flutter in combination with complete heart block is very rare.

8. This is a recommendation in: Wagner GS, Macfarlane P, et al. “Standardization and Interpretation of the Electrocardiogram, Part VI Acute Ischemia/Infarction.” *Circulation* 2009; 119:10. Available online at: http://circ.ahajournals.org.

Biventricular Pacemaker Detection



An increasing number of heart failure patients are receiving Cardiac Resynchronization Therapy (CRT), which uses pacing of both left and right ventricles to maximize cardiac output.

Nonsynchronous biventricular (biV) pacemaker pulses are closely spaced in time, and provide challenges for automated diagnostic ECG algorithms, which need to detect pulses from both ventricular chambers. A special pacemaker pulse detection algorithm is needed to recognize biV pulses, as undetected and unresolved pulses can have a detrimental impact on the diagnostic ECG algorithm's rhythm and morphology interpretations.

The projection of two biventricular pulses on a single lead often looks like one pulse due to the interval between pulses. The interval between two pulses can be as small as 5 ms, which means that the discharging wave of the second pulse overlaps the recharging wave of the first pulse. In addition, the sum of the overlapped vectors will show different characteristics. All of these factors make the detection of biventricular pacing difficult.

However, a component of the DXL algorithm analyzes the pulses identified by the existing non-biV pulse detection algorithm and combines that information with the spatial vector of the ECG signal to detect closely-spaced biV pulses.

Biventricular Pacing Validation Database

As part of the algorithm testing, we collected 500 sps continuous 12-lead ECG from two sources. The first source was four patients with biV pacemakers from three manufacturers. 10-sec samples were taken from the continuous ECG recording while gradually changing RV-to-LV pacing intervals from 70 ms to -70 ms and recording approximately 30 sec for each interval setting, resulting in 614 individual records. The two biV pulses could be as close as 2 ms apart. The ECG waveforms were frequently corrupted with high-frequency noise from nearby devices, such as the pacemaker programmer. The second source of ECGs was the single-ventricular-chamber pacing set used for general pacemaker detection performance (n = 8070)[9].

For algorithm training, 255 cases of 10-sec duration from the continuous biV-paced ECG recordings were used as positive cases. Negative cases came from 211 patients randomly selected from the second source single-chamber pacemaker database. Similarly, the positive cases for the validation dataset for

9. Helfenbein ED, Lindauer JM, Zhou SH, Gregg RE, Herleikson EC. "A Software-based Pacemaker Pulse Detection and Paced Rhythm Classification Algorithm." *Journal of Electrocardiology* 35:95 Supp, 2002.

algorithm testing had 403 biV-paced ECGs from the first source. Negative cases came from 696 single-chamber paced patients from the second source.

When more than 50% of the ventricular paced beats were found with biV pulses in a 10-sec ECG, the ECG was considered biV paced. This criterion is also used in the product algorithm.

Biventricular Pacing Detection Performance

Using the criteria in the previous section, the sensitivity was 94.3% and the specificity was 99.3% for the validation testing set[10].

Table 7-3 Performance of Training Set

Sensitivity	Specificity	PPV	NPV	Total
95.3%	100%	100%	94.6%	466

Table 7-4 Performance of Validation (test) Set

Sensitivity	Specificity	PPV	NPV	Total
94.3%	99.28%	98.7%	96.8%	1099

MI Size Estimated Using the Selvester QRS Score

The PH110C algorithm now supports estimation of size of myocardial infarct (MI).

AHA/ACC/HRS committees on the standardization of the electrocardiogram have recently recommended calculation and reporting of ECG estimated MI size based on the Selvester QRS scoring system[11]. The Selvester ECG-estimated MI size is a helpful clinical tool for patient risk stratification and patient management decision support. A large sized myocardial infarction is associated with poor patient outcomes in both short and long term follow up after acute MI[12].

10. Chien CS, Helfenbein ED, Gregg RE, Babaeizadeh S, Chang PC, Wo HT, Wang CC, Wen MS. “A software algorithm for detection of biventricular pacemaker pulses in the surface ECG.” *Journal of Electrocardiology* 46:622 Supp, 2013.

11. Wagner GS, Macfarlane P, Wellens H, et.al. AHA/ACCF/HRS Recommendations for the Standardization and Interpretation of the Electrocardiogram: Part VI: Acute Ischemia/Infarction. *Circulation*. 2009;119:e262-e270.

12. Barbagelata A, Di Carli MF, Califf RM, et.al. Electrocardiographic infarct size assessment after thrombolysis: Insights from the Acute Myocardial Infarction STudy ADenosine (AMISTAD) trial. *Am Heart J* 2005;150:659-65.

Selvester's ECG-estimated MI size scoring system has been thoroughly studied by numerous research groups. The scoring system has 50 ECG criteria, where each criterion is associated with points that contribute to MI size. Each criterion is also associated with a specific location in the left ventricle.

The Selvester score has been automated and validated against manual reading by Pope[13] and later by Horáček[14]. The Selvester score has also been validated against autopsy[15], ventriculogram, and most recently, MRI[16, 17].

The ECG database for algorithm validation was the Dalhousie body surface mapping (BSM) superset, which has been described previously[14]. The study population consisted of 377 subjects with clinically established MI and 328 controls with no evidence of infarct, for a total of 705 subjects. The patient selection for the test set started with these 705 subjects. After excluding ECGs with LBBB or very small R waves (R amp. < 20 µV), the test set included a total of 670 ECGs.

The Selvester QRS scoring system for estimating MI size was implemented in the Philips DXL ECG analysis algorithm in two steps.

1 All ECG measurements, by lead, were converted to global measurements, as suggested by Horáček.

2 The 50 ECG criteria enumerated in the scoring summary sheet developed by Selvester and studied by Hindman[18] were implemented.

The automated Selvester score was compared to scores manually coded by two cardiologists who are ECG experts with extensive knowledge of the Selvester scoring system. The scores coded by the cardiologists were based on a high-resolution display of waveforms using a time scale of 100 mm/s and an

13. Pope JE, Wagner NB, Dubow D, et. al. Development and validation of an automated method of the Selvester QRS scoring system for myocardial infarct size. *Am J Cardiol* 1988;61(10):734-738.

14. Horá**č**ek BM, Warren JW, Albano A, et. al. Development of an automated Selvester Scoring System for estimating the size of myocardial infarction from the electrocardiogram. *J Electrocardiol* 2006;39:162-168.

15. Strauss DG, Selvester RH. The QRS complex—a biomarker that "images" the heart: QRS scores to quantify myocardial scar in the presence of normal and abnormal ventricular conduction. *J Electrocardiol* 2009;42:85–96.

16. Engblom H, Hedström E, Heiberg E, et.al. Size and transmural extent of first-time reperfused myocardial infarction assessed by cardiac magnetic resonance can be estimated by 12-lead electrocardiogram. *Am Heart J* 2005;150:920.e1-920.e9.

17. Strauss DG, Selvester RH, Lima JAC, et.al. ECG Quantification of Myocardial Scar in Cardiomyopathy Patients With or Without Conduction Defects: Correlation With Cardiac Magnetic Resonance and Arrhythmogenesis. *Circ Arrhythmia Electrophysiol* 2008;1;327-336.

18. Hindman NB, Schocken DD, Widmann M, et al. Evaluation of a QRS scoring system for estimating myocardial infarct size. V. Specificity and method of application of the complete system. *Am J Cardiol* 1985;55:1485.

amplitude scale of 40 mm/mV, which is four times the standard 12-lead ECG scale. In addition, all leads were time aligned to make it easier to determine the global timing of the earliest onset and the latest offset of each QRS complex.

The manual and automated scores were compared. There was a 94% correlation, with a 2 ms adjustment to the computer interval measurements. The 95% confidence interval (CI) for the correlation coefficient was 93% – 95%.

ST Elevation Confounders for Acute MI

STE is generally considered to reveal the occurrence of acute MI. However, acute MI is not the only cause of STE. Other conditions, such as LBBB, RBBB, and LVH, also cause STE. Being able to distinguish the STE of acute MI from these confounders is a critical but difficult task for ECG diagnosis.

Figure 7-1 Positive acute MI with RBBB

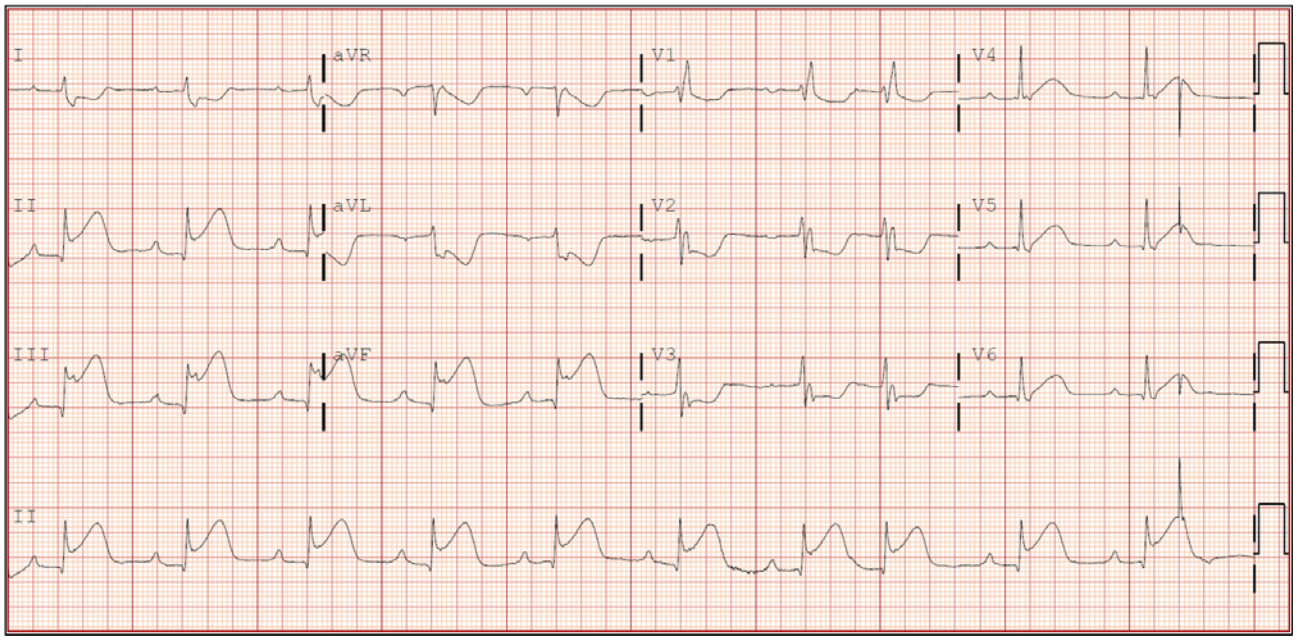


Figure 7-2 Negative acute MI with RBBB

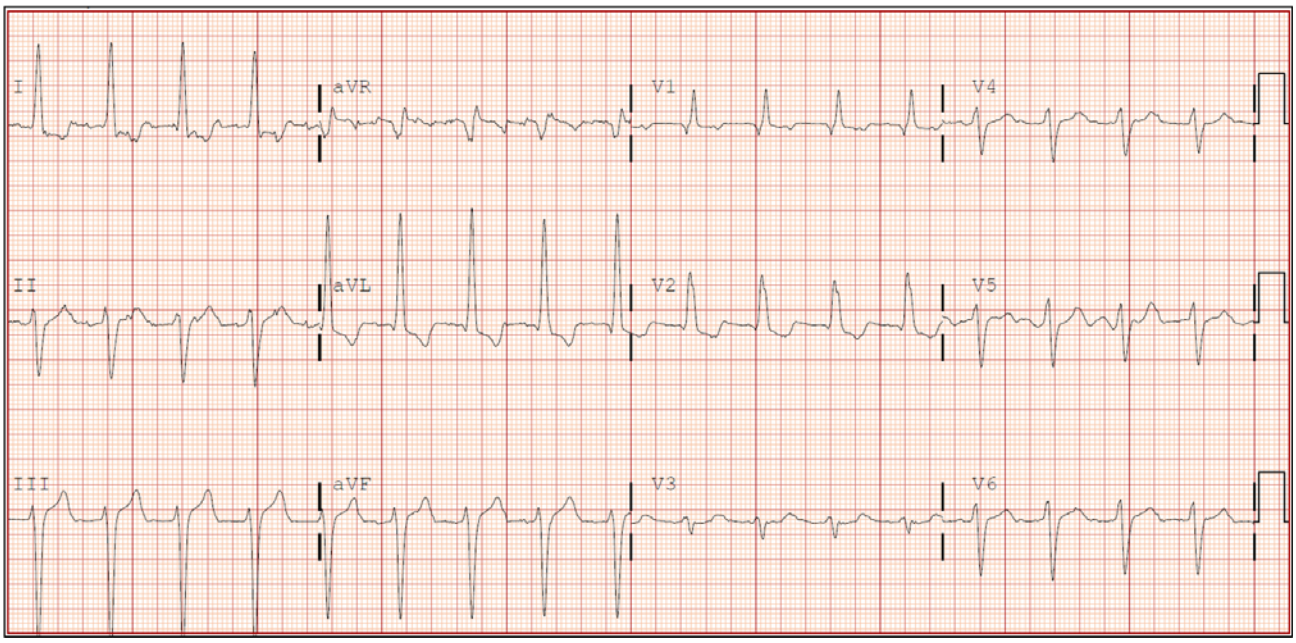


Figure 7-3 Positive acute MI with LBBB

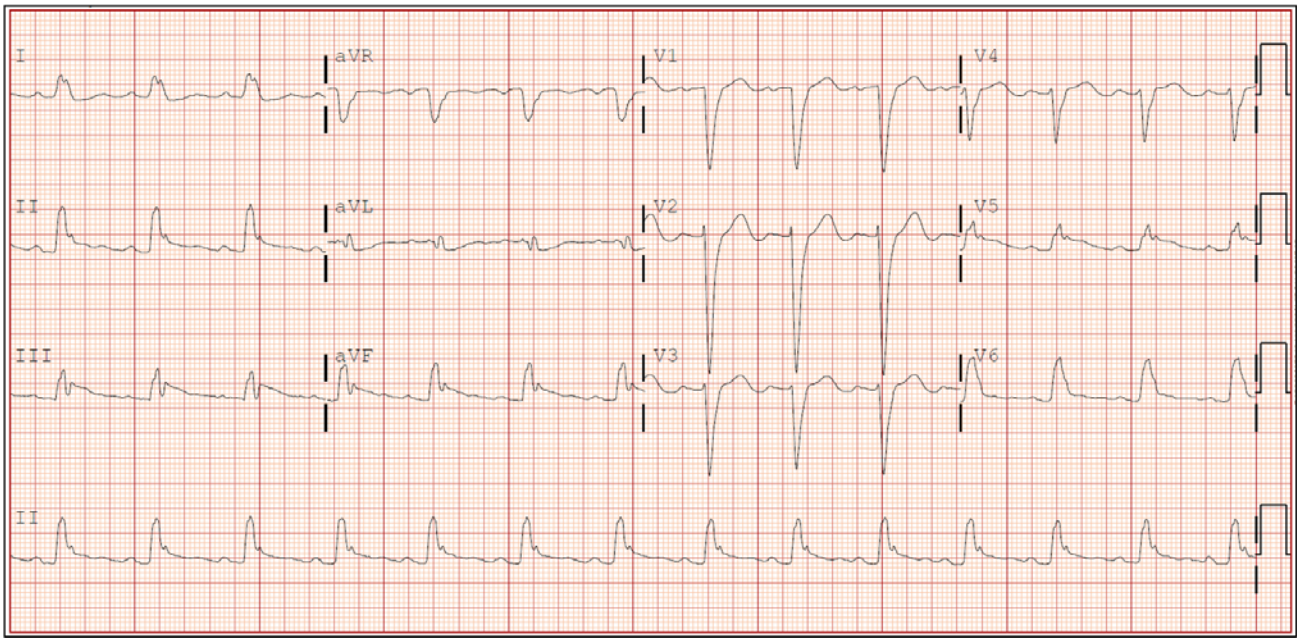
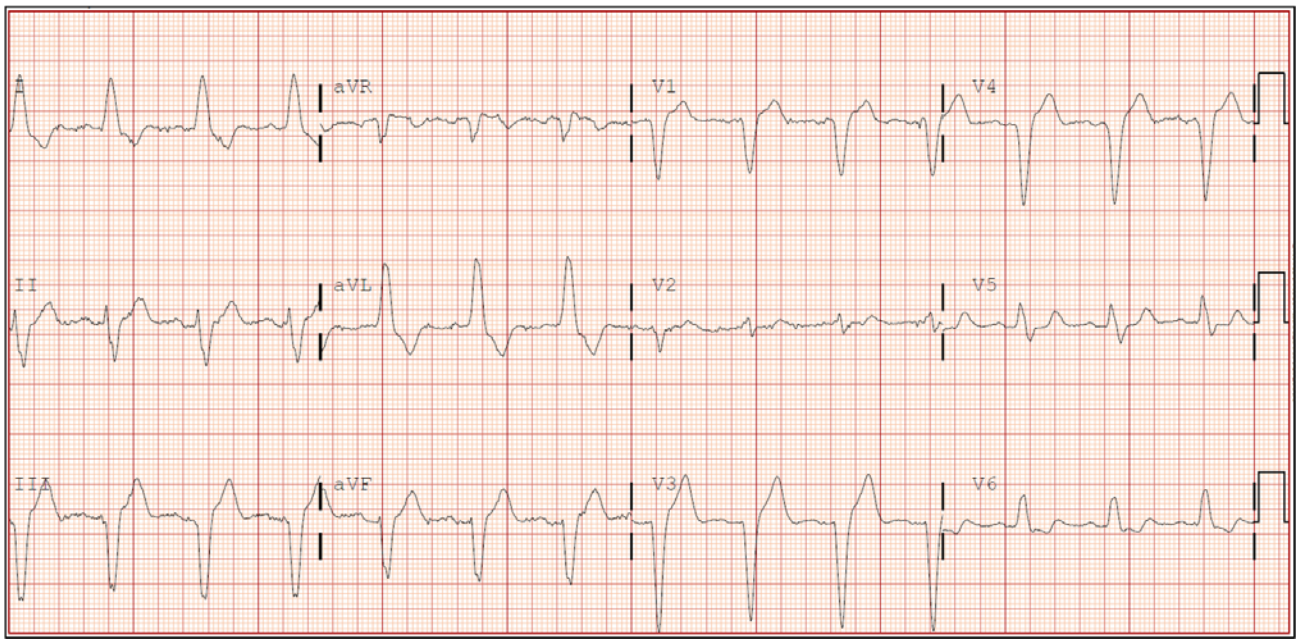


Figure 7-4 Negative acute MI with LBBB



Configurable Sensitivity for Acute Myocardial Infarction

Since detection of likely acute MI is difficult in the presence of ST elevation (STE) confounders, such as left bundle branch block (LBBB), right bundle branch block (RBBB), and left ventricular hypertrophy (LVH), a **Low Sensitivity Acute MI** setting is configurable for acute MI detection.

WARNING **The *Low Sensitivity Acute MI* setting is intended for the EMS pre-hospital setting where false-positive acute MI ECG interpretation may result in inappropriate cath lab activation. Before enabling this setting, carefully consider the clinical implications of this choice. Selecting this option, *Low Sensitivity Acute MI*, results in decreased AMI sensitivity, which could result in a reduction of automated Acute MI interpretation statements.**

When the **Low Sensitivity Acute MI** setting is enabled and confounders are present, the algorithm sets a higher threshold for ST-segment elevation before generating Acute MI interpretation statements. Since the higher the ST elevation, the more probable that the problem is acute MI rather than another condition, raising the threshold helps reduce the number of false positives, increasing confidence in the interpretation.

NOTE Thresholds do not change for ECGs without evidence of ST elevation confounders.

This feature, meant for the EMS pre-hospital environment, may not be available in all products; check your product's instructions for use. If available, the setting appears as a check box in Configuration:

Low Sensitivity Acute MI: Off (default) / On

Criteria to Exclude False Positive Acute MI Cases

Several methods have been proposed to identify STEMI from LBBB, such as the Sgarbossa score[19] or the modified Sgarbossa score by Smith[20]. Basically, they look at one key feature in the 12-lead ECG: the amplitude ratio between the ST segment and the S wave. We used this idea to develop unified criteria to distinguish the secondary STE confounders from acute MI ECGs for each of LBBB, RBBB, and LVH. For the remainder of the discussion, we lump LBBB, RBBB, and non-specific IVCD together using the general term *intra-ventricular conduction delay (IVCD)*.

The IVCD criterion is as follows:

Check an IVCD ECG with ST elevation for the likelihood of the opposite polarity of the ST segment and S wave. The closer the ST segment is to the opposite polarity of the S wave, the less likely the condition is to be acute MI. The associated statement is:

[STVCD] ST elevation secondary to IVCD

Similarly, the LVH criterion is as follows:

Check ECGs with elevated ST segments that meet LVH criteria for the likelihood of the opposite polarity of the ST segment and S wave.

When the **Low Sensitivity Acute MI** setting is enabled, we turn off detection of acute MI in the presence of LVH. Detection of acute MI in the presence of LVH is very difficult; false positive acute MI detection can lead to inappropriate coronary catheterization laboratory activation for STEMI.

[STLVH] ST elevation secondary to LVH

19. Sgarbossa EB, Pinski SL, Barbagelata A, et a. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. *N Engl J Med.* 1996; 334: p. 481-487.

20. Smith SW, Dodd KW, Henry TD, Dvorak DM, Pearce LA. Diagnosis of ST-Elevation Myocardial Infarction in the Presence of Left Bundle Branch Block with the ST-Elevation to S-Wave Ratio in a Modified Sgarbossa Rule. *Ann of Emerg Med.* 2012; 60(6): p. 766-776.

Acute MI Validation Database

The algorithm was developed based on existing MI database, using ECGs that meet the criteria listed in Table 7-5. The distribution of ECGs is shown in Table 7-6.

Table 7-5 Characteristics of ECGs in the IVCD and LVH training databases

IVCD training database ECGs	■ ECGs with LBBB, RBBB ■ QRS duration > 130 ms ■ ST elevation meets STEMI limits[11] ■ Recognized by the DXL algorithm ■ From the MI databases
LVH training database ECGs	■ ECGs with LVH ■ ST elevation meets STEMI limits[11] ■ Recognized by the DXL algorithm ■ From the MI databases

Table 7-6 Distribution of ECGs in training and validation databases

IVCD training database	■ 38 positive cases of acute MI ■ 50 negative cases of acute MI
LVH training database	■ 72 positive cases of acute MI ■ 501 negative cases of acute MI
IVCD validation database	■ 295 positive cases of acute MI ■ 1167 negative cases of acute MI, all with ST elevation
LVH validation database	■ 192 positive cases of acute MI ■ 581 negative cases of acute MI, all with ST elevation

The detection performance with IVCD and LVH are shown in Tables 7-7 and 7-8.

Table 7-7 Training database for acute MI detection performance with IVCD, LVH

	Low sensitivity AMI switch OFF		Low sensitivity AMI switch ON	
	Sensitivity	Specificity	Sensitivity	Specificity
IVCD with STelev	84.2%	88%	76%	94%
LVH with STelev	55.6%	97.4%	NA	NA

Table 7-8 Test database for acute MI detection performance with IVCD, LVH

	Low sensitivity AMI switch OFF		Low sensitivity AMI switch ON	
	Sensitivity	Specificity	Sensitivity	Specificity
IVCD with STelev	46.1%	91.4%	35.3%	94.5%
LVH with STelev	53.7%	60.9%	NA	NA

New DXL ECG Algorithm Interpretive Statements

The following interpretive statements have been added.

Table 7-9 New Acute Myocardial Infarct Critical Value Statements

Statement Code	Interpretive Statement
AMIAPD	Anterior infarct, acute (proxLAD)ST >0.2mV, V2-V5
IMIAPR[a]	Inferior infarct, acute (proxRCA)ST>0.10mV in III > II
LBBMIP	Probable acute infarct and LBBBconcordant ST
REPT6D	Abnormal T, suspect prox. LAD occlu. or CVASymmetric T<-0.5mV, V2-V4
STVCD	ST elevation secondary to IVCDMultiple VCG criteria
STLVH	ST elevation secondary to LVHMultiple VCG criteria
STHR	ST elevation secondary to high heart raten/a
STAFL	ST elevation secondary to atrial fluttern/a
BVPACE	Biventricular paced rhythmn/a
MISZDF	Estimated MI size: <percentage>50 criteria Selvester score
PERMIB[a]	Possible peri-infarction blockn/a
PERISB[a]	Peri-ischemic blockn/a

a. Edit-only

Asian Criteria for Left Ventricular Hypertrophy

Commonly used thresholds for the Sokolow-Lyon LVH (left ventricular hypertrophy) voltage criteria in Asian populations are 4.0 mV for men and 3.5 mV for women[21].

The DXL algorithm provides a configuration for using these Asian LVH thresholds. When Asian LVH thresholds are enabled, Sokolow-Lyon is the only voltage criteria used, and the common Asian thresholds are used.

21. Jie, Wu, Chunfang, Xu, Zaiying, Lu, Yongqun, Leng "Performance of Cornell Index and Sokolow Index criteria for left ventricular hypertrophy in Chinese subjects." Journal of Electrocardiology, 2006; 39(4):S35

A

Normal Measurement Values

Table A-1 Summary of Normal Values

Age Group	Heart Rate (beats/min)*	Frontal Plane QRS Vector (degrees)	PR Interval (sec)	QRS Duration V_5	Q III (mm)†‡	Q V_6 (mm)†	RV_1 (mm)	SV_1 (mm)
Less than 1 day	93–154 (123)	+59 to -163 (137)	0.08–0.16 (.11)	.03–0.07 (.05)	4.5	2	5–26 (14)	0–23 (8)
1 to 2 days	91–159 (123)	+64 to -161 (134)	0.08 - 0.14 (.11)	.03–.07 (.05)	6.5	2.5	5–27 (14)	0–21 (9)
3 to 6 days	91–166 (129)	+77 to -163 (132)	0.07–0.14 (.10)	.03–.07 (.05)	5.5	3	3–24 (13)	0–17 (7)
1 to 3 weeks	107–182 (148)	+65 to +161 (110)	0.07 - 0.14 (.10)	.03–.08 (.05)	6	3	3–21 (11)	0–11 (4)
1 to 2 months	121–179 (149)	+31 to +113 (74)	0.07–0.13 (10)	.03–.08 (.05)	7.5	3	3–18 (10)	0–12 (5)
3 to 5 months	106–186 (141)	+7 to +104 (60)	0.07–0.15 (.11)	.03–.08 (.05)	6.5	3	3–20 (10)	0–17 (6)
6 to 11 months	109–169 (134)	+6 to +99 (56)	0.07 - 0.16 (.11)	.03–.08 (.05)	8.5	3	1.5–20 (9.5)	.5–18 (4)
1 to 2 years	89–151 (119)	+7 to +101 (55)	0.08 - 0.15 (.11)	.04–.08 (.06)	6	3	2.5–17 (9)	.5–21 (8)
3 to 4 years	73–137 (108)	+6 to +104 (55)	0.09–0.16 (.12)	.04–.08 (.06)	5	3.5	1–18 (8)	.2–21 (10)
5 to 7 years	65–133 (100)	+11 to +143 (65)	0.09–0.16 (.12)	.04–.08 (.06)	4	4.5	.5–14 (7)	.3–24 (12)
8 to 11 years	62–130 (91)	+9 to +114 (61)	0.09–0.17 (.13)	.04–.09 (.06)	3	3	0–12 (5.5)	.3–25 (12)
12 to 15 years	60–119 (85)	+11 to +130 (59)	0.09–0.18 (.14)	.04–.09 (.07)	3	3	0–10 (4)	.3–21 (11)

Source: Garson A, Bricker JT, Fisher DJ, Neish SR (eds): *The Science and Practice of Pediatric Cardiology, Volume I (Second Edition)*, Baltimore, Williams & Wilkins p. 736 (1998). Reproduced by permission of the publisher.

* 2 to 98% (mean)
† Ninety-eighth percentile
‡ Millimeters at normal standarization
§ Undefined

Table A-2 Summary of Normal Values

Age Group	R/SV_1	RV_6 (mm)	SV_6 (mm)	R/SV_6	R + S V_4 (mm)†	$SV_1 + RV_6$ (mm)†
Less than 1 day	.1-U§ (2.2)	0-11 (4)	0-9.5 (3)	.1-U§ (2.0)	52.5	28
1 to 2 days	.1-U§ (2.0)	0-12 (4.5)	0-9.5 (3)	.1-U§ (2.5)	52	29
3 to 6 days	.2-U§ (2.7)	.5-12 (5)	0-10 (3.5)	.1-U§ (2.2)	49	24.5
1 to 3 weeks	1.0-U§ (2.9)	2.5-16.5 (7.5)	0-10 (3.5)	.1-U§ (3.3)	49	21
1 to 2 months	.3-U§ (2.3)	5-21.5 (11.5)	0-6.5 (3)	.2-U§ (4.8)	53.5	29
3 to 5 months	.1-U§ (2.3)	6.5-22.5 (13)	0-10 (3)	.2-U§ (6.2)	61.5	35
6 to 11 months	.1-3.9 (1.6)	6-22.5 (12.5)	0-7 (2)	.2-U§ (7.6)	53	32
1 to 2 years	.05-4.3 (1.4)	6.5-22.5 (13)	0-6.5 (2)	.3-U§ (9.3)	49.5	39
3 to 4 years	.03-2.8 (.9)	8-24.5 (15)	0-5 (1.5)	.6-U§ (10.8)	53.5	42
5 to 7 years	.02-2.0 (.7)	8.5-26.5 (16)	0-4 (1)	.9-U§ (11.5)	54	47
8 to 11 years	0-1.8 (.5)	9-25.5 (16)	0-4 (1)	1.5-U§ (14.3)	53	45.5
12 to 15 years	0-1.7 (.5)	6.5-23 (14)	0-4 (1)	1.4-U§ (14.7)	50	41

Source: Garson A, Bricker JT, Fisher DJ, Neish SR (eds): *The Science and Practice of Pediatric Cardiology, Volume I (Second Edition)*, Baltimore, Williams & Wilkins p. 736 (1998). Reproduced by permission of the publisher.

* 2 to 98% (mean)
† Ninety-eighth percentile
‡ Millimeters at normal standarization
§ Undefined

B

Interpretive Statements, by Category

Introduction

Appendix B contains a listing (by diagnostic category) of all of the Adult, Pediatric, and Technical Quality statements available in the Philips DXL ECG Algorithm.

NOTE **The symbol *** in an interpretive statement is replaced with a numeric value on the ECG report.**

Pediatric Header and Age Unknown

Table B-1 Pediatric Header and Age Unknown Statements

Pediatric Header and Age Unknown Statements		
Statement Code	Interpretive Statement	Notes
PEDH	Pediatric Header & Age Unknown	
AGEUNK	Age is not entered, assumed to be ** years old for purpose of ECG interpretation	
GEMUNK	Gender not entered, assumed to be male for purpose of ECG interpretation	New statement
GEFUNK	Gender not entered, assumed to be female for purpose of ECG interpretation	New statement
AGMUNK	Age and gender not entered, assume AGE yo male	New statement
AGFUNK	Age and gender not entered, assume AGE yo female	New statement
PED	---------- Pediatric ECG interpretation ----------	

Technical Quality

Table B-2 Technical Quality Notification Statements

Technical Quality Notification Statements		
Statement Code	Interpretive Statement	Notes
PLMP	Misplaced precordial electrode(s)	Statement added only at editing
LALLV	Left arm and left leg electrode reversal	Statement added only at editing
RALLV	Right arm and left leg electrode reversal	New statement
CHLDP	Consider high precordial lead placement	Statement added only at editing
PEERV	Probable extremity electrode reversal.....** reversed	New statement
PPERV	Probable precordial electrode reversal.....** reversed	New statement
RALARV	Right and left arm electrode reversal	
ECGSIM	Input from ECG simulator or V1-V4 shorted ...no analysis performed	
NAPHF	No analysis due to possible hardware failure.....Channels 1, 2, 3 identical	
TPT	Poor-quality data - please repeat ECG!	
12ML	All 12 leads are missing	
MISLDS	Incomplete analysis due to missing data in precordial lead(s)	
MAGNET	ECG acquired with magnet in place	

Paced Rhythm

Table B-3 Paced Rhythm

Paced Rhythm Statements		
Statement Code	Interpretive Statement	Notes
VPRMPT	Ventricular pacing preempted by intrinsic complex	Statement added only at editing
VPNAO	Ventricular pacing of non-right ventricular apical origin	Statement added only at editing
PFNAC	Pacemaker failure to capture, atrial	Statement added only at editing
PFNVC	Pacemaker failure to capture, ventricular	Statement added only at editing
PFNAI	Pacemaker failure to inhibit, atrial	Statement added only at editing
PFNVI	Pacemaker failure to inhibit, ventricular	Statement added only at editing
PFNAP	Pacemaker failure to pace, atrial	Statement added only at editing
PFNVP	Pacemaker failure to pace, ventricular	Statement added only at editing
UNKRM	Uncertain rhythm: review.....rhythm measurements incomplete	
PSAR	Pacemaker spikes or artifacts.....timing non-diagnostic	
PCMMC	A-V dual-paced complexes w/ some inhibition.....other complexes also detected	
PCMM	A-V dual-paced rhythm with some inhibition.....atrial and/or vent inhibition	
APACEC	Atrial-paced complexes.....other complexes also detected	

Table B-3 Paced Rhythm *(continued)*

Paced Rhythm Statements		
Statement Code	Interpretive Statement	Notes
APACED	A-paced complexes with some inhibition.....non-paced complexes also detected	
APACE	Atrial-paced rhythm	
VPACEC	Ventricular-paced complexes.....other complexes also detected	
VPACCF	Afib/Flut and V-paced complexes.....other complexes, A-rate>240	
VPACCD	V-paced complexes with some inhibition.....non-paced complexes also detected	
VPACFD	Afib/Flut, v-paced complexes with inhibition. ....non-paced complexes, A-rate>240	
VPACE	Ventricular-paced rhythm	
ASVPS	Atrial-sensed ventricular-paced complexes..... other complexes also detected	New statement
ASVP	Atrial-sensed ventricular-paced rhythm..... ventricular pacing tracks p-waves	
VPACEF	Afib/Flutter and ventricular-paced rhythm.....V-paced rhythm, A-rate>240	
AVDPC	Atrial-ventricular dual-paced complexes.....other complexes also detected	
AVDPCF	Dual-pacemaker w/ a-noncapt due to Afib/Flut...other complexes and A-rate>240	
AVDP	Atrial-ventricular dual-paced rhythm	
AVDPF	Dual-pacemaker w/ a-noncapt due to Afib/Flut.....dual pacing with A-rate>240	
BVPACE	Biventricular paced rhythm.....non-simultaneous bi-vent pacing	
ABVPC	Atrial- biventricular paced rhythm.....non-simultaneous bi-vent pacing	
PACENC	Pacemaker failure to capture	
PACENS	Pacemaker failure to sense	
PCNSNC	Pacemaker failure to capture and sense	

Table B-3 Paced Rhythm *(continued)*

Paced Rhythm Statements		
Statement Code	Interpretive Statement	Notes
PACEM	Failure to sense and/or capture (?magnet).....fixed pacing with async rhythm	

Paced Rhythm Disclaimer

Table B-4 Paced Rhythm Disclaimer

Paced Rhythm Disclaimer Statements		
Statement Code	Interpretive Statement	Notes
NFRA	No further rhythm analysis attempted due to paced rhythm	
NFAD	No further analysis attempted due to paced rhythm	

Insufficient Lead Measurements

Table B-5 Insufficient Lead Measurements

Paced Rhythm Disclaimer Statements		
Statement Code	Interpretive Statement	Notes
NFAMLD	No further analysis attempted for this ECG - not enough leads could be measured	

Basic Cardiac Rhythm

Table B-6 Basic Cardiac Rhythm

Basic Cardiac Rhythm Statements		
Statement Code	Interpretive Statement	Notes
SVRHY	Supraventricular rhythm	Statement added only at editing
BRDYNS	Bradycardia, nonsinus	Statement added only at editing

Table B-6 Basic Cardiac Rhythm *(continued)*

Basic Cardiac Rhythm Statements		
Statement Code	Interpretive Statement	Notes
LLAR	Low left atrial rhythm	Statement added only at editing
HLAR	High left atrial rhythm	Statement added only at editing
LRAR	Low right atrial rhythm	Statement added only at editing
HRAR	High right atrial rhythm	Statement added only at editing
CAVNRE	Consider AV nodal reentry	Statement added only at editing
CAVRE	Consider AV reentry	Statement added only at editing
SR	Sinus rhythm.....normal P axis, V-rate ** - **	
SB	Sinus bradycardia.....rate **	
ST	Sinus tachycardia.....rate> **	
SEAR	Sinus or ectopic atrial rhythm.....P axis (-45,135)	
SEAB	Sinus or ectopic atrial bradycardia.....P axis (-45,135), rate< **	
SEAT	Sinus or ectopic atrial tachycardia.....P axis (-45,135), rate> **	
EAR	Ectopic atrial rhythm.....abnormal P axis, normal rate	
EARM	Ectopic atrial rhythm, multifocal	Statement added only at editing
EAB	Ectopic atrial bradycardia.....abnormal P axis, V-rate< **	
EAT	Ectopic atrial tachycardia, unifocal.....abnormal P axis, V-rate> **	

Table B-6 Basic Cardiac Rhythm *(continued)*

Basic Cardiac Rhythm Statements		
Statement Code	Interpretive Statement	Notes
JER	Junctional rhythm.....absent P waves, slow V-rate	
JRA	Accelerated junctional rhythm.....absent P waves, accele'd V-rate	
IDOVR	Idioventricular rhythm	Statement added only at editing
AIDOVR	Accelerated idioventricular rhythm	Statement added only at editing
JT	Junctional tachycardia.....absent P waves, rapid V-rate	
RVAR	Unknown rhythm, irregular rate ..V-rate ** - ** , variation>**	
BWRV	Bradycardia with irregular rate .....V-rate ** - ** , mean < **	
TWRV	Sinus tachycardia with irregular rate .....V-rate ** - ** , variation>**	
SA	Sinus arrhythmia.....V-rate ** - ** , variation>**	
SAB	Slow sinus arrhythmia.....V-rate ** - ** , mean< **	
SAT	Fast sinus arrhythmia.....V-rate ** - ** , mean> **	
WPACE	Wandering atrial pacemaker.....varying PR interval & P axis	
MFAT	Ectopic atrial tachycardia, multifocal	Statement added only at editing
AVDIS	AV dissociation.....PR variation>**	
ETACH	Extreme tachycardia.....V-rate >(220-age)	
NQRST	Narrow-QRS tachycardia	Statement added only at editing
VT	Ventricular tachycardia	Statement added only at editing

Table B-6 Basic Cardiac Rhythm *(continued)*

Basic Cardiac Rhythm Statements		
Statement Code	Interpretive Statement	Notes
SVT	Supraventricular tachycardia.....V-rate>(220-age), QRSd< **	
AFIBT	Atrial fibrillation with rapid V-rate.....A-rate **	
TACHW	Wide-QRS tachycardia.....V-rate> ** , QRSd> **	
VTACH	Extreme tachycardia with wide complex, no further rhythm analysis attempted	
ARYP	Possible atrial arrhythmia.....A-rate ** , multiple Ps	
FLFIB	Atrial flutter/fibrillation.....A-rate ** , multiple Ps	
AFIB0	Atrial fibrillation.....? atrial activity	
AFIB	Atrial fibrillation.....V-rate ** - ** , irregular A-activity	
AFLT	Atrial flutter.....A-rate ** **	
AFLT2	A-flutter w/ predom 2:1 AV block.....A-rate ** , multiple Ps	
AFL2	Atrial flutter with 2:1 AV block.....A-rate ** , V-rate> **	
AFLT3	A-flutter w/ predom 3:1 AV block.....A-rate ** , multiple Ps	
AFLT4	A-flutter w/ predom 4:1 AV block.....A-rate ** , multiple Ps	
AFLTV	A-flutter w/ varied AV block, ...A-rate ** , varied AV conduction	
2AVB	Second degree AV block, Mobitz II.....multiple P waves	
2AVB2	Predominant 2:1 AV block.....most complexes 2 Ps	
2AVB3	Predominant 3:1 AV block.....most complexes 3 Ps	
2AVB4	Predominant 4:1 AV block.....most complexes 4 Ps	
2AVBV	AV block, varying conduction.....multiple Ps, varied AV conduction	
3AVB	AV block, complete (third-degree)....V-rate<45, AV dissociation	
3AVBIR	Complete AV block with wide QRS complex.....V-rate< ** , QRSd> ** , AV dissoc	

Table B-6 Basic Cardiac Rhythm *(continued)*

Basic Cardiac Rhythm Statements		
Statement Code	Interpretive Statement	Notes
3AVBFF	A-flutter/fibrillation w/ complete AV block...A-rate>220, V-rate< ** , AV dissoc	

Premature Complexes

Table B-7 Premature Complexes

Premature Complexes Statements		
Statement Code	Interpretive Statement	Notes
FASCR	Fascicular rhythm	Statement added only at editing
PARSYS	Parasystole	Statement added only at editing
FASCT	Fascicular tachycardia	Statement added only at editing
UNKBIG	Bigeminal pattern, uncertain mechanism	Statement added only at editing
UNKTRI	Trigeminal pattern, uncertain mechanism	Statement added only at editing
SVTRI	Supraventricular trigeminy	Statement added only at editing
SVUNK	Uncertain supraventricular rhythm	Statement added only at editing
JBIG	Junctional rhythm with VPCs in a bigeminal pattern	Statement added only at editing
JTRI	Junctional rhythm with VPCs in a trigeminal pattern	Statement added only at editing

Table B-7 Premature Complexes *(continued)*

Premature Complexes Statements		
Statement Code	Interpretive Statement	Notes
JESC	Junctional escape complex(es)	Statement added only at editing
ABAPC	Aberrant conduction of supraventricular complex(es)	Statement added only at editing
APCNC	Atrial premature complex(es), nonconducted	Statement added only at editing
RECA	Retrograde atrial activation	Statement added only at editing
UNKSV	Supraventricular complex(es)	Statement added only at editing
UNKPC	Premature complex(es), uncertain mechanism	Statement added only at editing
VSVPC	Premature complex, vent or aberrant supravent	Statement added only at editing
FUSN	Fusion complex(es)	Statement added only at editing
VESC	Ventricular escape complex(es)	Statement added only at editing
VTPOLY	Ventricular tachycardia, polymorphous	Statement added only at editing
TORSAD	Ventricular tachycardia, torsades de pointes	Statement added only at editing
VFIB	Ventricular fibrillation	Statement added only at editing

Table B-7 Premature Complexes *(continued)*

Premature Complexes Statements		
Statement Code	Interpretive Statement	Notes
APC	Atrial premature complex....SV complex w/ short R-R interval	
JPC	Junctional premature complex(es)...SV complex w/ short R-R, absent P	
MAPC	Atrial premature complexes.....SV complexes w/ short R-R intvls	
SVBIG	Supraventricular bigeminy.....bigeminy string>4 w/ SV complexes	
APCPR	Atrial premature complexes in couplets.....pair SV complexes w/ short R-R	New statement
SVTNS	Supraventricular tachycardia, non-sustained.....run SV complexes w/ short R-R	New statement
IVPC	Interpolated ventricular premature complex.....interpolated complex, wide QRS	
MIVPC	Multi interpolated vent premature complexes.interpolated complexes, wide QRSd	
VPC	Ventricular premature complex.....V complex w/ short R-R interval	
MVPC	Multiple ventricular premature complexes....V complexes w/ short R-R intervls	
MVSPC	Multiple premature complexes, vent & supraven.V and SV complexes w/ short R-R	
VBIG	Ventricular bigeminy.....bigeminy string>4 w/ V complexes	
VTRI	Ventricular trigeminy.....trigeminy string>6 w/ V complexes	
MFVPC	Multiform ventricular premature complexes.....short R-R, variable morphology	
PVPC	Paired ventricular premature complexes.....sequence of 2 V complexes	
RVPC	Ventricular tachycardia, unsustained.....sequence of 3 or more V complexes	
MFPVPC	Paired multiform ventricular complexes.....sequence of 2 V complexes	

Table B-7 Premature Complexes *(continued)*

Premature Complexes Statements		
Statement Code	Interpretive Statement	Notes
MFRVPC	Run of multiform ventricular complexes.....sequence of 3 or more V complexes	

Pauses, AV Block

Table B-8 Pauses, AV Block

Pauses, AV Block Statements		
Statement Code	Interpretive Statement	Notes
SABLK1	Sinoatrial block, type 1	Statement added only at editing
SABLK2	Sinoatrial block, type 2	Statement added only at editing
SADIS	Suggest sinoatrial disorder	Statement added only at editing
SAPU	Pause of uncertain mechanism	Statement added only at editing
SARSV	Sinus pause.....long R-R interval, normal QRSd	
SARN	Sinus pause with junctional escape	Statement added only at editing
SARA	Sinus pause with atrial escape	Statement added only at editing
I2AVB	Second degree AV block, intermittent..long R-R with multiple Ps	Statement added only at editing
A2AVB	Second degree AV block, alternating.....alternating long R-R, multiple Ps	Statement added only at editing

Table B-8 Pauses, AV Block *(continued)*

Pauses, AV Block Statements		
Statement Code	Interpretive Statement	Notes
2AVBA	AV block, advanced (high-grade)	Statement added only at editing
LRRV	Long r-r with ventricular escape.....R-R>** of normal, wide QRS	
SARV	Sinus pause with ventricular escape..long R-R interval, wide QRS	
WENCK	Second deg AVB, Mobitz I (Wenckebach)....PR lengthens & dropped complexes	

Miscellaneous Arrythmias

Table B-9 Miscellaneous Arrythmias

Miscellaneous Arrythmias Statements		
Statement Code	Interpretive Statement	Notes
ABC	Aberrant complex.....small R-R variation, aberrant QRS	
ABCS	Aberrant conduction of SV complex(es).....aberrant shape, PR 80-220	

AV Conduction

Table B-10 AV Conduction

AV Conduction Statements		
Statement Code	Interpretive Statement	Notes
SPRB	Borderline short PR interval.....PR int < ** mS	
SPR	Short PR interval.....PR < ** mS	
BAVCD	Borderline prolonged PR interval.....PR > ** , V-rate ** - **	
1AVB	Prolonged PR interval.....PR > ** , V-rate ** - **	

Dextrocardia, Preexcitation

Table B-11 Dextrocardia, Preexcitation Statements

Dextrocardia, Preexcitation Statements		
Statement Code	Interpretive Statement	Notes
VPERP	Ventricular preexcitation (WPW), a right posteroseptal accessory pathway	Statement added only at editing
VPELP	Ventricular preexcitation (WPW), a left posteroseptal accessory pathway	Statement added only at editing
VPERA	Ventricular preexcitation (WPW), a right anteroseptal accessory pathway	Statement added only at editing
VPELA	Ventricular preexcitation (WPW), a left anteroseptal accessory pathway	Statement added only at editing
VPERL	Ventricular preexcitation (WPW), a right lateral accessory pathway	Statement added only at editing
VPELL	Ventricular preexcitation (WPW), a left lateral accessory pathway	Statement added only at editing
CDEXP	Consider dextroposition	Statement added only at editing
DEXC	Consider dextrocardia.....P, QRS axis rightward	
VPE	Ventricular preexcitation(WPW).....Delta waves	
VPEL	Vent pre-excitat'n (WPW), left acces'y pathway...Delta wave & initial axis (30,120)	
VPER	Vent pre-excit'n (WPW), right access'y pathway...Delta wave & initial axis (-60,29)	

Right Atrial Abnormality

Table B-12 Right Atrial Abnormality

Right Atrial Abnormality Statements		
Statement Code	Interpretive Statement	Notes
RAA	Right atrial conduction abnormality	
CRAE	Consider right atrial enlargement.....P >0.24mV limb lead	CRAA
PRAE	Probable right atrial enlargement....biphasic P >0.20 mV in V1	PRAA
RAE	Right atrial enlargement...P>0.25mV 2 lds or <-0.24mV aVR/aVL	RAA

Left Atrial Abnormality, Biatrial Abnormality

Table B-13 Left Atrial Abnormality, Biatrial Abnormality

Left Atrial Abnormality, Biatrial Abnormality Statements		
Statement Code	Interpretive Statement	Notes
LAA	Left atrial conduction abnormality	Statement added only at editing
OSBW	Osborn wave suggests hypothermia	Statement added only at editing
CLAE	Consider left atrial enlargement.....wide or notched P waves	CLAA
PLAE	Probable left atrial enlargement.....P >50mS, <-0.10mV V1	PLAA
PPND	Prominent P waves, nondiagnostic....wide/notched/ biphasic P waves	
LAE	Left atrial enlargement.....P, P'>60mS, <-0.15mV V1	LAA
LAECB	LAE, consider biatrial enlargement.....P>80mS <-.15mV V1&>.25mV limb lds	LAACB
RAECB	RAE, consider biatrial enlargement.....P>0.30mV 2 lds & <-0.30mV aVR/aVL	RAACB
BAE	Biatrial enlargement..P>80mS,<-0.15mV V1 &>0.30mV 2 lds	BAA

QRS Axis Deviation

Table B-14 QRS Axis Deviation

QRS Axis Deviation Statements		
Statement Code	Interpretive Statement	Notes
ELALT	Electrical alternans	Statement added only at editing
ABPAX	Abnormal P-wave axis	Statement added only at editing
AXR	Borderline right-axis deviation.....QRS axis (** , **)	
RAD	Right-axis deviation.....QRS axis (** , **)	
AXL	Borderline left-axis deviation.....QRS axis (** , **)	
LAD	Left-axis deviation.....QRS axis (** , **)	
AXSUP	Right superior axis.....QRS axis (-91,240)	
AXIND	Indeterminate axis.....QRS axis indeterminate	
S123	S1,S2,S3 pattern.....S >30mS & >0.2mV, I II III	
AXPST	Markedly posterior QRS axis.....late V-lead transition	

Pediatric Ventricular Conduction Delay

Table B-15 Pediatric Ventricular Conduction Delay

Pediatric Ventricular Conduction Delay Statements		
Statement Code	Interpretive Statement	Notes
IVCDP	Nonspecific intraventricular conduction delay.....QRS > ** mS	
LAFBP	Left anterior fascicular block.....QRS axis (-60,-90)	
LBBBP	Left bundle-branch block...QRSd> ** mS, late forces leftward	
IRBBTA	Incomplete right bundle-branch block.....RSR' in V1, late forces anterior	
IRBBBP	Incomplete right bundle-branch block.....QRSd > ** , RSR' or pure R	

Table B-15 Pediatric Ventricular Conduction Delay *(continued)*

Pediatric Ventricular Conduction Delay Statements		
Statement Code	Interpretive Statement	Notes
RBBBP	Right bundle-branch block.....QRSd > ** , RSR' or pure R or QR	
RBBBM	Marked right bundle-branch block.....QRSd >160 mS	
RLAFBP	RBBB and LAFB.....QRSd>90, QRS(-60,-90)	

Ventricular Conduction Delay

Table B-16 Ventricular Conduction Delay Statements

Ventricular Conduction Delay Statements		
Statement Code	Interpretive Statement	Notes
IVCD	Intraventricular conduction delay	Statement added only at editing
EPSWV	Epsilon wave	Statement added only at editing
BIVCD	Borderline intraventricular conduction delay.....QRSd > ** mS	
BIVCDL	Borderline IVCD with LAD.....QRSd > ** mS, axis(-90,-30)	
NIVCD	Nonspecific intraventricular conduction delay.....QRSd > ** mS, not LBBB/RBBB	
NIVCDL	Nonspecific IVCD with LAD.....QRSd > ** mS & LAD	
IRBBB	Incomplete right bundle-branch block.....QRSd > ** , terminal axis (90,270)	
ARBBB	IVCD, consider atypical RBBB.....QRSd>120mS, terminal axis (90,270)	
CLAFB	LAD, consider left anterior fascicular block....axis (240,-40), S>R II III aVF	
LAFB	Left anterior fascicular block.....axis (240,-40), init forces inf	
CAFBII	LAD, consider LAFB or inferior infarct.....axis (240,-30), Q&R II III aVF	

Table B-16 Ventricular Conduction Delay Statements *(continued)*

Ventricular Conduction Delay Statements		
Statement Code	Interpretive Statement	Notes
IRAFB	Incomplete RBBB and LAFB....axis (240,-40), S>R II III aVF	
LPFB	Left posterior fascicular block.....trm axis (110,210), init force sup	
IRPFB	IRBBB and LPFB.....RAD, QRSd>120, term axis(90,270)	
RBBB	Right bundle-branch block..QRSd>120, terminal axis (90,270)	
RLAFB	RBBB and LAFB.....QRSd >120mS, axis (-40,240)	
RLPFB	RBBB and LPFB.....QRSd >120mS, axis (90,210)	
ILBBB	Incomplete left bundle-branch block.....QRSd>110mS, terminal axis (-90,-1)	
ALBBB	IVCD, consider atypical LBBB...QRSd> ** , notch/slur R I aVL V5-6	
LBBB	Left bundle-branch block.....QRSd> ** , broad/notched R	

Low Voltage, Pulmonary Disease Pattern

Table B-17 Low Voltage, Pulmonary Disease Pattern

Low Voltage, Pulmonary Disease Pattern Statements		
Statement Code	Interpretive Statement	Notes
CPEMB	Consider acute pulmonary embolism	Statement added only at editing
CPULM	Consider pulmonary disease	Statement added only at editing
CMYX	Consider hypothyroidism	Statement added only at editing
LVOLFB	Borderline low voltage, extremity leads.....all extremity leads <0.6mV	
LVOLF	Low voltage, extremity leads.....all extremity leads <0.5mV	

Table B-17 Low Voltage, Pulmonary Disease Pattern *(continued)*

Low Voltage, Pulmonary Disease Pattern*(continued)*Statements		
Statement Code	Interpretive Statement	Notes
LVOLP	Low voltage, precordial leads.....precordial leads <1.0mV	New statement
LVOLT	Low voltage, extremity and precordial leads extremity<0.5mV, precordial<1.0mV	
LVORAD	Low voltage with right-axis deviation.....low voltage, RAD	
CPDP	Pattern suggests chronic pulm disease.....P rightward, QRS small & vertical	
CPDLV	Low voltage consistent with COPD..low voltage and Dx COPD	

Pediatric Right Ventricular Hypertrophy

Table B-18 Pediatric Right Ventricular Hypertrophy Statements

Pediatric Right Ventricular Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
RSRNV	RSR' in V1, normal variation.....term-vector post-rightward	
IRBBRV	IRBBB, the RSR' pattern may also reflect RVH...IRBBB, R or R' >0.5mV in V1-V3	
RVHS6	Consider right ventricular hypertrophy.....deep S in V6	
RVHS5	Consider right ventricular hypertrophy.....deep S in V5	
RVHRS6	Consider right ventricular hypertrophy.....R/S <*.*** in V6	
RVHTA	Consider right ventricular hypertrophy.....late forces posterior rightward	
RVHA	Right-axis deviation, consider RVH.....frontal & init-horiz'l axis right	
RVHRP1	Consider right ventricular hypertrophy.....R' >0.5mV in V1	
RVHRS	Consider right ventricular hypertrophy.....R V1 + S V5 > *.***mV	

Table B-18 Pediatric Right Ventricular Hypertrophy Statements *(continued)*

Pediatric Right Ventricular Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
RVHR1	Probable right ventricular hypertrophy.....prominent R in 1 OF V1 V2 V3R V4R	
RVHPR1	Probable right ventricular hypertrophy.....pure R>*.***mV in V1	
RVHT1	Upright T in V1 or V2, probable RVH.....T >0.10 V1, 3d-9y	
RVHRD	Probable right ventricular hypertrophy.....RAD & 1 of R/R'1/2, S5/6, R1S5, T1	
RVHQR	Probable right ventricular hypertrophy...QR pattern V1, 0h-2d	
RVH2V	Right ventricular hypertrophy.....2 of R/R'V1/2, SV5/6, RV1SV5, TV1	
RVHAT	Right ventricular hypertrophy.....RAD & upright T	
RVHVT	Right ventricular hypertrophy.....TV1 & 1 of R/R'V1/2, SV5/6, R1S5	
RVHQRV	Right ventricular hypertrophy.....QRV1 & 1 of R/R'V1/2, SV5/6, R1S5	
RVHQR3	Right ventricular hypertrophy.....QR pattern V1, 3d-15y	

Pediatric Left Septal Hypertrophy

Table B-19 Pediatric Left Septal Hypertrophy

Pediatric Left Septal Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
LSHC	Prominent Q, consider left septal hypertrophy.....deep Q in V5-6	
LSH	Left septal hypertrophy...deep Q in V5-6, tall R in V1	

Pediatric Left Ventricular Hypertrophy

Table B-20 Pediatric Left Ventricular Hypertrophy

Pediatric Left Ventricular Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
LVHQ	Consider left ventricular hypertrophy.....deep Q in V5-6 or II III aVF	
LVHTA	Consider left ventricular hypertrophy...prominent left-ward forces	
LVHR6	LVH by voltage.....R >*.***mV in V6	
LVHS12	LVH by voltage.....S <*.*** in V1 or *.*** in V2	
LVHRS	Consider left ventricular hypertrophy.....RV6+SV1 >*.***mV	
LVHQR	Probable left ventricular hypertrophy.....Q>0.4 & R>*.*** V5 or *.*** V6	
LVHQV	Probable left ventricular hypertrophy.....Q56/II-aVF & 1 of S1/2, R6, S1R6	
LVHSTE	Repolarization abnormality suggests LVH.....ST>0.1mV, T>1.0 mV, I aVL V4-6	
LVHSTD	Repolarization abnormality suggests LVH.....ST<-0.01mV, T<-0.05, I aVL V4-6	
LVHR	Repolarization abnormality suggests LVH.....ST dep, T neg, I aVL V4-V6	
LVHVA	Probable left ventricular hypertrophy.....LAD & 1 of SV1/2, RV6, SV1+RV6	
LVHP	Probable LVH w/ secondary repol abnormalities...LAD, S1/2, R6, S1R6 & repol abnrm	
LVHEV	Left ventricular hypertrophy.....extreme leftward forces	
LVHVAQ	Left ventricular hypertrophy.....LAD, Q or 1 of SV1/2, RV6, SV1RV6	
LVHRE	LVH w/ secondary repolarization abnormalities...LAD, Q/SV12/RV6/S1R6, repol abnrm	

Pediatric Biventricular Hypertrophy

Table B-21 Pediatric Biventricular Hypertrophy

Pediatric Biventricular Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
RCLVH	RVH, consider associated LVH.....RVH & Q<-.07mV, R >1 mV V6	
BVHVC	Consider biventricular hypertrophy.....LVH & 1 of R/R'1/2, S5/6,R1+S5,T1	
BVHC	Consider biventricular hypertrophy.....R + S >6.0 mV in 2 of V2-V4	
BVHPED	Biventricular hypertrophy.....R/R'1/2, S5/6, R1S5 & S1/2, R6, S1R6	

Right Ventricular Hypertrophy

Table B-22 Right Ventricular Hypertrophy

Right Ventricular Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
RSR1	RSR' in V1 or V2, probably normal variant.....small R' only	
LT	Abnormal R-wave progression, late transition.....QRS area <0 in V5/V6	
ET	Abnormal R-wave progression, early transition.....QRS area>0 in V2	
ETRSR1	RSR' in V1 or V2, right VCD or RVH.....QRS area positive & R' V1/V2	
CRHPI	Consider RVH or posterior infarct.....large R in V1	
CRHPIR	Consider RVH or PMI w/ sec repol abnormality....large R V1, repol abnormality	
CRVH	Consider right ventricular hypertrophy.....large R or R' V1/V2	
CRVHR	Consider RVH w/ secondary repol abnormality....large R in V1/V2 & repol abnrm	
PRVH	Probable right ventricular hypertrophy.....prominent R or R' w/ RAD or RAE	

Table B-22 Right Ventricular Hypertrophy *(continued)*

Right Ventricular Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
PRVHR	Probable RVH w/ secondary repol abnormality.....prominent R or R′ & repol abnrm	
RVH	Right ventricular hypertrophy.....prominent R or R′ w/ RAD or RAE	
RVHR	RVH with secondary repolarization abnormality...prom R/ R′, RAD/RAE & repol abnrm	

Left Ventricular Hypertrophy

Table B-23 Left Ventricular Hypertrophy

Left Ventricular Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
LVHST	LVH with secondary repolarization changes	Statement added only at editing
HVOLT	High QRS voltage	Statement added only at editing
LVHV	LVH by voltage.....R >*.*** in aVL	
LVHR56	LVH by voltage.....R >*.***mV in V5 or V6	
LVHRSI	LVH by voltage.....(R I+S III) >*.***mV	
LVHSR	Consider left ventricular hypertrophy..... (S V1/V2 + R V5/V6) >*.***0mV	
LVHCNV	Consider left ventricular hypertrophy..... (R aVL+S V3) >*.***mV	
LVHC	Consider left ventricular hypertrophy.....multiple voltage criteria	
LVHVP	Probable left ventricular hypertrophy.....multiple LVH criteria	
LVHCNP	Probable left ventricular hypertrohy.....(R aVL + SV3) x RSd > **	
LVHPRE	Probable LVH with secondary repol abnrm.....multiple LVH criteria	

Table B-23 Left Ventricular Hypertrophy *(continued)*

Left Ventricular Hypertrophy Statements		
Statement Code	Interpretive Statement	Notes
LVH	Left ventricular hypertrophy.....multiple voltage criteria	
LVH1	Left ventricular hypertrophy.....multiple LVH criteria	
LVHREP	LVH with secondary repolarization abnormality..... multi-LVH criteria, repol abnrm	
LVHCO	LVH with IVCD and secondary repol abnrm.....multi-criteria, wQRSd, repol abnr	
LVHCOL	LVH with IVCD, LAD and secondary repol abnrm... multi-criteria, wQRSd, repol abnr	
BVH	Biventricular hypertrophy.....multiple LVH & RVH criteria	

Pediatric Abnormal Q Wave and Myocardial Infarction

Table B-24 Pediatric Abnormal Q Wave and Myocardial Infarction

Pediatric Abnormal Q Wave and Myocardial Infarction Statements		
Statement Code	Interpretive Statement	Notes
NORVAR	Consider normal variant	Statement added only at editing
PQIN	Borderline Q waves in inferior leads.....Qs add to 80 mS in II III aVF	
PQLA	Borderline Q waves in lateral leads...Q >35mS in I aVL V5 V6	
PQAN	Borderline Q wave in anterior leads.....Q >30mS in V2-V5	
PQAL	Borderline Q wave in anterolateral leads.....Q >35mS, I aVL V3-V6	
PIMI	Abnormal Q suggests inferior infarct.....Q >35mS in II III aVF	
PLMI	Abnormal Q suggests lateral infarct..Q >35mS in I aVL V5 V6	
PASMI	Abnormal Q suggests anteroseptal infarct...Q >30mS in V1 V2	

Table B-24 Pediatric Abnormal Q Wave and Myocardial Infarction *(continued)*

Pediatric Abnormal Q Wave and Myocardial Infarction Statements		
Statement Code	Interpretive Statement	Notes
PAMI	Abnormal Q suggests anterior infarct.....Q >30mS in V2-V4	
PALMI	Abnormal Q suggests anterolateral infarct.....Q>30mS I aVL V4-V6	

Inferior Infarct

Table B-25 Inferior Infarct

Inferior Infarct Statements		
Statement Code	Interpretive Statement	Notes
IBMI	Inferobasal MI	Statement added only at editing
IAPMI	Inferoapical MI	Statement added only at editing
INFQ	Abnormal inferior Q waves.....Qs add to 80 mS in II III aVF	IMI3
IMIC	Consider inferior infarct.....Q >35mS in II III aVF	IMI10
IQNV	Inferior Q waves, probably normal variation.... Q >30mS, age<21 male, <30 female	IMI18
IMIOP	Probable inferior infarct, old.....Q>35mS, II III aVF	IMI24
IMIQP	Probable inferior infarct, age indeterminate....Q>35mS, T neg, II III aVF	IMI26
ILMIQP	Probable inferolateral infarct, age indeterm.....Q >30mS, inf-lat leads	IMI30
IMIPR	Probable inferior infarct, possibly recent....Q>35mS, ST>0.1mV, T neg, II-aVF	IMI49M
IMIO	Inferior infarct, old.....Q >35mS, II III aVF	IMI64
IMIQ	Inferior infarct, age indeterminate...Q>35mS, T neg, II III aVF	IMI66
IQBBB	Inferior Q waves, possibly due to LBBB.....Q >35mS, II III aVF & LBBB	IMI80

Table B-25 Inferior Infarct *(continued)*

Inferior Infarct Statements		
Statement Code	Interpretive Statement	Notes
IMIBBB	Probable inferior infarct with LBBB.....Q>35mS, II III aVF & LBBB	IMI82
IMIRP	Probable inferior infarct, recent.....Q>25mS, ST>0.07mV, T neg, II-aVF	IMI54
IMIAP	Probable inferior infarct, acute.....ST>0.10mV, II III aVF	IMI50
IMIPA	Inferior infarct, possibly acute.....Q >30mS, ST >0.10mV, II III aVF	IMI67
ISTBBB	Inferior ST elevation, possibly due to LBBB.....ST>0.15mV, II III aVF & LBBB	IMI81
IMIR	Inferior infarct, recent....Q>35mS, ST>0.07mV, T neg, II-aVF	IMI74
IMIA	Inferior infarct, acute.....ST>0.10mV, T upright, II III aVF	
IMIAR	Inferior infarct, acute (RCA).....ST>0.10mV in III > II	New statement
IMIAX	Inferior infarct, acute (LCx).....ST>0.10mV, II III aVF, STd V1-V3	New statement

Posterior Infarct

Table B-26 Posterior Infarct

Posterior Infarct Statements		
Statement Code	Interpretive Statement	Notes
RPMIC	Tall R wave in V2, consider RVH or PMI.....R/S ratio >3, T >0.30mV V1 V2	CRPMI
PMIC	Consider posterior infarct.....prom R & T, V1-V3 or Q V7-V9	CPMI
IPMIC	Consider inferoposterior infarct.....inf Q, ant R or ST dep V1-3	CIPMI
PMIOP	Probable posterior infarct, old.....prom R, V1-V3 or Q >40mS, V7-V9	New statement
PMIO	Posterior infarct, old.....prom R T, V1-V3 or Q >40mS, V7-V9	New statement

Table B-26 Posterior Infarct *(continued)*

Posterior Infarct Statements		
Statement Code	Interpretive Statement	Notes
IPMIO	Inferoposterior infarct, old.....Q II-aVF & prom R T, V1-V3	New statement
PMIQP	Probable posterior infarct, age indeterminate...prom R T, V1-V3 or Q, Tneg, V7-V9	New statement
PMIQ	Posterior infarct, age indeterminate.....prom R, T, V1-3 or Q, Tneg, V7-9	
IPMIQ	Inferoposterior infarct, age indeterminate.....Q II-aVF & prom R T, V1-V3	New statement
PMIRP	Probable posterior infarct, recent.....prom R, STd, V1-3 or Q, STe, V7-9	New statement
PMIR	Posterior infarct, recent.....prom R & STd V1-3 or Q & STe V7-9	New statement
IPMIR	Inferoposterior infarct, recent.....prom R & STd V1-3 or Q & STe V7-9	New statement
PMIAP	Probable posterior infarct, acute.....ST<-.05 V1-V3 or >.05 V7-V9	New statement
PMIA	Posterior infarct, acute.....ST<-0.1 V1-V3 or ST>.05 V7-V9	
PMIAX	Posterior infarct, acute (LCx).....ST<-0.1 V1-V3 or ST>.05 V7-V9	New statement
IPMIA	Inferoposterior infarct, acute.....ST>.1 inf, <-.1 V1-3 or >.05 V7-9	
IPMIAR	Inferoposterior infarct, acute (RCA).....ST>.1 inf, <-.1 ant or>.05 V3R-5R	New statement
IPMIAX	Inferoposterior infarct, acute (LCx).....ST>.1 inf, <-.1 V1-3 or >.05 V7-9	New statement

Right Ventricular Infarct

Table B-27 Right Ventricular Infarct

Right Ventricular Infarct Statements		
Statement Code	Interpretive Statement	Notes
RMIOP	Probable right ventricular infarct, old.....Q >60mS, V3R-V5R	New statement
RMIO	Right ventricular infarct, old.....Q >80mS, V3R-V5R	New statement
RMIQP	Probable right ventricular infarct, age indet.....Q >60 & ST>.05, V3R-V5R	New statement
RMIQ	Right ventricular infarct, age indeterminate.....Q >70mS, ST >.05, V3R-V5R	New statement
RMIRP	Probable right ventricular infarct, recent.....Q > 50, ST >0.05, V3R-V5R	New statement
RMIR	Right ventricular infarct, recent.....ST >0.05, T upright, V3R-V5R	New statement
RMIAP	Probable right ventricular infarct, acute.....ST>.08, V3R-V5R, aVR & STd in lat	New statement
RMIA	Right ventricular infarct, acute.....ST>.10, V3R-V5R, aVR & STd in lat	New statement
RMIAR	Right ventricular infarct, acute (RCA).....ST>.08, aVR V3R-V5R & STd lat lds	New statement
RVINV	Right ventricle is also involved.....prom Q or STe V3R-V5R, ST dep lat	New statement

Lateral Infarct

Table B-28 Lateral Infarct

Lateral Infarct Statements		
Statement Code	Interpretive Statement	Notes
LATQ	Abnormal lateral Q waves.....Q >35mS, I aVL V5 V6	LMI10
LMIOP	Probable lateral infarct, old.....Q>35mS, abnormal ST-T, I aVL V5-6	LMI24
LQLVH	Lateral Q waves, probably due to LVH.....Q >35mS, I aVL V5 V6 & LVH	LMI28

Table B-28 Lateral Infarct *(continued)*

Lateral Infarct Statements		
Statement Code	Interpretive Statement	Notes
LQNV	Lateral Q waves, probably normal variation...Q >35mS, age<31 male, <40 female	LMI49
LMIO	Lateral infarct, old.....Q>40mS, flat T, I aVL V5 V6	LMI64
ILMIO	Inferolateral infarct, old.....Q >40mS, inf-lat leads	New statement
LMIQP	Probable lateral infarct, age indeterminate.....Q >35mS, I aVL V5 V6	LMI20
LMIQ	Lateral infarct, age indeterminate.....Q>35mS, T neg, I aVL V5 V6	LMI66
ILMIQ	Inferolateral infarct, age indeterminate.....Q >30mS, T neg, inf-lat leads	
LMIRP	Probable lateral infarct, recent.....Q>35mS, ST>.07mV,T neg, I aVL V5-6	LMI54
LMIR	Lateral infarct, recent.....Q>35, ST>.05mV, T neg, I aVL V5-6	LMI74
ILMIR	Inferolateral infarct, recent....ST>.05mV, T neg, inf-lat leads	New statement
LMIAP	Probable lateral infarct, acute.....Q >28mS, ST>0.10mV, I aVL V5 V6	LMI50
LMIPA	Lateral infarct, possibly acute.....Q >28mS, ST >0.10mV, I aVL V5 V6	LMI67
LMIA	Lateral infarct, acute.....ST >.10mV, I aVL V5 V6	
LMIAD	Lateral infarct, acute (LAD).....ST >.10mV, I aVL V5 V6	
ILMIA	Inferolateral infarct, acute.....ST>.10mV, inf-lat leads	
ILMIAR	Inferolateral infarct, acute (RCA)....ST>.10mV, inf-lat leads	New statement
ILMIAX	Inferolateral infarct, acute (LCx)....ST>.10mV, inf-lat leads	New statement

Anteroseptal and Anterior Infarct

Table B-29 Anteroseptal and Anterior Infarct

Anteroseptal and Anterior Infarct Statements		
Statement Code	Interpretive Statement	Notes
ABMI	Anterobasal MI	Statement added only at editing
AAPMI	Anteroapical MI	Statement added only at editing
MILBBB	MI in presence of left bundle-branch block	Statement added only at editing
ANTQ	Abnormal Q wave in V1.....Q >15mS in V1	AMI3
ANTR	Abnrm R prog, consider ASMI or lead placement....Q >30mS, diminished R, V1-V3	AMI4
ASMIC	Consider anteroseptal infarct.....Q >30mS, dimin R, V1-V3	AMI8
ASQBBB	Anterior Q waves, possibly due to ILBBB.....Q >30mS, V1 V2 & ILBBB	AMI16
ASQLVH	Anterior Q waves, possibly due to LVH.....Q >30mS, V1 V2 & LVH	AMI17
AMIC	Consider anterior infarct.....Q >30mS in V3-V5	AMI44
ASMIOP	Probable anteroseptal infarct, old.....Q >30mS & abn ST-T, V1-V3	AMI20
AMIOP	Probable anterior infarct, old.....Q >30mS, V2-V5	New statement
AQLVH	Anterior Q waves, possibly due to LVH.....Q >30mS in V2-V5 & LVH	New statement
ASMIO	Anteroseptal infarct, old.....Q >40mS, V1-V3	New statement
AMIO	Anterior infarct, old.....Q >30mS, abnormal ST-T, V2-V5	AMI60
ASMIQP	Probable anteroseptal infarct, age indeterm.....Q >30mS, T neg, V1-V3	AMI21

Table B-29 Anteroseptal and Anterior Infarct *(continued)*

Anteroseptal and Anterior Infarct Statements		
Statement Code	Interpretive Statement	Notes
AMIQP	Probable anterior infarct, age indeterminate.....Q >30mS, T neg, V2-V5	New statement
ASMIQ	Anteroseptal infarct, age indeterminate.....Q >35mS, T neg, V1-V3	
AMIQ	Anterior infarct, age indeterminate.....Q >30mS, T neg, in V2-V5	
ASMIRP	Probable anteroseptal infarct, recent.....Q, ST>0.15mV, T neg, V1-V3	New statement
AMIRP	Probable anterior infarct, recent.....Q >30mS, ST >0.15mV, T neg, V2-V5	AMI52
ASMIR	Anteroseptal infarct, recent....ST >0.15mS, T neg, V1-V3	AMI26
AMIR	Anterior infarct, recent.Q >30mS, ST >0.15mV, T neg, V2-V5	AMI66
ASMIPA	Anteroseptal infarct, possibly acute.....Q>30mS, ST>0.15mV, V1-V3	AMI10
ASMIAP	Probable anteroseptal infarct, acute.....ST>0.15mV, T upright, V1-V3	AMI21A
AMIAP	Probable anterior infarct, acute.....ST >0.15mV, upright T, V2-V5	AMI50
AMIPA	Anterior infarct, possibly acute.....ST >0.15mV, upright T, V2-V5	AMI61A
ASMIA	Anteroseptal infarct, acute.....ST >0.25mV, V1-V3	
ASMIAD	Anteroseptal infarct, acute (LAD).....ST >0.25mV, V1-V3	New statement
AMIA	Anterior infarct, acute.....ST >0.25mV, V2-V5	
AMIAD	Anterior infarct, acute (LAD).....ST >0.25mV, V2-V5	New statement

Anterolateral and Extensive Anterior Myocardial Infarct

Table B-30 Anterolateral and Extensive Anterior Myocardial Infarct

Anterolateral and Extensive Anterior Myocardial Infarct Statements		
Statement Code	Interpretive Statement	Notes
ALIC	Consider anterolateral infarct.....Q >30mS, I aVL V3-V6	ALI10
ALIOP	Probable anterolateral infarct, old.....Q >30mS, abnormal ST-T, V2-V6	ALI24
ALQLVH	Anterolateral Q waves, probably due to LVH.....Q >35mS in V4-V6 & LVH	ALI48
ALQNV	Anterolateral Q wave, probably normal for age...Q >35mS, age<31 male, <40 female	ALI49
EAMIO	Extensive anterior infarct, old.....Q >35mS, V1-V6	ALI86
ALIO	Anterolateral infarct, old.....Q>35mS, abnrm ST-T, V3-V6	ALI64
ALIQP	Probable anterolateral infarct, age indeterm.....Q >30mS, T neg, V2-V6	ALI26
EAMIQ	Extensive anterior infarct, age indeterminate.....Q >35mS, flat/neg T, V1-V6	
ALIQ	Anterolateral infarct, age indeterminate.....Q >35mS & >0.10mV in V3-V6	
ALIRP	Probable anterolateral infarct, recent.....Q >30mS, ST >0.07mV, T neg, V2-V6	ALI54
EAMIR	Extensive anterior infarct, recent.....Q >35mS, ST >0.10mV, T neg, V1-V6	ALI94
ALIR	Anterolateral infarct, recent.....Q >35mS, ST >0.10mV, T neg, V2-V6	
ALIAP	Probable anterolateral infarct, acute.....ST >0.15mV, V2-V5	ALI50
EAMIPA	Extensive anterior infarct, possibly acute.....Q >35mS, ST >0.15mV, V1-V6	ALI88
ALIPA	Anterolateral infarct, possibly acute.....Q >35mS, ST >0.15mV, V2-V6	ALI67
EAMIA	Extensive anterior infarct, acute.....ST >0.15mV, V1-V6	
EAMIAD	Extensive anterior infarct, acute (LAD)....ST >0.15mV, V1-V6	New statement

Table B-30 Anterolateral and Extensive Anterior Myocardial Infarct *(continued)*

Anterolateral and Extensive Anterior Myocardial Infarct Statements		
Statement Code	Interpretive Statement	Notes
ALIA	Anterolateral infarct, acute.....Q >35mS, ST >0.20mV, V2-V6	
ALIAD	Anterolateral infarct, acute (LAD).....Q >35mS, ST >0.20mV, V1-V6	New statement

ST Depression and Ischemia

Table B-31 ST Depression and Ischemia

ST Depression and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
NDSTD	Nondiagnostic ST depression	Statement added only at editing
SDANP	Nonspecific ST depression, anterior leads.....ST <-0.10mV, V2-V5	
SDINP	Nonspecific ST depression, inferior leads.....ST <-0.10mV, II III aVF	
SDALP	Nonspecific ST depression, anterolateral lds.....ST <-0.10mV, I aVL V2-V6	
SDJ	Junctional ST depression.....ST <-0.10mV any 3 leads	
SDM	Minimal ST depression.....ST <-0.05mV in 2 leads	
SDCU	Minimal ST depression.....ST concave upward	
SD0NS	Minimal ST depression.....ST <-0.04mV, T neg, any 2 leads	
SD0AN	Minimal ST depression, anterior leads....ST <-0.03mV, V2-V4	
SD0LA	Minimal ST depression, lateral leads.....ST <-0.04mV, I aVL V5 V6	
SD0AL	Minimal ST depression, anterolateral leads.....ST <-0.04mV, I aVL V2-V6	
SD0IN	Minimal ST depression, inferior leads.....ST <-0.04mV, II III aVF	

Table B-31 ST Depression and Ischemia *(continued)*

ST Depression and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
SD0DI	Minimal ST depression, diffuse leads.....ST <-0.03mV, ant/lat/inf	
SD1AN	Borderline ST depression, anterior leads.....ST <-0.07mV, V2-V4	
SD1LA	Borderline ST depression, lateral leads.....ST <-0.07mV, I aVL V5 V6	
SD1AL	Borderline ST depression, anterolateral leads.....ST <-0.07mV, I aVL V2-V6	
SD1IN	Borderline ST depression, inferior leads.....ST <-0.07mV, II III aVF	
SD1DI	Borderline ST depression, diffuse leads.....ST <-0.07mV, ant/lat/inf	
SD15NS	Nonspecific ST depression.....ST <-0.10mV any 2 leads	
SD15AN	Nonspecific ST depression, anterior leads.....ST <-0.10mV, V2-V4	
SD15LA	Nonspecific ST depression, lateral leads.....ST <-0.10mV, I aVL V5 V6	
SD15AL	Nonspecific ST depression, ant-lat leads.....ST <-0.10mV, I aVL V2-V6	
SD15IN	Nonspecific ST depression, inferior leads.....ST <-0.10mV, II III aVF	
SD15WI	Nonspecific ST depression, diffuse leads.....ST <-0.10mV, ant/lat/inf	
SD2NS	Nonspecific ST depression.....ST <-0.10mV, any 2 leads	
SD2AN	ST depression, consider ischemia, ant leads.....ST <-0.10mV, V2-V4	
SD2LA	ST depression, consider ischemia, lat leads.....ST <-0.10mV, I aVL V5 V6	
SD2AL	ST depression, consider ischemia, ant-lat lds.....ST <-0.10mV, I aVL V2-V6	
SD2IN	ST depression, consider ischemia, inf leads.....ST <-0.10mV, II III aVF	

Table B-31 ST Depression and Ischemia *(continued)*

ST Depression and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
SD2WI	ST depression, consider ischemia, diffuse lds.....ST <-0.10mV, ant/lat/inf	
SDPRR	ST depression, probably rate related.....ST <-0.10mV & extreme tachycardia	

T Wave Abnormality and Ischemia

Table B-32 T Wave Abnormality and Ischemia

T Wave Abnormality and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
PUW	Prominent U waves	Statement added only at editing
INVU	Inverted U waves	Statement added only at editing
TUFUS	TU fusion	Statement added only at editing
TIN1	Abnormal T waves, inferior leads.....T neg, II III aVF	
TAS1	Abnormal T waves, anteroseptal leads.....T neg, V1 V2 V3	
TARVH	Abnormal T, prob secondary to RVH, ant leads.....RVH & T neg, V1-V3	
TAN1	Abnormal T waves, anterior leads.....T neg, V1-V5	
TLA1	Abnormal T waves, lateral leads.....T neg, I aVL V5-V6	
TAL1	Abnormal T waves, anterolateral leads.....T neg, I aVL V2-V6	
TALVH	Abnormal T, probably due to LVH, ant-lat lds.....LVH & T neg, I aVL V2-V6	
LOWT	Borderline T wave abnormalities.....flat T	
TAXAB	Borderline T wave abnormalities.....T axis not between (-10,100)	

Table B-32 T Wave Abnormality and Ischemia *(continued)*

T Wave Abnormality and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
TAXQT	Borderline T wave abnormalities.....QRS-T axis angle (91,180)	
T0NS	Borderline T wave abnormalities.....T/QRS ratio < 1/20 or flat T	
T0AN	Borderline T abnormalities, anterior leads.....T flat or neg, V2-V4	
T0LA	Borderline T abnormalities, lateral leads.....T flat/neg, I aVL V5 V6	
T0AL	Borderline T abnormalities, ant-lat leads.....T flat/neg, I aVL V2-V6	
T0IN	Borderline T abnormalities, inferior leads.....T flat/neg, II III aVF	
T0DI	Borderline T abnormalities, diffuse leads.....T flat/ neg	
T1AN	Nonspecific T abnormalities, anterior leads.....T <- 0.10mV, V2-V4	
T1LA	Nonspecific T abnormalities, lateral leads.....T <- 0.10mV, I aVL V5 V6	
T1AL	Nonspecific T abnormalities, ant-lat leads.....T <- 0.10mV, I aVL V2-V6	
T1IN	Nonspecific T abnormalities, inferior leads.....T <- 0.10mV, II III aVF	
T1DI	Nonspecific T abnormalities, diffuse leads.....T <- 0.10mV, ant/lat/inf	
T3AN	Abnormal T, consider ischemia, anterior leads.....T <- 0.20mV, V2-V4	
TIALVH	LVH w/ repol abnormalities, possible ischemia....T <- 0.20mV, V1-V3 & LVH	
T3LA	Abnormal T, consider ischemia, lateral leads.....T <- 0.20mV, I aVL V5 V6	
T3AL	Abnormal T, consider ischemia, ant-lat leads.....T <- 0.20mV, I aVL V2-V6	
T3IN	Abnormal T, consider ischemia, inferior leads.....T <- 0.20mV, II III aVF	

Table B-32 T Wave Abnormality and Ischemia *(continued)*

T Wave Abnormality and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
T3WI	Abnormal T, consider ischemia, diffuse leads.....T <- 0.20mV, ant/lat/inf	
T6AN	Abnormal T, consider ischemia, anterior leads.....T <- 0.50mV, V2-V4	
T6LA	Abnormal T, consider ischemia, lateral leads.....T <- 0.50mV, I aVL V5 V6	
T6AL	Abnormal T, consider ischemia, anterolateral leads.....T <-0.50mV, I aVL V2-V6	
T6IN	Abnormal T, consider ischemia, inferior leads.....T <- 0.40mV, II III aVF	
T6IL	Abnormal T, consider ischemia, inferolateral.....T <- 0.40mV, I-III aVL aVF V5-6	
T6WI	Abnormal T, consider ischemia, widespread.....T <- 0.50mV, ant/lat/inf	

Repolarization Abnormality and Ischemia

Table B-33 Repolarization Abnormality and Ischemia

Repolarization Abnormality and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
REPB	Borderline repolarization abnormality....ST dep & abnormal T	
REPBAN	Borderline repol abnormality, ant leads.....ST dep, T flat/neg, V2-V4	
REPBLA	Borderline repol abnormality, lateral leads...ST dep, T flat/neg, I aVL V5 V6	
REPBAL	Borderline repol abnormality, ant-lat leads.....ST dep, T flat/neg, I aVL V2-V6	
REPBIN	Borderline repol abnormality, inferior leads.....ST dep, T flat/neg, II III aVF	
REPBIL	Borderline repol abnormality, inf-lat leads.....ST dep, T flat/neg, inf/lat	

Table B-33 Repolarization Abnormality and Ischemia *(continued)*

Repolarization Abnormality and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
REPBDI	Borderline repol abnormality, diffuse leads...ST dep, T flat/neg, ant/lat/inf	
REPNS	Nonspecific repolarization abnormalities.....ST dep, T neg, 2-3 leads	
REPAN	Nonspecific repol abnormality, anterior leads.....ST dep, T neg, V2-V4	
REPLA	Nonspecific repol abnormality, lateral leads.....ST dep, T neg, I aVL V5 V6	
REPAL	Nonspecific repol abnormality, ant-lat leads.....ST dep, T neg, I aVL V2-V6	
REPLVH	Repol abnormality probably secondary to LVH.....ST dep, T neg, I aVL V2-V6	
REPIN	Nonspecific repol abnormality, inferior leads.....ST dep, T neg, II III aVF	
REPIL	Nonspecific repol abnormality, inf-lat leads...ST dep, T neg, I-III aVL aVF V5-6	
REPDI	Nonspecific repol abnormality, diffuse leads..ST dep, T flat/neg, ant/lat/inf	
REPIA	Repol abnrm suggests ischemia, anterior leads.....ST dep, T neg, V2-V4	
REPILA	Repol abnrm suggests ischemia, lateral leads.....ST dep, T neg, I aVL V5 V6	
REPIAL	Repol abnrm suggests ischemia, ant-lat leads.....ST dep, T neg, I aVL V2-V6	
REPII	Repol abnrm suggests ischemia, inferior leads.....ST dep, T neg, II III aVF	
REPIIL	Repol abnrm suggests ischemia, inferolateral...ST dep, T neg, I-III aVL aVF V5-6	
REPIDI	Repol abnrm suggests ischemia, diffuse leads.....ST-T neg, ant/lat/inf	
REPPAN	Repol abnrm, consider ischemia, anterior leads.....ST dep, T neg, V2-V4	

Table B-33 Repolarization Abnormality and Ischemia *(continued)*

Repolarization Abnormality and Ischemia Statements		
Statement Code	Interpretive Statement	Notes
REPPLA	Repol abnrm, consider ischemia, lateral leads.....ST dep, T neg, I aVL V5 V6	
REPPAL	Repol abnrm, consider ischemia, anterolateral lds.....ST dep, T neg, I aVL V2-V6	
REPPIN	Repol abnrm, consider ischemia, inferior leads.....ST dep, T neg, II III aVF	
REPPIL	Repol abnrm, consider ischemia, inferolateral lds...ST dep, T neg, I-III aVL aVF V5-6	
REPPWI	Repol abnrm, global ischemia, diffuse leads.....ST dep, T neg, ant/lat/inf	
LMVD	Repol abnrm, severe global ischemia (LM/VD).....STe aVR, STd & Tneg, ant/lat/inf	New statement
REPRR	Repolarization abnormality, prob rate related.....ST dep, T neg, tachycardia	

ST Elevation, Pericarditis, Early Repolarization and Injury

Table B-34 ST Elevation, Pericarditis, Early Repolarization, and Injury

ST Elevation, Pericarditis, Early Repolarization, and Injury Statements		
Statement Code	Interpretive Statement	Notes
STEND	Nondiagnostic ST elevation	Statement added only at editing
STE	ST elevation, subepicardial injury	Statement added only at editing
STBRUG	ST elevation suggests Brugada abnormality	Statement added only at editing
CCNS	Suggest CNS disease	Statement added only at editing

Table B-34 ST Elevation, Pericarditis, Early Repolarization, and Injury *(continued)*

ST Elevation, Pericarditis, Early Repolarization, and Injury Statements		
Statement Code	Interpretive Statement	Notes
COPASD	Suggest Ostium primum ASD	Statement added only at editing
CPEFUS	Suggest pericardial effusion	Statement added only at editing
CLVAN	Consider left ventricular aneurysm	Statement added only at editing
SEANP	ST elev, probably normal variation, ant leads.....ST>0.15mV, V2-V5	
SEINP	ST elevation, probably normal variation, inf.....ST>0.15mV, II III aVF	
SEALP	ST elevation, prob normal variation, ant-lat.....ST >0.15 mV, I aVL V2-V6	
MSTEA	Minimal ST elevation, anterior leads.....ST >0.10mV, V1-V4	
MSTEL	Minimal ST elevation, lateral leads.....ST >0.06mV, I aVL V5 V6	
MSTEAL	Minimal ST elevation, anterolateral leads.....ST >0.08mV, I aVL V2-V6	
MSTEI	Minimal ST elevation, inferior leads....ST >0.06mV, II III aVF	
MSTED	Minimal ST elevation, diffuse leads....ST >0.10mV, ant/lat/inf	
BSTE	Borderline ST elevation.....ST >0.10 mV in 2 leads	
BSTEA	Borderline ST elevation, anterior leads.....ST >0.15mV in V1-V4	
STELVH	Anterior ST elevation, probably due to LVH.....ST >0.20 mV in V1-V4 & LVH	
BSTEL	Borderline ST elevation, lateral leads....ST >0.06mV, I aVL V5 V6	
BSTEAL	Borderline ST elevation, anterolateral leads.....ST >0.06mV, I aVL V2-V6	

Table B-34 ST Elevation, Pericarditis, Early Repolarization, and Injury *(continued)*

ST Elevation, Pericarditis, Early Repolarization, and Injury Statements		
Statement Code	Interpretive Statement	Notes
BSTEI	Borderline ST elevation, inferior leads...ST >0.06mV, II III aVF	
PERI	ST elevation suggests acute pericarditis.....ST >0.06mV, ant/lat/inf	
CINJI	ST elevation, consider inferior injury.....ST >0.08mV, II III aVF	
CINJA	ST elevation, consider anterior injury.....ST >0.15mV, V1-V5	
CINJL	ST elevation, consider lateral injury.....ST >0.10mV, I aVL V5 V6	
CINJAL	ST elevation, consider anterolateral injury.....ST >0.15mV, I aVL V2-V6	
EREPOL	ST elev, probable normal early repol pattern.....ST elevation, age<55	
PERI1	ST elevation suggests acute pericarditis.....ST >0.10mV, ant/lat/inf	

Lateral Leads Involved

Table B-35 Lateral Leads Involved

Lateral Leads Involved Statement		
Statement Code	Interpretive Statement	Notes
LLINV	Lateral leads are also involved.....lat Q or ST-T abnormalities	

Tall T Waves

Table B-36 Tall T Waves

Tall T Waves Statements		
Statement Code	Interpretive Statement	Notes
TTW	Tall T waves	Statement added only at editing
TTW1	Tall T, probably normal variant, ant-lat lds.....T >1.0mV, I aVL V2-V6	
TTW10	Tall T, consider metabolic/ischemic abnrm.....T >1.2mV	
TTW20	Tall T waves suggest hyperkalemia.....widespread tall T	
TTW30	Tall T waves, probably normal variant....T >1.2mV, age 16-30	
TWALT	T wave alternans	Statement added only at editing

QT Interval, Electrolyte and Drug Effects

Table B-37 QT Interval, Electrolyte and Drug Effects

QT Interval, Electrolyte and Drug Effects Statements		
Statement Code	Interpretive Statement	Notes
PDGTOX	Suggest digitalis toxicity	Statement added only at editing
SQT	Short QT interval.....QTc <340mS	
HPRCA	Short QT interval suggests hypercalcemia.....QTc <310mS	
LQTB	Borderline prolonged QT interval.....QTc > ** mS	
LQTS	Prolonged QT, probably secondary to wide QRS.....QTc > ** mS w/ VCD/RVH/LVH	
LQT	Prolonged QT interval.....QTc > ** mS	
HPOCA	Prolonged QT interval suggests hypocalcemia.....QTc >520mS	

Table B-37 QT Interval, Electrolyte and Drug Effects *(continued)*

QT Interval, Electrolyte and Drug Effects Statements		
Statement Code	Interpretive Statement	Notes
HPOK	Prolonged QT suggests hypokalemia/drug.....QTc >520mS & ST-T abnormalities	
DIG1	Repol abnormality suggests digitalis effect.....short QTc & negative ST	
DIG2	Repol abnormality suggests digitalis effect.....ST concave upward & digitalis	
DIG3	Repol abnormality suggests digitalis effect.....ST-T negative & digitalis	

Pediatric Congenital Heart Defects

Table B-38 Pediatric Congenital Heart Defects

Pediatric Congenital Heart Defects Statements		
Statement Code	Interpretive Statement	Notes
ARVO	Acute right ventricular overload	Statement added only at editing
ACP	Acute cor pulmonale	Statement added only at editing
ASD	Atrial septal defect	Statement added only at editing
AVSD	Atrioventricular septal defect	Statement added only at editing
CHCM	Suggest hypertrophic cardiomyopathy	Statement added only at editing
CTA	Consider tricuspid atresia	Statement added only at editing
CECD	Consider endocardial cushion defect	Statement added only at editing

Table B-38 Pediatric Congenital Heart Defects *(continued)*

Pediatric Congenital Heart Defects Statements		
Statement Code	Interpretive Statement	Notes
CASD	Consider atrial septal defect, septum secundum	Statement added only at editing
CAOCA	Probable ant-lat infarct, consider anomalous origin of the coronary artery	Statement added only at editing
CEA	Consider Ebstein anomaly	Statement added only at editing

Right Precordial Leads

Table B-39 Right Precordial Leads

Right Precordial Leads Statement		
Statement Code	Interpretive Statement	Notes
V4R	Acute IMI, suggest recording right precordial leads	New statement

Lead(s) Not Used for Analysis

Table B-40 Leads Not Used for Analysis

Leads Not Used for Analysis Statement		
Statement Code	Interpretive Statement	Notes
QMA04	Lead(s) ** were not used for morphology analysis	

Quality Monitor Codes: Artifact and Wander

Table B-41 Quality Monitor Codes: Artifact and Wander

Quality Monitor Codes: Artifact and Wander Statements		
Statement Code	Interpretive Statement	Notes
QMART	Artifact in lead(s) **	

Table B-41 Quality Monitor Codes: Artifact and Wander *(continued)*

Quality Monitor Codes: Artifact and Wander Statements		
Statement Code	Interpretive Statement	Notes
QMBW	Baseline wander in lead(s) **	
QMAB	Artifact in lead(s) ** and baseline wander in lead(s) **	

Quality Monitor Codes: Missing Leads

Table B-42 Quality Monitor Codes: Missing Leads

Quality Monitor Codes: Artifact and Wander Statements		
Statement Code	Interpretive Statement	Notes
QMRGT	Right-sided precordial electrode(s)	Statement added only at editing
QMPST	Posterior electrode(s)	Statement added only at editing
QMPML	Partial lead(s): ***	New statement
QMMLD	Missing lead(s): ***	
QMML	Missing lead(s): *** and partial lead(s): **	New statement

Critical Value Statements

Table B-43 Critical Value Statements

Critical Value Statements		
Statement Code	Interpretive Statement	Notes
CMPLHB	>>> COMPLETE HEART BLOCK <<<	New statement
XTACH	>>> EXTREME TACHYCARDIA <<<	New statement
ACUISC	>>> ACUTE ISCHEMIA <<<	New statement

Table B-43 Critical Value Statements *(continued)*

Critical Value Statements		
Statement Code	Interpretive Statement	Notes
ACUTMI	>>> ACUTE MI <<<	New statement

C

Suppressed Borderline Interpretive Statements

Introduction

This Appendix includes a listing of all borderline interpretive statements that are suppressed using the *Borderline Statement Suppression* feature that is available with the Philips DXL ECG Algorithm. This feature is used to exclude interpretive statements from appearing on the ECG report that indicate a borderline or otherwise normal condition. Borderline interpretive statements are generated by measurements that are above an abnormal threshold, but may in fact indicate a non-pathological condition. These statements indicate to the clinician that a condition may be present, but there is no decisive indicator. These statements often include the terms “minimal,” “consider,” or “borderline.”

Exclude Low Certainty Suppressed Statements

The following interpretive statements listed in Table C-1 are suppressed when the **Exclude Low Certainty** setting is selected.

Table C-1 Exclude Low Certainty - Suppressed Statements

Statement Code	Interpretive Statement
BAVCD	Borderline prolonged PR interval.....PR > ** , V-rate ** - **
BIVCD	Borderline intraventricular conduction delay.....QRSd > ** mS
CLAE	Consider left atrial enlargement.....wide or notched P waves
CRAE	Consider right atrial enlargement.....P >0.24mV limb lead
ET	Abnormal R-wave progression, early transition.....QRS area>0 in V2
ETRSR1	RSR′ in V1 or V2, right VCD or RVH.....QRS area positive & R′ V1/V2
LOWT	Borderline T wave abnormalities.....flat T

Table C-1 Exclude Low Certainty - Suppressed Statements *(continued)*

Statement Code	Interpretive Statement
LT	Abnormal R-wave progression, late transition.....QRS area <0 in V5/V6
LVHQ	Consider left ventricular hypertrophy.....deep Q in V5-6 or II III aVF
LVOLFB	Borderline low voltage, extremity leads.....all extremity leads <0.6mV
NFAD	No further analysis attempted due to paced rhythm
NFRA	No further rhythm analysis attempted due to paced rhythm
QMAB	Artifact in lead(s) ** and baseline wander in lead(s) **
QMART	Artifact in lead(s) **
QMBW	Baseline wander in lead(s) **
REPB	Borderline repolarization abnormality....ST dep & abnormal T
RSR1	RSR′ in V1 or V2, probably normal variant.....small R′ only
RSRNV	RSR′ in V1, normal variation.....term-vector post-rightward
SDALP	Nonspecific ST depression, anterolateral lds.....ST <-0.10mV, I aVL V2-V6
SDANP	Nonspecific ST depression, anterior leads.....ST <-0.10mV, V2-V5
SDCU	Minimal ST depression.....ST concave upward
SDINP	Nonspecific ST depression, inferior leads.....ST <-0.10mV, II III aVF
SDJ	Junctional ST depression.....ST <-0.10mV any 3 leads
SDM	Minimal ST depression.....ST <-0.05mV in 2 leads
SEALP	ST elevation, prob normal variation, ant-lat.....ST >0.15 mV, I aVL V2-V6
SEANP	ST elev, probably normal variation, ant leads.....ST>0.15mV, V2-V5
SEINP	ST elevation, probably normal variation, inf.....ST>0.15mV, II III aVF
SPRB	Borderline short PR interval.....PR int < ** mS
TAXAB	Borderline T wave abnormalities...T axis not between (-10,100)

Table C-1 Exclude Low Certainty - Suppressed Statements *(continued)*

Statement Code	Interpretive Statement
TAXQT	Borderline T wave abnormalities.....QRS-T axis angle (91,180)
TTW1	Tall T, probably normal variant, ant-lat lds.....T >1.0mV, I aVL V2-V6

Exclude All Suppressed Statements

The interpretive statements listed in Table C-1, “Exclude Low Certainty - Suppressed Statements,” on page C-1, and the interpretive statements listed in Table C-2, “Exclude All Setting - Suppressed Statements,” on page C-3, are suppressed when the **Exclude ALL** setting is selected.

Table C-2 Exclude All Setting - Suppressed Statements

Statement Code	Interpretive Statement
ANTQ	Abnormal Q wave in V1.....Q >15mS in V1
AXL	Borderline left-axis deviation.....QRS axis (** , **)
AXR	Borderline right-axis deviation.....QRS axis (** , **)
BIVCDL	Borderline IVCD with LAD.....QRSd > ** mS, axis(-90,-30)
CRHPI	Consider RVH or posterior infarct.....large R in V1
CRHPIR	Consider RVH or PMI w/ sec repol abnormality....large R V1, repol abnormality
RPMIC	Tall R wave in V2, consider RVH or PMI.....R/S ratio >3, T >0.30mV V1 V2
CRVH	Consider right ventricular hypertrophy.....large R or R′ V1/ V2
INFQ	Abnormal inferior Q waves.....Qs add to 80 mS in II III aVF
IQNV	Inferior Q waves, probably normal variation.... Q >30mS, age<21 male, <30 female
IRBBRV	IRBBB, the RSR′ pattern may also reflect RVH...IRBBB, R or R′ >0.5mV in V1-V3
LATQ	Abnormal lateral Q waves.....Q >35mS, I aVL V5 V6
LQNV	Lateral Q waves, probably normal variation...Q >35mS, age<31 male, <40 female
LQTB	Borderline prolonged QT interval.....QTc > ** mS

Table C-2 Exclude All Setting - Suppressed Statements *(continued)*

Statement Code	Interpretive Statement
LVHR56	LVH by voltage.....R >*.***mV in V5 or V6
LVHR6	LVH by voltage.....R >*.***mV in V6
LVHRS	Consider left ventricular hypertrophy.....RV6+SV1 >*.***mV
LVHRSI	LVH by voltage.....(R I+S III) >*.***mV
LVHS12	LVH by voltage.....S <*.*** in V1 or *.*** in V2
LVHTA	Consider left ventricular hypertrophy...prominent leftward forces
LVHV	LVH by voltage.....R >*.*** in aVL
MSTEA	Minimal ST elevation, anterior leads.....ST >0.10mV, V1-V4
MSTEAL	Minimal ST elevation, anterolateral leads.....ST >0.08mV, I aVL V2-V6
MSTEI	Minimal ST elevation, inferior leads....ST >0.06mV, II III aVF
MSTEL	Minimal ST elevation, lateral leads...ST >0.06mV, I aVL V5 V6
PLAE	Probable left atrial enlargement.....P >50mS, <-0.10mV V1
PQAL	Borderline Q wave in anterolateral leads.....Q >35mS, I aVL V3-V6
PQAN	Borderline Q wave in anterior leads.....Q >30mS in V2-V5
PQIN	Borderline Q waves in inferior leads.....Qs add to 80 mS in II III aVF
PQLA	Borderline Q waves in lateral leads...Q >35mS in I aVL V5 V6
PRAE	Probable right atrial enlargement....biphasic P >0.20 mV in V1
REPBAL	Borderline repol abnormality, ant-lat leads.....ST dep, T flat/neg, I aVL V2-V6
REPBAN	Borderline repol abnormality, ant leads.....ST dep, T flat/neg, V2-V4
REPBIL	Borderline repol abnormality, inf-lat leads.....ST dep, T flat/neg, inf/lat
REPBIN	Borderline repol abnormality, inferior leads.....ST dep, T flat/neg, II III aVF
REPBLA	Borderline repol abnormality, lateral leads...ST dep, T flat/neg, I aVL V5 V6

Table C-2 Exclude All Setting - Suppressed Statements *(continued)*

Statement Code	Interpretive Statement
RVHS5	Consider right ventricular hypertrophy.....deep S in V5
RVHS6	Consider right ventricular hypertrophy.....deep S in V6
SD0AL	Minimal ST depression, anterolateral leads.....ST <-0.04mV, I aVL V2-V6
SD0AN	Minimal ST depression, anterior leads....ST <-0.03mV, V2-V4
SD0IN	Minimal ST depression, inferior leads...ST <-0.04mV, II III aVF
SD0LA	Minimal ST depression, lateral leads.....ST <-0.04mV, I aVL V5 V6
SD0NS	Minimal ST depression.....ST <-0.04mV, T neg, any 2 leads
SPR	Short PR interval.....PR < ** mS
SQT	Short QT interval.....QTc <340mS
T0AL	Borderline T abnormalities, ant-lat leads.....T flat/neg, I aVL V2-V6
T0AN	Borderline T abnormalities, anterior leads...T flat or neg, V2-V4
T0IN	Borderline T abnormalities, inferior leads.....T flat/neg, II III aVF
T0LA	Borderline T abnormalities, lateral leads.....T flat/neg, I aVL V5 V6
T0NS	Borderline T wave abnormalities.....T/QRS ratio < 1/20 or flat T
TTW30	Tall T waves, probably normal variant....T >1.2mV, age 16-30

D

Validation of the DXL ECG Algorithm

NOTE Validation data for the PH110C algorithm version starts on page D-26.

Utility of Automated ECG Interpretation

Automated ECG interpretation has been in use for over 40 years. The initial motivation for developing automatic interpretation varied from practical help for overreaders to the research aim of replicating human pattern recognition. While the latter goal has clearly not been met, continuing use of automated interpretation systems is the best proof that time is saved and resulting interpretations are improved.

Automatic interpretation provides several major benefits:

- Automated measurements are usually better than manual measurements[1]
- Uniform application of criteria
- Uniform adjustment of criteria for important variables such as age and sex, especially important for pediatric ECG interpretation
- Analysis of more data than is usually displayed. Typical presentations show 2.5 seconds of each lead. The computer has access to 10 seconds of each lead.

Automated interpretation is not perfect for the following reasons:

- Important clinical information is unknown to the program
- Population information is unknown to the program
- Artifacts can mimic true ECG signals and confuse the program
- Artifacts can interfere with measurements and cause unreliable data to be analyzed

1. Laguna P, Thakor NV, Caminal P, Jane R, Yoon HR, Bayes de Luna A, et al. "New algorithm for QT interval analysis in 24-hour Holter ECG: performance and applications." *Medical and Biological Engineering and Computing* 28:67-73, 1990. Algra A, le Brun H, Zeelenberg C. "An algorithm for computer measurement of QT intervals in the 24 hour ECG." *Computers in Cardiology* 1986. Los Alamitos: IEEE Computer Society Press, 117-119 1987. Ahnve S. "Errors in the visual determination of corrected QT (QTc) interval during acute myocardial infarction." *Journal of the American College of Cardiology* 5:699-702, 1985. Savelieva I, Yi G, Guo X, Hnatkova K, Malik M. "Agreement and Reproducibility of Automatic Versus Manual Measurement of QT Interval and QT Dispersion." *American Journal of Cardiology* 81:471-477, 1998.

- Mixtures of rhythms do not provide enough data to make multiple diagnoses
- Very rare phenomena do not occur frequently enough to allow development of automatic criteria

Comparisons of automated interpretation with physicians' readings show that experienced physicians usually perform better. The computer sometimes picks up diagnoses that are missed by even experienced readers, so the computer approach is at least complementary.

For these reasons, the ACC/AHA recommendation is for all ECGs to be reviewed by a qualified physician. Available references[2] summarizes results of computer comparisons as well as defining ECG clinical competence.

Purpose of this Appendix

This appendix describes the various approaches to validation used for the development of the Philips DXL ECG Algorithm. It provides an explanation of several methods as well as the results of applying those methods.

Types of Validation

No single approach to validation is sufficient to develop a complex program such as the DXL Algorithm. At least three separate types are necessary, described in the sections that follow.

- Artificial signal
- Anatomical
- Expert reader

Artificial Signal Validation

The first approach to validation is concerned only with simple measurements. Artificial signals of known amplitude and duration are presented to the cardiograph/algorithm and the resulting measurements are compared to the known values. This approach has been proposed as one of the main techniques by IEC 60601-2-51. The results of this approach are detailed in "Artificial Signals" on page D-7.

2. Kadish AH, Buxton AE, Kennedy HL, Knight BP, Mason JW, Schuger CD, Tracy CM. "ACC/AHA clinical competence statement on electrocardiography and ambulatory electrocardiography: a report of the American College of Cardiology/American Heart Association/American College of Physicians-American Society of Internal Medicine Task Force on Clinical Competence (ACC/AHA Committee to Develop a Clinical Competence Statement on Electrocardiography and Ambulatory Electrocardiography)." *Journal of the American College of Cardiology* 38:2091–100, 2001.

Anatomical Validation

Although this would seem to be the best form of validation, anatomical validation has several weaknesses:

- Some ECG diagnoses have no clear anatomical correlate (e.g., RBBB, rhythms)
- The ECG reviewer must use visible criteria from the ECG and also does not usually have access to anatomical information
- Imaging methods usually show structural rather than functional problems
- Anatomical changes vary over time, particularly with myocardial infarctions, which can disappear as the scar retracts in size

Nevertheless, imaging methods are demonstrably better than ECG criteria for some conditions, such as ultrasound for LVH. Magnetic resonance imaging will probably add additional information in sizing infarcts. Anatomical methods often serve as the basis when developing new ECG criteria.

Expert Reader Validation

Since automated interpretation programs are in essence attempts to emulate an expert reader, using expert readers is the most common approach to validation. However, expert readers may introduce their own set of well documented issues[3]:

- Experts may disagree amongst themselves
- Experts may disagree with their own earlier diagnoses
- Experts sometimes hedge their diagnoses with either/or statements
- Expert interpretations may be difficult to implement; for example, statements that contain the phrases *consider*, *cannot exclude*, and *possible*

Signal Acquisition/Conditioning

Filtering

Some degree of filtering is unavoidable in any digital acquisition system. At the most basic level, effective filtering avoids distortion of the signal. Any additional filtering generally does cause some distortion, although this may be inconsequential. Heavy filtering can cause artificial ST segment deviation, loss of small notches, and some reduction in true amplitude. For this reason, the DXL Algorithm always analyzes unfiltered data, with the exception of AC filtering.

3. Salerno SM, Alguire PC, and Waxman HS. "Competency in Interpretation of 12-Lead Electrocardiograms: A Summary and Appraisal of Published Evidence." *Annals of Internal Medicine* 138:751-760, 2003.

AC Interference Filtering

It is difficult to entirely avoid AC interference. Special hardware removes most of the AC interference during acquisition using the right leg electrode to cancel common AC signals. Usually a small residual signal can be seen and this must be removed by software filters. Poor electrode contact often leaves a very large residual of AC interference. AC filtering usually does not cause significant distortion as the frequency removed (50 or 60 Hz) does not contribute significantly to the ECG signal.

Artifact Removal

Artifact removal is occasionally necessary for physiologic noise, such as muscle artifact due to breathing, or due to shivering and Parkinsonian tremors. Many artifacts may also be due to poor patient preparation, and to poor electrode contact with the skin. These patient preparation and electrode contact issues can usually be corrected with thorough skin preparation, however, the physiologic causes are mostly unavoidable.

Artifact removal filters are controlled by bandpass settings, which are used to determine the lowest and highest frequencies that will be displayed. Artifact removal filters do not affect the data recorded, which is always at full fidelity.

Artifact Detection

Baseline Wander

This type of artifact is typically the result of poor electrode contact. Slow oscillation of the baseline is superimposed on the ECG waveform. Often, this artifact will appear as a respiratory oscillation. It is difficult to remove this artifact without distortion, and this distortion can produce inaccurate measurements and result in the misapplication of criteria.

Muscle Artifact

Muscle artifact that originates from the intercostal muscles, such as shivering or tremors, is also very difficult to remove by filtering as the frequencies contained in this artifact and the frequencies of valid ECG signals often overlap. The most effective means of removal is in the formation of representative beats, as explained “Rationale for Representative Beats” on page D-5.

Spike Artifacts

Spike artifacts are similar to pacemaker artifacts, and occasionally appear as sudden changes in baseline voltage. These artifacts are best reduced by effective and thorough electrode site preparation.

Pacemaker Artifacts

Pacemaker artifacts are an important class of artifact since they can control or interfere with the basic rhythm of the heart. An effective algorithm must be able to differentiate these pacemaker artifacts from spike artifacts, and also differentiate pacemaker artifacts from the very narrow QRS complexes typical of neonates. This is the crux of the detection problem. The classification problem arises from trying to describe the relationships between the artificial pacemaker and the resulting cardiac rhythm.

Representative Beat Formation

Rationale for Representative Beats

ECG complexes in similar pattern are grouped into families for the purposes of rhythm classification and of forming averaged beats[4]. ECG complexes vary slightly from one complex to another. Nevertheless, a *typical* complex is selected for classification purposes. Because muscle artifact cannot easily be removed by filtering, it is preferable to average it out by aligning and adding up the ECG complexes. This process attempts to preserve all of the detail of the ECG complex while mitigating the random fluctuations generated by the muscle noise. When this process is applied to several hundred ECG complexes, it results in a Signal-Averaged ECG, which possesses very low residual noise. In a typical 10-second sample diagnostic ECG recording that contains fewer complexes, the application of this process will not achieve the same amount of muscle noise reduction.

Although it is noted that some clinicians prefer to avoid the term *beat* when describing an ECG complex, the usage is widespread, and for the purposes of this Appendix these terms are used interchangeably.

Ways of Forming Representative Beats

Median Selection

Once the complexes are aligned, the values are then sorted at each sample point, and a middle value is selected. This process derives the median point for each sample. When strung together, these points form the *median beat*. This technique is effective at mitigating extreme values, but it does not reduce the effect of small amplitude muscle noise, and may in fact even enhance it. Usually, median beats are filtered to make them appear smoother, however, this process eliminates most of the data, so it is not efficient from a statistical point of view.

4. Gregg R, Helfenbein E, Lindauer J, Zhou S. “Performance of a 12-Lead ECG Fuzzy Beat Classifier.” *Journal of Electrocardiology* 36:110 Supp, 2003. Gregg R, Helfenbein E, Lindauer J, Zhou S. “Automatic Discrimination of Atrial Flutter from Other Supraventricular Rhythms by Autocorrelation and Power Spectral Analysis.” *Journal of Electrocardiology* 37:79 Supp, 2004.

Mean Formation

The mean value is derived from aligning the complexes, and then taking the average at each sample point. Stringing these points together creates an *averaged representative beat*. This method is very sensitive to extreme values, however, this sensitivity can be removed by only considering complexes that are in the same template family. That is, all beats are compared against each other, and only the beats that appear similar are used. Because this approach uses all of the appropriate available data, it can be considered to be efficient. It automatically provides a smooth signal suitable for measurement while reducing the contribution of skeletal muscle noise. This approach is used by the DXL Algorithm.

Single Beat Selection

Both median and mean representative beats can be considered "artificial beats" in the sense that it is highly unlikely that any actual beat would exactly match. If a single actual beat is selected, it will be real, but it may also be corrupted by noise. For very clean ECGs with no physiologic variation, all three methods will produce an equivalent beat, however, these very clean ECGs are not very common.

DXL Algorithm Process

The DXL Algorithm uses the mean representative beat process to provide the cleanest possible measurements. But individual beat measurements are also made and the variation of these measurements is recorded. In the case of small amplitude signals with large respiratory variation, the representative beat is not very representative or reliable and there really is no representative beat in this situation. This information is used to exclude such a lead from consideration for the global measurements.

Measurements of Onsets, Offsets, Amplitudes and Areas

IEC 60601-2-51

Onset, offset, and amplitude measurements are validated according to the guidance from the IEC 60601-2-51 standard that provides requirements for essential performance of analyzing electrocardiographs. Three types of signals are specified for testing cardiographs that provide measurements. Two of the three are artificial signals that can be specified with mathematical functions so that their true measurement values are known. One set of artificial ECGs mimics true ECG signals, while the other set is designed mainly for testing extreme amplitude values. The third set comprises true biological ECGs collected and annotated as part of the CSE study[5].

Artificial Signals

- IEC-60601-2-51 section 50.101.2, requirements for amplitude measurements
- Amplitude measurements of P, Q, R, S, ST and T waveforms must be within 25μV for low amplitudes (< 500μV) and 5% of the true value for larger amplitudes
- The total number of tests (1,024) equals the number of ECGs (16) multiplied by 8 primary leads (I,II,V1-V6) per ECG multiplied by the number of waveforms measured (8)
- Table D-1 on page D-7 shows a pass condition for all amplitude tests

Table D-1 Pass Condition for All Amplitude Tests

Measurement (16 records X 8 leads per record = 128)	N PASS
P1 amplitude	128
P2 amplitude	128
Q amplitude	128
R amplitude	128
S amplitude	128
J amplitude	128
ST amplitude, 80ms from J	128
T amplitude	128

- IEC-60601-2-25 section 201.12.1, requirements for absolute interval and wave duration measurements
- Intervals and wave durations are measured for the artificial ECGs
- Differences between the measured and true values are tabulated by mean difference and standard deviation of the differences as listed in Table D-2 on page D-8

5. Willems JL, Arnaud P, van Bemmel JH, Degani R, Macfarlane PW, Zywietz C for the CSE Working Party. "Common Standards for Quantitative Electrocardiography: Goals and Main Results." *Methods of Information in Medicine* 29:263-271, 1990.

- All values are well within tolerance

Table D-2 Accuracy of Absolute Interval and Wave Duration Measurements

Measurement	Mean difference (ms)	Acceptable difference (ms)	Error SD (ms)	Acceptable SD (ms)	N
P duration	-1.8	+/- 10	0.9	8	12
PR interval	-3.6	+/- 10	0.7	8	12
QRS duration	3.3	+/- 6	0.8	5	12
QT interval	0.7	+/- 12	1.4	10	12
Q duration	0.0	+/- 6	1.4	5	124
R duration	1.2	+/- 6	1.7	5	124
S duration	-2.4	+/- 6	2.5	5	92

Expert Annotated Biological Signals

- IEC-60601-2-25 section 201.12.1, requirements for interval measurements on biological ECGs
- P wave duration, PR interval, QRS duration and QT interval are measured on all 100 biological ECGs
- The differences between the expert annotation and the measured values are given in Table D-3 on page D-8, along with the acceptable performance limits
- The mean difference and standard deviation of the differences are well within the acceptable performance limits

Table D-3 Expert Annotated Biological Signals

Measurement	Mean difference (ms)	Acceptable difference (ms)	Difference SD (ms)	Acceptable SD (ms)	N
P duration	2.1	+/- 10	5.1	15	92
PR interval	0.1	+/- 10	5.1	10	92
QRS duration	-0.7	+/- 10	4.2	10	92
QT interval	1.5	+/- 25	7.7	30	92

Pacemaker ECGs

Analysis of ECGs from patients with implanted pacemakers involves two main steps: pacemaker pulse detection, and paced rhythm classification.

Pacemaker Pulse Detection

The accurate detection of pacemaker pulse locations is required in order for pulse removal to occur prior to QRS detection, and is also required for classifying the paced status of each beat. This classification of the paced status of each beat forms the basis for paced rhythm classification, and also allows morphology analysis on atrial-paced and non-paced beats, in addition to the ability to identify proper pacemaker function or malfunction.

The DXL Algorithm uses a patented software pulse detection method to identify the location of pacemaker pulses in the ECG[6]. The detector is run on each of the 12 leads of data, and a multi-channel resolver is used to intelligently combine detections from the individual leads.

For pacemaker pulse detection testing, ECGs were randomly selected from a database of 1,108 adult ECGs that contained a variety of pacemaker types and pacing modes. This database contained 61 cases with atrial pacing, 746 cases with ventricular pacing, and 301 cases with dual chamber pacers. The ECG records contained varying amount of noise and artifact. The global locations of 16,029 true pacemaker pulses across the multi-channel, 10-second ECGs were digitally annotated after visual examination using both 12-lead simultaneous rhythm strip printouts, and a high resolution waveform display and annotation program.

For detection of individual pulses in the pacemaker pulse database, the pulse detection and multi-lead resolver algorithm achieved a sensitivity of 99.7% with a positive predictive value of 99.5%. The 54 out of 16,029 pulses missed were usually due to periods of muscle or electrode noise that inhibited the detector, or were extremely small atrial pulses. Of the 77 false positive detections, there were no false detections on adult QRS complexes. A few false positive detections did occur on muscle tremor spikes which managed to trigger the detector, but most were on sharp, isolated noise spikes of unknown origin. About half of these occurred during noise spike trains that were probably secondary to bad electrode contact. The remainder occurred from isolated noise spikes.

False pulse detection on narrow neonatal or pediatric QRS complexes is a potential problem for pacemaker pulse detection algorithms. When the multi-lead detector/resolver was run on 1,382 non-paced neonatal and pediatric ECGs, only 4 individual narrow QRS complexes were falsely detected as pacemaker pulses.

A separate test has shown that the pacemaker pulse detector has excellent performance on pacemaker ECGs in the pediatric population.

6. Helfenbein ED, Lindauer JM, Zhou SH, Gregg RE, Herleikson EC. “A Software-based Pacemaker Pulse Detection and Paced Rhythm Classification Algorithm.” *Journal of Electrocardiology* 35:95 Supp, 2002.

Paced Rhythm Classification

The DXL Algorithm classifies the observed paced rhythm using a number of interpretation statements. For testing purposes, the statements have been combined into categories.

A paced rhythm database was created with 2,190 paced ECGs. A 12-lead rhythm strip was used for ECG classification. The ECGs in this database were classified into 5 groups based on observed paced rhythms:

- 93 cases with atrial pacing
- 1,385 cases with ventricular pacing
- 477 cases with dual chamber pacing (both chambers paced when pacing present)
- 175 cases with dual chamber pacing with intermittent inhibition of one chamber
- 60 cases with non-capture/non-sensing asynchronous pacing (fixed rate pacing with no pulse inhibition, usually due to placement of a pacemaker magnet).

The groups in this database contain both continuous and intermittent pacing.

A non-paced ECG database containing a total number of 10,965 ECGs was developed to stress the algorithm and to ensure good specificity:

- 1,686 ECGs with extreme noise in one or more leads
- 1,209 ECGs with narrow QRS complexes from newborns and the pediatric population randomly selected from an existing pediatric database
- 8,070 randomly selected non-paced adult ECGs

Identification of a 10-second, 12-lead ECG as paced with any pacing present, versus non-paced achieved an overall performance of 97.2% sensitivity and 99.9% specificity. The 2.8% missed were usually due to presence of only a single paced beat. The 11 false positives were due to random noise spikes that perfectly mimicked a pacing mode.

As shown in Table D-4 on page D-10, the three basic rhythms could be classified with 95% sensitivity with positive predictive value from 91 to 97%. Identification of dual pacing with intermittent inhibition of one chamber was more difficult, since one pacemaker pulse often made the difference in classification. Detection of asynchronous pacing is performed with a high positive predictive value.

Table D-4 Paced Rhythm

	Sensitivity (%)	Positive Predictive Value (%)	N
Atrial	95.7	95.7	93
Ventricular	95.5	97.1	1,385

Table D-4 Paced Rhythm

	Sensitivity (%)	Positive Predictive Value (%)	N
Dual	95.4	91.2	477
Dual w/ intermittent inhibition of 1 chamber	57.7	76.5	175
Non-Sensing / Asynchronous	65.6	95.2	60

Computer Interpretation/Development Process

When the discussion progresses from precise measurements to actual clinical diagnoses, a much more complex validation problem emerges, largely due to the sheer number of possible diagnoses. This problem is compounded by the simultaneous occurrence of many conditions. For example, if only ten diagnoses were considered, this would result in more than 3 billion possible combinations. In actuality, more than ten diagnoses must be considered, however, databases are created that only contain a sample of cases. Both *biased* and *unbiased* selections are useful.

Databases

Population Sample

A population sample is an example of an *unbiased* selection. The basic concept is to randomly select samples from a large set of sequentially obtained ECGs. Naturally, this is not truly unbiased since any source tends to select healthier or sicker patients. The incidence of abnormalities in the main sources for population samples is about 60%, so the database is biased toward sicker subjects.

Population databases can be characterized in terms of sensitivity and positive predictive value and this will generally provide a good indicator of how the algorithm will perform in daily use as applied to common diagnoses. The statistics are meaningless for rare conditions as the number of available cases is so small (perhaps only one or two).

Diagnosis sample

One of the weaknesses with population samples is that rare conditions occur so infrequently. To address this, some databases are enriched with a large number of these rare conditions in order to perform regression testing in order to improve the algorithm.

Such regression databases are useful for ensuring that any algorithm modifications do not have unintended consequences for the rare diagnoses. Because this database is not representative, statistics are not employed to predict real world behavior.

Measures of Quality

Measures of quality can be expressed as ratios (0 to 1 range) or multiplied by 100 for percentages from 0 to 100%. The charts and tables contained in the following pages use both methods.

When the algorithm is applied to a set of known cases, the algorithm results are compared to the actual clinical case. The algorithm may generate the correct answer (true positive, true negative) or an incorrect answer (false positive, false negative).

Many combinations of these results are possible, and ideally we want the algorithm to find all the database positives and have no false positives. Table D-5 on page D-12 is a useful way to illustrate the possible results.

Table D-5 Measures of Quality

		True	
		Pos	Neg
Algorithm	Pos	TP	FP
	Neg	FN	TN

Sensitivity

Sensitivity is defined as the ratio of **True Positive (TP)** to the sum of **TP** and **False Negative (FN)**. It is defined as the frequency that a specific diagnosis is detected from the database.

Positive Predictive Value

Positive Predictive Value is defined as the ratio of TP to the sum of TP and FP. It is defined as the frequency of a correct diagnosis.

Other Classification Measures

Many other combinations derived from Table D-5 on page D-12 are used in specific situations.

- *Specificity* is the ratio of TN to the sum of TN and FP, or how often true negatives are detected
- *Negative predictive value* is the ratio of TN to the sum of TN and FN, or how often true negatives are detected

Specificity and negative predictive value are useful when excluding a diagnosis is important. These measures are not generally used because they are not useful in evaluating the quality of the algorithm when the finding is relatively uncommon. Specificity and NPV always have high values under these conditions.

Single Measures of Quality

It is possible to develop a single measure that represents the overall quality of the algorithm. In general, sensitivity and positive predictive value are inversely related to one other. If we always call a particular diagnosis, our sensitivity will be high (1) but our positive predictive value will be low. If we diagnose only the most obvious cases, our positive predictive value will be high but the sensitivity will be low.

If we assume that sensitivity and positive predictive value are equally important, we can multiply the two for a representative index. Of course, the result does not seem as effective because the numbers are low. For a sensitivity and PPV of 0.8 each, the product will be 0.64. Such numbers are typical of many medical tests. This measure parallels a more complicated measure called *kappa* although this value is even lower than PPV times Sensitivity. We will not discuss these values in this Appendix as they are most useful in comparing two different algorithms.

Another single measure is called the test accuracy, and this is defined as the proportion of all tests that are correct classifications (true positives and true negatives)[7]:

(TP + TN)/(TP + TN + FP + FN) = Test Accuracy

This measure is shown as a summary at the end of each section.

Accuracy of Computer Classification in Adults

The bar graphs included in this section are derived from an analysis of a population database. From a series of 60,000 adult ECGs, 1,785 records were randomly selected and were subsequently annotated by an expert electrocardiographer. This database was not used in any way to develop the algorithm, but was only used to test its performance. This is important since it is relatively easy to calibrate an algorithm for a specific set of ECGs. Such calibration produces impressive numbers, but provides poor performance in a general population.

7. Wassertheil-Smoller S. *Biostatistics and Epidemiology*. 3rd edition, Springer-Verlag 2003.

The graphs show results for conditions with a prevalence of at least 4% in the database. The category is given with prevalence numbers as % (cases/ population). Sensitivity, Positive Predictive Value, Specificity and Accuracy are charted and represented in the following tables.

Adult Rhythm

Of the many rhythm disturbances that can be classified, some occur so rarely in the population database that the statistics not readily interpretable. Note in Figure D-1 on page D-14 that all the specificity values are quite high; this is a reflection of the low prevalence of these rhythms with the exception of sinus rhythm.

Figure D-1 Common Adult Rhythms

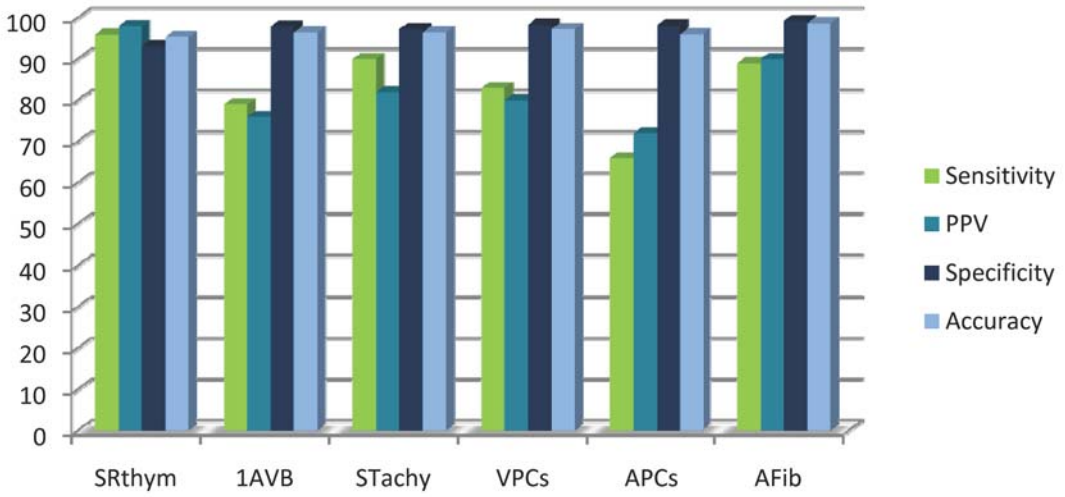


Table D-6 Common Adult Rhythms

	Sensitivity %	PPV %	Specificity %	Accuracy %
SRthym	96	98	93.22	95.4
1AVB	79	76	97.9	96.5
STachy	90	82	97.3	96.5
VPCs	83	80	98.4	97.3
APCs	66	72	98.2	96.1
AFib	89	90	99.3	98.7

Conduction Defects

Figure D-2 Adult Conduction Defects

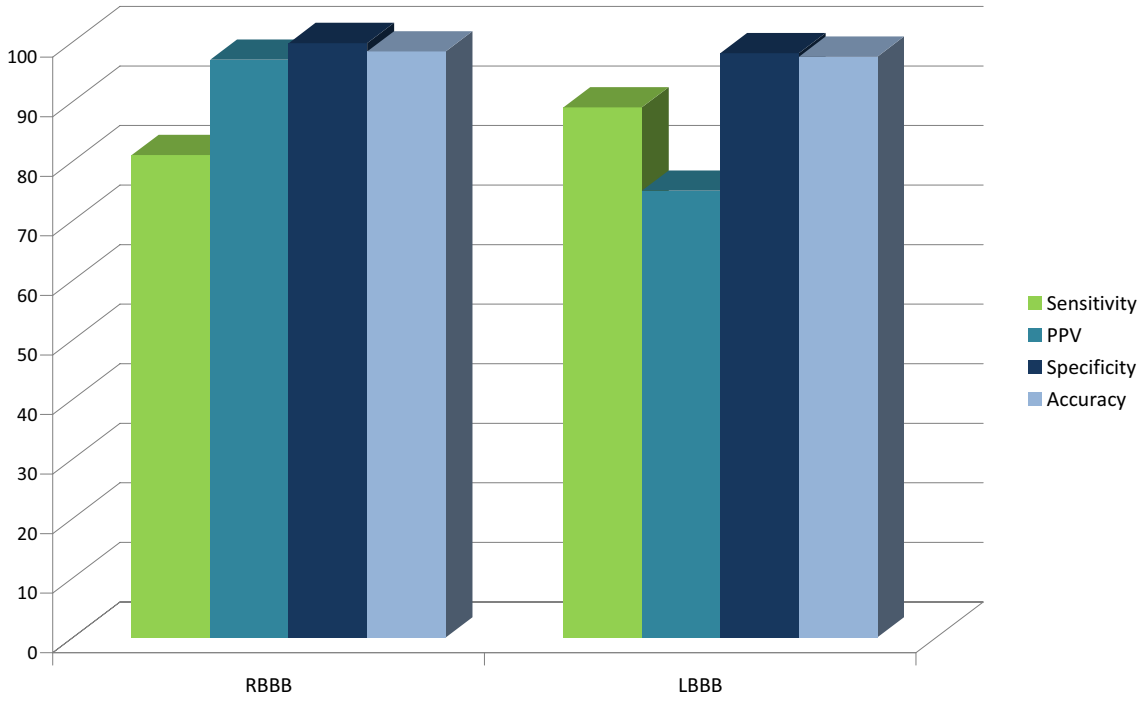


Table D-7 Adult Conduction Defects

	Sensitivity %	PPV %	Specificity %	Accuracy %
RBBB	81	97	99.8	98.4
LBBB	89	75	98.1	97.5

Hypertrophy

Figure D-3 on page D-16 shows the results for adult hypertrophy. Right Ventricular Hypertrophy deserves some further comment. In the adult series, RVH occurs in about 2% of cases. In the pediatric series, the prevalence is 21% (see below). Anatomical correlation of RVH criteria has shown that results are better in populations with a high prevalence of congenital heart disease and worse in adult populations[8].

8. Duda RO, Hart PE. *Pattern Classification and Scene Analysis* Wiley 1973. Chou T, Knilans TK. *Electrocardiology in Clinical Practice*. Fifth Edition, Saunders 2001.

Figure D-3 Adult Hypertrophy

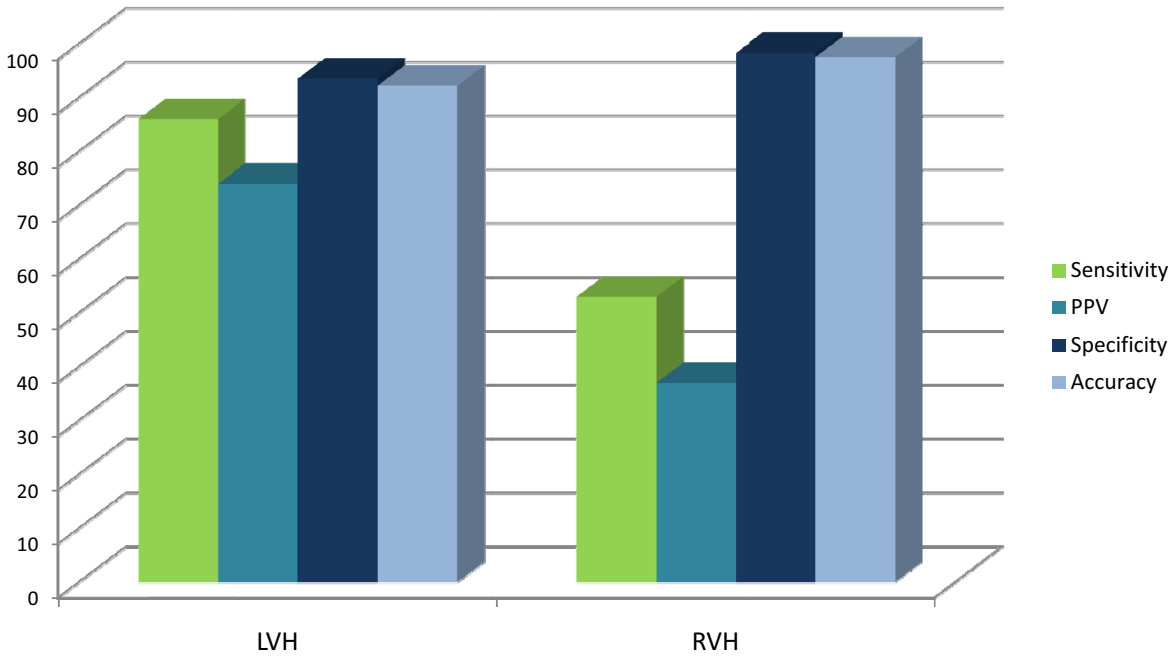


Table D-8 Adult Hypertrophy

	Sensitivity %	PPV %	Specificity %	Accuracy %
LVH	86	74	93.5	92.2
RVH	53	37	98.3	97.5

Infarction

Pericarditis and early repolarization variants confound ST-elevation acute infarcts. An enriched diagnostic database has been used to study this problem and to improve differentiation[9]. The Philips DXL ECG Algorithm provides posterior infarct interpretations using information in the anterior leads, such as ST depression and R/S ratio in V1-V3[10]. The accuracy of myocardial infarction detection by age of infarct and anatomical location of the infarct is tested on standard 12-lead ECGs for anterior and inferior locations, and acute versus old infarcts. When testing infarct age, old versus acute, all infarct anatomical locations are grouped together. When testing location, anterior versus inferior, all infarct ages are grouped together. In this way, each test is specific to just one parameter, anatomical location, or age.

9. Zhou SH, Helfenbein ED, Lindauer JM, Clifton J, Selvester RH, Wagner GS. "Classification of ST-Elevation Acute Myocardial Infarction, Acute Pericarditis and Benign Early Repolarization." *Journal of Electrocardiology* 33:251 Supp, 2000.

10. Zhou SH, Statt/Selvester RH, Rautaharju P, Haisty WK, Horacek BM, et al. "Computer Classification Algorithm for Strictly Posterior Myocardial Infarction." *Journal of Electrocardiology* 36:41 Supp, 2003.

The data set consists of acute MI, and recent and old MI of various infarct locations, in addition to a set of normals. The acute/recent MI cases were confirmed by two cardiologists using serial ECG changes. Old MI cases from the CSE diagnostic set were confirmed by two cardiologists independent from the CSE project. The normal cases were taken from a large community health study.

Performance results are included for the Acute MI (**>>> Acute MI <<<**) Critical Value statement. For more information on the optional Critical Values feature, see "Critical Value Statements" on page 5-1. The Acute MI Critical Value Statement is part of the configurable Critical Values feature that provides a simple alert statement that employs simple terminology and is intended for the non-electrocardiographer. This statement appears along with any definite ST elevation MI (STEMI) statement. The emphasis is on specificity so that this feature can be used as an alert that the patient likely requires urgent care.

Figure D-4 12-Lead Infarcts

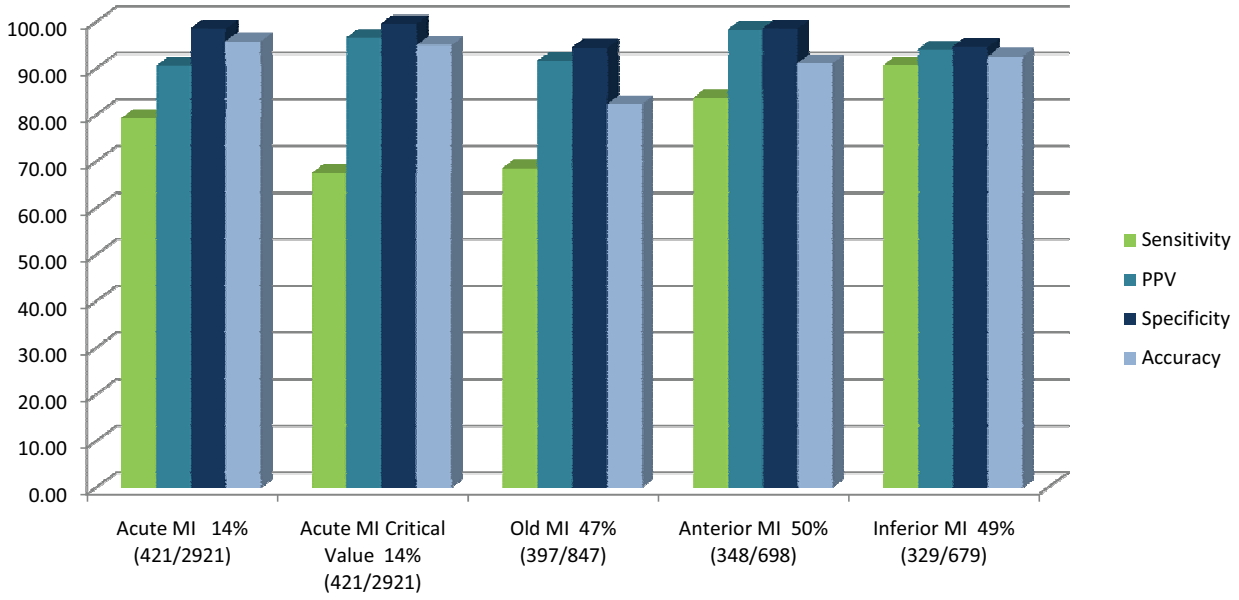


Table D-9 12-Lead Infarcts

	Sensitivity %	PPV %	Specificity %	Accuracy %
Acute MI 14% (421/2921)	79.3	90.5	98.6	95.8
Acute MI Critical Value 14% (421/2921)	67.5	96.6	99.6	95.0
Old MI 47% (397/847)	68.5	91.6	94.4	82.3
Anterior MI 50% (348/698)	83.6	98.3	98.6	91.1

Table D-9 12-Lead Infarcts

	Sensitivity %	PPV %	Specificity %	Accuracy %
Inferior MI 49% (329/679)	90.6	94.0	94.6	92.6

Infarction Using Additional Leads

It is apparent from many recent studies, and from current ACC/AHA guidelines, that additional electrode positions can improve the ability to detect right ventricular and posterior left ventricular infarcts[11].

To study this, we employed a database that included laboratory confirmation of myocardial infarction and extensive surface maps.

Right ventricular infarcts are basically invisible on a standard 12-lead ECG, but can be detected with increasing sensitivity using more right-sided leads. Figure D-5 on page D-19 shows that the addition of V3R does not add any further improvement.

11. Multiple Authors. "ACC/AHA Guidelines for the Management of Patients With ST-Elevation Myocardial Infarction A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1999 Guidelines for the Management of Patients With Acute Myocardial Infarction) Developed in Collaboration With the Canadian Cardiovascular Society." *Journal of the American College of Cardiology* 44:E1-E211 2004. See especially pages e110, e127 and e181 for recommendations on additional electrode positions. Tragardh E, Claesson M, Wagner GS, Zhou S, Pahlm O. "Detection of acute myocardial infarction using the 12-lead ECG plus inverted leads versus the 16-lead ECG (with additional posterior and right-sided chest electrodes)." *Clinical Physiology and Functional Imaging* 2007 Nov; 27(6):368-74. SH Zhou, RH Startt/Selvester, X Liu, EW Hancock et al. "An Automated Algorithm to Improve ECG Detection of Posterior STEMI Associated with Left Circumflex Coronary Artery Occlusion." *Computers in Cardiology* 2006;33:33-36. X Liu1, E Tragardh, SH Zhou, O Pahlm et al. "Right Precordial Leads V4R and V5R in ECG Detection of Acute ST Elevation MI Associated with Proximal Right Coronary Artery Occlusion." *IEEE Computers in Cardiology* 2005 Vol 32, p.651-654. Zalenski RJ, Rydman RJ, Sloan EP, et al. "Value of posterior and right ventricular leads in comparison to the standard 12-lead electrocardiogram in evaluation of ST-segment elevation in suspected acute myocardial infarct." *American Journal of Cardiology* 1997: 79:1579-1585.

Figure D-5 RMI – Additional Right-sided Leads

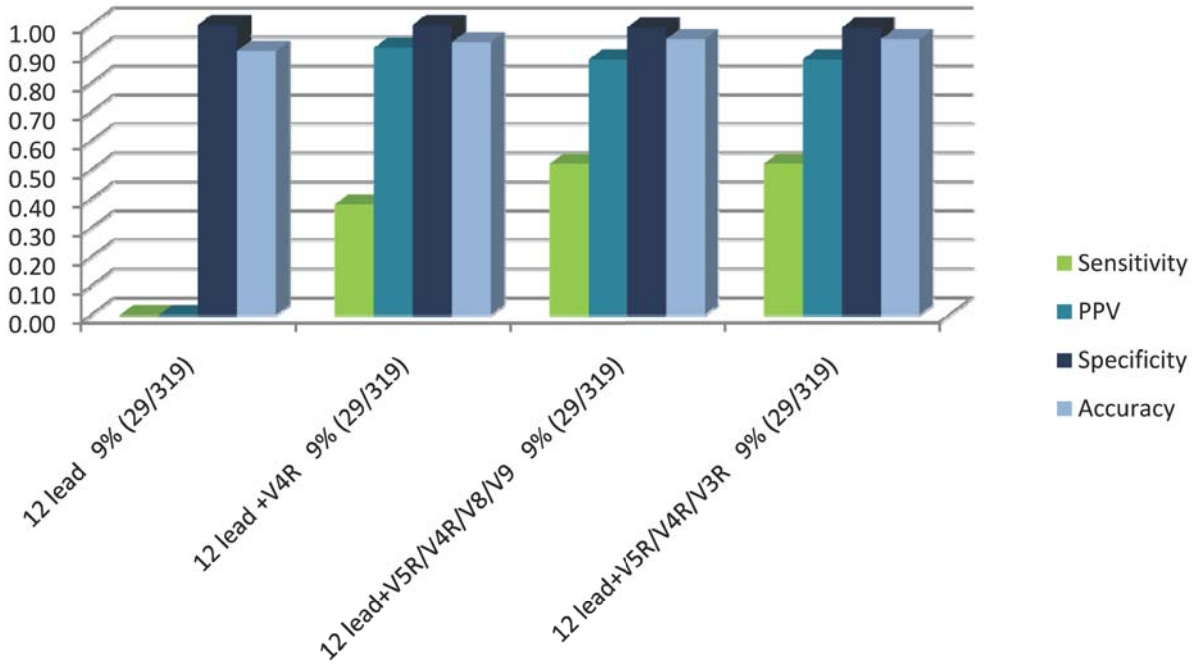


Table D-10 RMI – Additional Right-sided Leads

	Sensitivity	PPV	Specificity	Accuracy
12 lead 9% (29/319)	0.00	0.00	1.00	.91
12 lead +V4R 9% (29/319)	.38	.92	1.00	.94
12 lead+V5R/V4R/V8/ V9 9% (29/319)	.52	.88	.99	.95
12lead+V5R/V4R/V3R 9% (29/319)	.52	.88	.99	.95

The use of at least one posterior lead (V8) largely overcomes the extremely poor sensitivity of a standard 12-lead ECG. Additional posterior leads further increase the sensitivity, but at a cost in positive predictive values (there are more false positives).

Figure D-6 PMI – Additional Posterior Leads

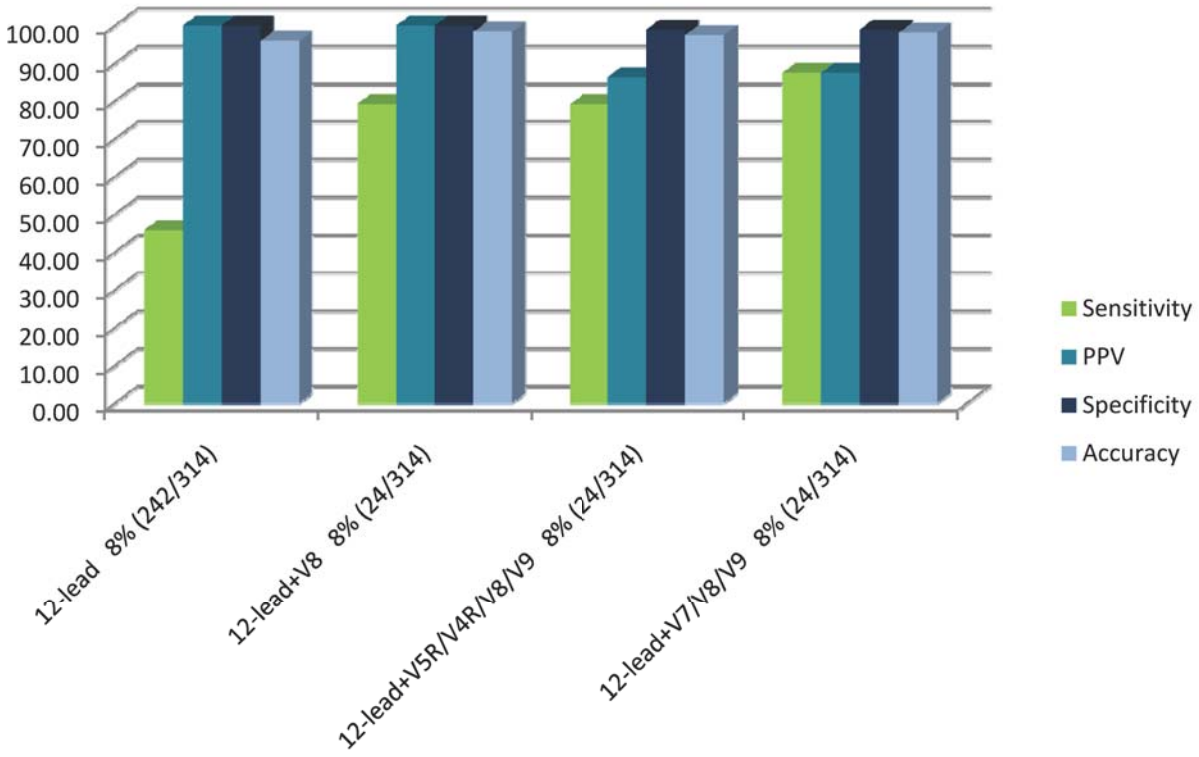


Table D-11 PMI – Additional Posterior Leads

	Sensitivity	PPV	Specificity	Accuracy
12-lead 8% (242/314)	45.80	100.00	100.00	95.90
12-lead+V8 8% (24/314)	79.20	100.00	100.00	98.40
12-lead+V5R/V4R/V8/V9 8% (24/314)	79.20	86.40	99.00	97.50
12-lead+V7/V8/V9 8% (24/314)	87.50	87.50	99.00	98.10

Culprit Vessel Evaluation

To identify culprit vessel performance, a database of patients with suspected myocardial infarction seen in Long Beach Memorial Hospital Emergency Department between 2003-2004 was employed. This included 268 patients with a discharge diagnosis of Acute MI who also had an available admission ECG, and 266 patients matched for age, gender and symptoms who did not have an acute MI diagnosis at discharge.

It is not possible to evaluate the algorithm using a simple discharge diagnosis, because the admission ECG often did not show ST elevation that matched the recommended criteria[12]. It is therefore necessary to select only those patients with measurable ST elevation, and then compare those patients to the results of their angiography. The following charts show the results of this process.

When STE meets the criteria, differentiation of the offending vessel is generally sensitive. Circumflex detection is not as sensitive but is highly specific. Left Mainstem is the least sensitive but appears to be quite reliable in this series of patients.

Figure D-7 Vessel Classification Given STEMI – 12 Lead

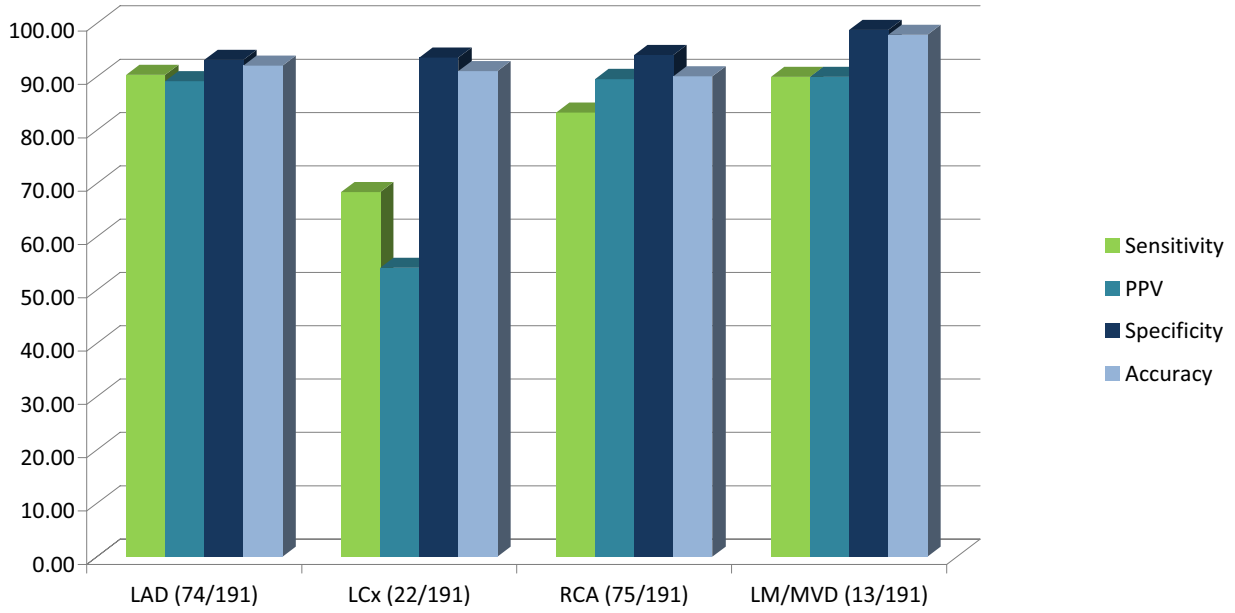


Table D-12 Vessel Classification Given STEMI – 12 Lead

	Sensitivity	PPV	Specificity	Accuracy
LAD (74/191)	90.40	89.2	93.2	92.1
LCx (22/191)	68.4	54.2	93.6	91.1
RCA (75/191)	83.3	89.6	94.1	90.1

12. "2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care." *Circulation*. 2005;112:IV-89 – IV-110.

Table D-12 Vessel Classification Given STEMI – 12 Lead *(continued)*

	Sensitivity	PPV	Specificity	Accuracy
LM/MVD	90.0	90.0	98.8	97.9

Overall Accuracy in Adult Cardiograms

Figure D-8 on page D-22 shows the accuracy (in %) for all the major categories and for the overall average. Recall that accuracy is the proportion of cases in which negatives and positives are correctly detected.

Figure D-8 Adult Accuracy

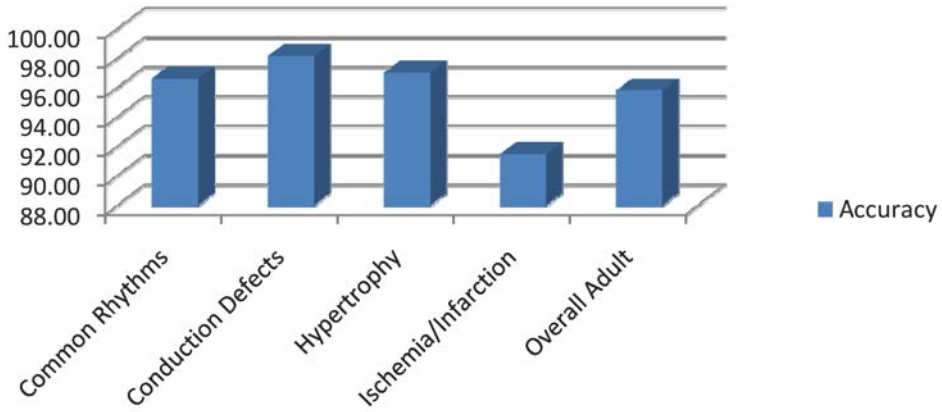


Table D-13 Adult Accuracy

	Accuracy
Common Rhythms	96.63
Conduction Defects	98.15
Hypertrophy	97.00
Ischemia/Infarction	91.48
Overall Adult	95.81

Accuracy of Computer Classification in Pediatric Subjects

The following bar graphs are derived from an analysis of a pediatric population database. From a series of 4,000 pediatric ECGs, 424 records were randomly selected and annotated by an expert electrocardiographer. This database was not used in the development of the algorithm. The graphs show results for entities with a prevalence of at least 4%.

Pediatric Rhythms

Figure D-9 Pediatric Rhythms

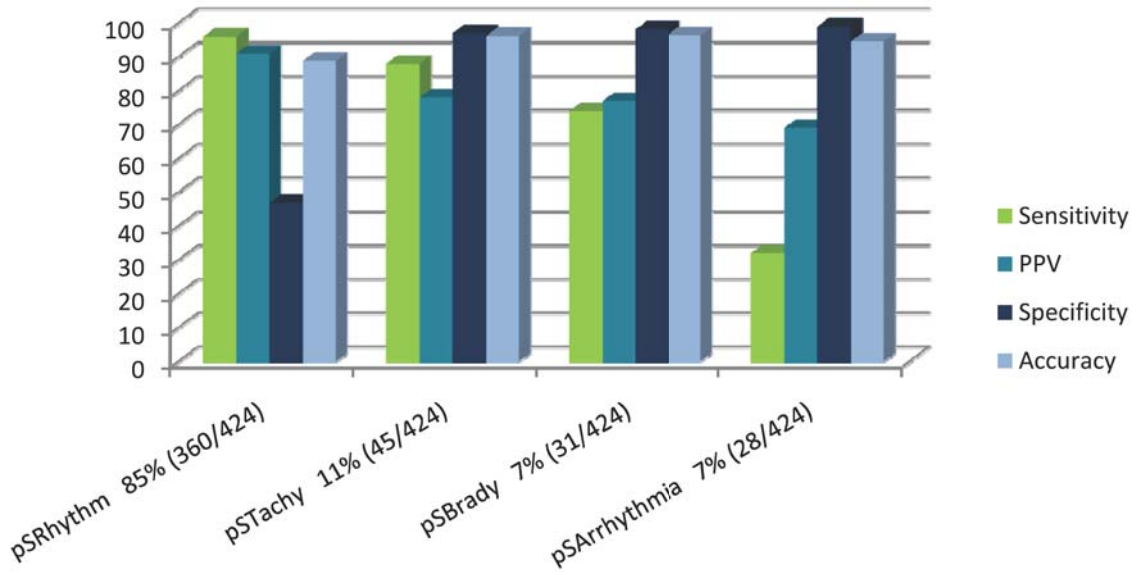


Table D-14 Pediatric Rhythms

	Sensitivity %	PPV %	Specificity %	Accuracy %
pSRhythm 85% (360/424)	96	91	46.9	88.9
pSTachy 11% (45/424)	88	78	97.1	96.2
pSBrady 7% (31/424)	74	77	98.2	96.5
pSArrhythmia 7% (28/424)	32	69	99	94.6

Pediatric Morphology

Right ventricular hypertrophy and right bundle branch block are easily confused in the pediatric population. Good performance has been achieved using measurements from synthesized vectors[13].

Figure D-10 Pediatric Morphologies

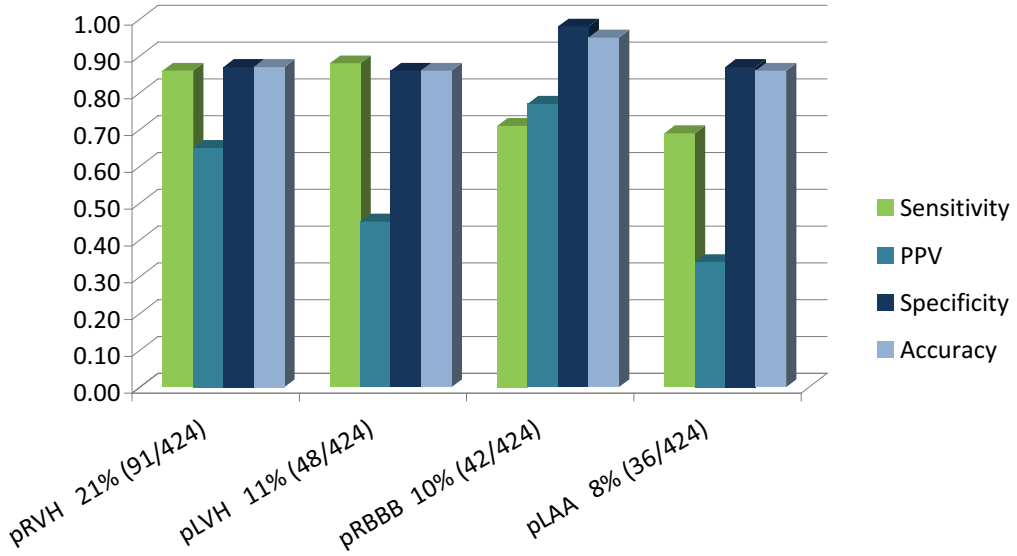


Table D-15 Pediatric Morphologies

	Sensitivity	PPV	Specificity	Accuracy
pRVH 21% (91/424)	.86	.65	.87	.87
pLVH 11% (48/424)	.88	.45	.86	.86
pRBBB 10% (42/422)	.71	.77	.98	.95
pLAA 8% (36/424)	.69	.34	.87	.86
pRAE 8% (35/424)	.71	.60	.96	.94

13. Zhou SH, Liebman J, Dubin AM, Gillette PC, Gregg RE, Helfenbein ED, Lindauer JM. "Using 12-Lead ECG and Synthesized VCG in Detection of Right Ventricular Hypertrophy with Terminal Conduction Delay versus Partial Right Bundle Branch Block in the Pediatric Population." *Journal of Electrocardiology* 34:249-257 Supp, 2001.

Extra leads are often used for pediatric ECGs because they provide more information. In a separate database of 1112 consecutive pediatric ECGs overread by two pediatric cardiologists, the results are shown in Table D-16 on page D-25.

Figure D-11 12 vs 15 Lead

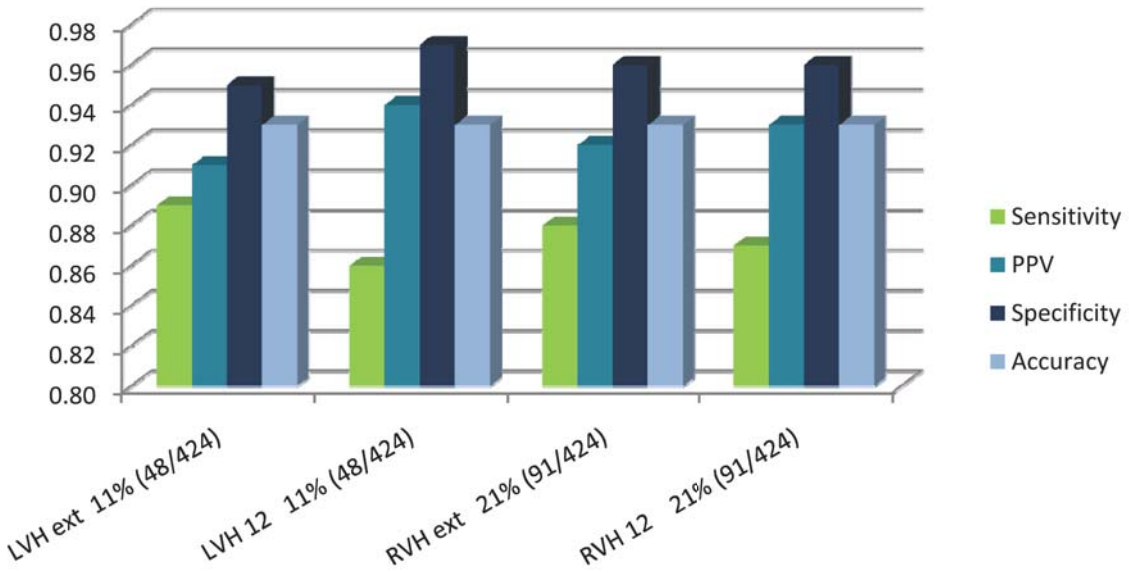


Table D-16 12 vs 15 Lead

	Sensitivity	PPV	Specificity	Accuracy
LVH ext 11% (48/424)	.89	.91	.95	.93
LVH 12 11% (48/424)	.86	.94	.97	.93
RVH ext 21% (91/424)	.88	.92	.96	.93
RVH 12 21% (91/424)	.87	.93	.96	.93

As Table D-16 demonstrates, there is a small increase in sensitivity with a concomitant decrease in positive predictive value, so the overall accuracy remains constant.

Pediatric Accuracy

A comparison of adult and pediatric algorithm accuracy shows that pediatric diagnoses are more challenging. This is due to the rapidly changing standards for different age groups and the generally smaller number of cases tested. It is also due to the very high prevalence of abnormalities in this pediatric population. Of the 425 subjects, only 36 had sinus rhythm with no other abnormalities, reflecting the referral nature of the hospital practice.

Figure D-12 Pediatric Accuracy %

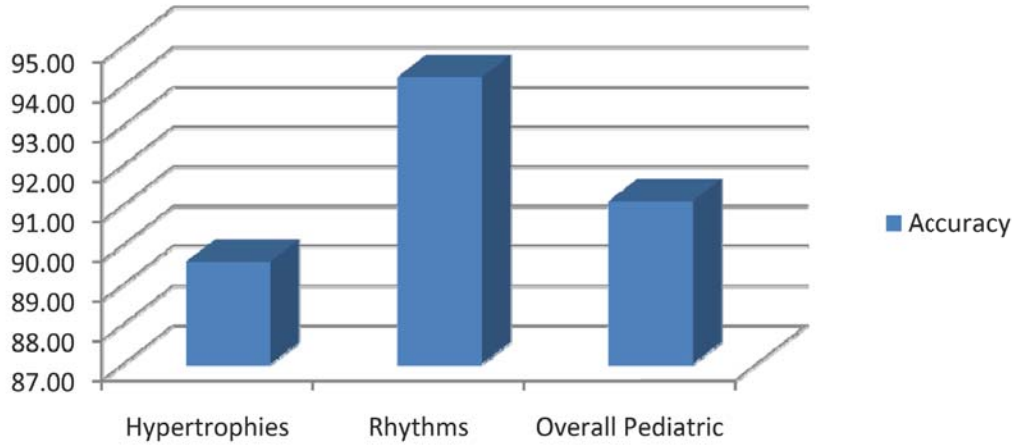


Table D-17 Pediatric Accuracy

	Accuracy
Hypertrophies	89.58
Rhythms	94.22
Overall Pediatric	91.90

Validation Data for the DXL ECG Algorithm vPH110C

The following tables present the validation data for the PH110C version of the DXL algorithm. Refer to the rest of this appendix for details about the various validation procedures.

Table D-18 Pass Condition for All Amplitude Tests, PH110C

Measurement (16 records X 8 leads per record = 128)	N PASS
P1 amplitude	128
P2 amplitude	128
Q amplitude	128
R amplitude	128
S amplitude	128
J amplitude	128

Table D-18 Pass Condition for All Amplitude Tests, PH110C *(continued)*

Measurement (16 records X 8 leads per record = 128)	N PASS
ST amplitude, J + 60 ms	128
T amplitude	128

Table D-19 Accuracy of Absolute Interval and Wave Duration Measurements, PH110C

Measurement	Mean difference (ms)	Acceptable difference (ms)	Error SD (ms)	Acceptable SD (ms)	N
P duration	-1.8	±10	0.9	8	12
PR interval	-2.7	±10	0.7	8	12
QRS duration	2.9	± 6	1.2	5	12
QT interval	-0.2	±12	1.6	10	12
Q duration	-0.2	± 6	1.6	5	124
R duration	0.7	± 6	1.9	5	124
S duration	-2.2	± 6	2.5	5	92

Table D-20 Expert Annotated Biological Signals, PH110C

Measurement	Mean difference (ms)	Acceptable difference (ms)	Difference SD (ms)	Acceptable SD (ms)	N
P duration	1.9	± 10	5.4	15	92
PR interval	0.1	± 10	4.8	10	92
QRS duration	-0.5	± 10	3.8	10	92
QT interval	0.4	± 25	7.5	30	92

Table D-21 Common Adult Rhythms, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
SRthym (1345/1785)	97	98	93	96
1AVB (138/1785)	75	73	98	96
STachy (209/1785)	91	88	98	97
VPCs (130/1785)	84	80	98	97
APCs (115/1785)	74	73	98	97
AFib (109/1785)	85	83	99	98

Table D-22 Adult Conduction Defects, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
RBBB (136/1785)	82	95	100	98
LBBB (107/1785)	90	71	98	97

Table D-23 Adult Hypertrophy, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
LVH (317/1785)	86	75	94	92
RVH (32/1785)	53	47	99	98

Table D-24 12-Lead Infarcts, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
Acute MI 14% (421/2921)	75	92	99	96
Acute MI Critical Value 14% (421/2921)	68	96	100	95
Old MI 47% (397/847)	68	90	94	82
Anterior MI 50% (348/698)	82	98	98	90

Table D-24 12-Lead Infarcts, PH110C *(continued)*

	Sensitivity %	PPV %	Specificity %	Accuracy %
Inferior MI 49% (329/679)	87	94	94	91

Table D-25 RMI – Additional Right-sided Leads, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
12 lead 10% (42/404)	0	0	100	90
12 lead+V4R 10% (42/404)	50	88	99	94
12 lead+V5R/V4R/V8/V9 10% (42/404)	55	89	99	95
12 lead+V5R/V4R/V3R 10% (42/404)	57	69	97	93

Table D-26 PMI – Additional Posterior Leads, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
12-lead 8% (32/60)	28	94	100	90
12-lead+V8 8% (34/62)	52	88	99	93
12-lead+V5R/V4R/V8/V9 8% (34/63)	54	76	97	92
12-lead+V7/V8/V9 8% (34/62)	54	73	97	91

Table D-27 Culprit Vessel Classification Given STEMI – 12 Lead, ED Training Set, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
Proximal LAD 12% (9/75)	33	81	98	83
LAD 38% (61/161)	99	96	97	98
LCx 14% (22/161)	43	75	98	92
RCA 48% (78/161)	96	89	88	92
LM/MVD 11% (18/167)	89	84	98	97

Table D-28 Culprit Vessel Classification Given STEMI – 12 Lead, ED Prehospital Test Set[a], PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
Proximal LAD 12% (10/80)	50	100	100	94
LAD (proximal and nonproximal) 25% (21/80)	90	100	100	98
LCx 24% (18/80)	50	90	98	88
RCA 51% (41/80)	98	78	72	85

a. The prehospital set comprised 111 patients (21% female), with ages ranging from 38 to 87 years, collected during a period of 3 years from 2008 through 2010 in Spain. 102 ECGs could be located. Inclusion criteria were STEMI by algorithm, not paced or LBBB, and angiographic confirmation of a culprit single-vessel stenosis of 70% or greater (n=80).

Table D-29 Adult Accuracy, PH110C

	Accuracy %
Common Rhythms	97
Conduction Defects	98
Hypertrophy	95
Ischemia/Infarction	92
Overall Adult	96

Table D-30 Pediatric Rhythms, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
pSinusRhythm 85% (360/424)	98	91	45	90
pSTachy 11% (45/424)	90	74	96	96
pSBrady 7% (31/424)	77	73	98	96
pSArrhythmia 7% (28/424)	36	71	99	95

Table D-31 Pediatric Morphologies, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
pRVH 21% (91/424)	88	65	87	87
pLVH 11% (48/424)	83	42	85	85
pRBBB 10% (42/424)	79	79	98	96
pLAA 8% (36/424)	67	33	87	86
pRAE 8% (35/424)	77	60	95	94

Table D-32 12 vs 15 Lead, PH110C

	Sensitivity %	PPV %	Specificity %	Accuracy %
LVH, 15 lead 34% (236/702)	84	79	89	87
LVH, 12 lead 34% (263/702)	81	87	94	90
RVH, 15 lead 50% (470/936)	86	85	84	85
RVH, 12 lead 50% (470/936)	72	94	95	84

Table D-33 Pediatric Accuracy, PH110C

	Accuracy %
Hypertrophy	89
Rhythms	94
Overall Pediatric	92

E

List of Abbreviations and Statistical Measures

Table E-1 List of Abbreviations

Abbreviation	Definition
1AVB	First degree AV block
Acute Ant MI	Acute anterior myocardial infarction
Acute Inf MI	Acute inferior myocardial infarction
Acute Lat MI	Acute lateral myocardial infarction
Afib	Atrial fibrillation
APCs	Atrial Premature Complexes
LAA	Left atrial abnormality
LBBB	Left Bundle Branch Block
LVH	Left Ventricular Hypertrophy
Old Ant MI	Old anterior myocardial infarction
Old Inf MI	Old inferior myocardial infarction
RAE	Right atrial enlargement
RBBB	Right Bundle Branch Block
RVH	Right Ventricular Hypertrophy
S Arrhythmia	Sinus arrhythmia (respiratory or irregular variation in sinus rhythm)
S Brady	Sinus Bradycardia (rate < 50)
S Rhythm	Sinus Rhythm (normal rate)
STachy	Sinus Tachycardia (rate > 100)
VPCs	Ventricular premature complexes

Statistical Measures of Classification

Table E-2 Statistical Measures of Classification

		Disease		
		Positive	Negative	
Test	Positive	True Positive (TP)	False Positive (FP)	TP + FP
	Negative	False Negative (FN)	True Negative (TN)	FN + TN
		TP + FN	FP + TN	

SENSITIVITY = TP / TP+FN

SPECIFICITY = TN / TN+FP

POSITIVE PREDICTIVE VALUE (PPV) = TP / TP+FP

NEGATIVE PREDICTIVE VALUE (NPV) = TN / FN+TN

$$\text{TEST ACCURACY} = \frac{TP + TN}{TP + TN + FP + FN}$$

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