Chamber Hypertrophy/Enlargement and Ventricular Conduction Defects

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UTHSCSA and STVAHCS

P wave

The normal atrial wall is only 1-2 mm thick

Each atrium weighs about 20 gm

Interatrial septum weighs about 10-20 gm

Atrial Abnormalities

• Right atrial abnormality
• Left atrial abnormality
• Interatrial conduction disturbances

Friedman 1971; Chou 1974; Surawicz 2001
Normal P wave

Note the different planes

Transverse plane
Right Sagittal plane
Frontal plane
Normal P wave

Transverse plane

Right Sagittal plane

Frontal plane
Transverse Plane

- E point is the beginning of the P wave, the end of the T-P segment
- O point is the end of the PR segment, the beginning of the QRS
Transverse Plane

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Transverse Plane

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RAE vs LAE

[Diagrams of heart anatomy showing left atrial enlargement (RAE) and right atrial enlargement (LAE)]
RAE
RAE
LEFT ATRIAL ENLARGEMENT

LAE
### Atrial Enlargement

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<th>II</th>
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<tr>
<td><strong>RAE</strong></td>
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<td><strong>LAE</strong></td>
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<td><strong>RAE + LAE</strong></td>
<td><img src="image5" alt="Waveform" /></td>
<td><img src="image6" alt="Waveform" /></td>
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**Surawicz B et al.** Chou’s ECG… 2001, p.35

**Wagner GS.** Marriott’s Practical Electrocardiography 1994, p.58
Atrial Enlargement or Hypertrophy

- Atrial abnormalities are usually with enlargement of one or both ventricles
- RAE (may wax and wane)
  - 1.5 mm tall P in V1 or V2 and rightward (>75) P axis are best
  - 2.5 mm tall P wave in II has false positives (sympathetic tone, standing position, low diaphragm position), can represent LAE too (“pseudo P pulmonale”)

Surawicz 2001 Ch. 2
Atrial Enlargement or Hypertrophy

- Atrial abnormalities are usually with enlargement of one or both ventricles
- LAE (may wax and wane)
  - Prolonged P duration >0.12 sec, and notch in P wave with 0.04 sec between peaks, also P axis <15 degrees
  - P terminal force more than 1 mm deep and wide in V1

Surawicz 2001 Ch. 2
Atrial Enlargement or Hypertrophy

- False positives:
  - RA abnormality:
  - II tall in standing, in low diaphragm and in sympathetic tone
  - V1 tall in LA enlargement (pseudo P pulmonale)

Surawicz 2001 Ch. 2
Atrial Enlargement or Hypertrophy

- False positives:
  - LA abnormality:
  - V1 P terminal force abnormal in COPD
  - V1 P terminal force abnormal in straight back syndrome and pectus excavatum
  - Occasionally in congenital disease with massive RA

Surawicz 2001 Ch. 2
Atrial Enlargement or Hypertrophy

- **Echo Correlation**
  - RA abnormality:
    - Using V1, RAD of QRS and R/S>1 in V1, sens 48% and spec 100%
  - LA abnormality:
    - V1 Pterm>0.06 had PPV .58, NPV .83
    - V1 P term plus P dur >100ms had sens .82
Atrial Enlargement or Hypertrophy

- Biatrial enlargement:
  - V1 has both RAE and LAE criteria
  - V1 has RAE criteria and inferior or lateral leads show wide notched P
  - Limb leads have both >2.5mm and >0.12 sec
- Atrial enlargement in atrial fibrillation (>1mm)
- Intra-atrial conduction disturbance
- Atrial repolarization may last 0.45 sec, about 1/3 height of P

Surawicz 2001 Ch. 2
2.5 mm tall P wave in II, prominent initial force in V1
ECG - LAE

- Notch in P wave with 0.04 sec between peaks
- P terminal force more than 1 mm deep and wide in V1

- P duration >0.12 sec (3 mm)
Left Ventricular Hypertrophy
Left Ventricular Hypertrophy

Left Ventricular Hypertrophy

Chou et al. Clinical Vectorcardiography 2\textsuperscript{nd} ed 1974. p. 73.
Left Ventricular Hypertrophy

- The LV normally dominates the QRS, since it is about 10 mm thick and the RV is only a few mm thick.
- When the LV mass increases, the ECG forces directed leftward and inferiorly and posteriorly increase.
Left Ventricular Hypertrophy

• Summary:
  – increased QRS amplitude,
  – widened QRS-T angle, and
  – tendency to left axis deviation
  – intraventricular conduction delay manifested by delayed intrinsicoid deflection
Left Ventricular Hypertrophy
Point Score System (Romhilt and Estes, 1968)

- 3 points: Voltage of R or S of 20 mm in limb lead, or 30 mm in chest lead
- 3 points: Left atrial abnormality in V1
- 3 points: Repolarization abnormal off dig.
- 1 point: Repolarization abnormal on dig.
- 1 point: Intrinsicsoid deflection in V5-V6 >0.05 sec
- 1 point: QRS duration 0.09-0.10 sec
- 2 points: LAD <-30 degrees (not very helpful)

Sensitivity 54%
Specificity 97%

Surawicz 2001 Ch. 3.
Left Ventricular Hypertrophy

- Cornell Voltage Criteria
- R wave in aVL + S wave in lead V3!
- Women: >2.0 mV (20 mm)
- Men: >2.8 mV (28 mm)
ECG LVH
Right Ventricular Hypertrophy

- Septal Depolarization
- Apical Depolarization
- Ventricular Depolarization
- Terminal Depolarization

Tall R in V1 and V2; Deep S in V5, V6, and lead I
Right Ventricular Hypertrophy

• Tougher than LVH, because RV forces must increase more to overcome the normally dominant LV forces

• RV forces are rightward and anterior and inferior

• Criteria
  - Right axis >110 degrees
  - R/S in V1 or V3R >1
  - R in V1 >7mm
  - S in V1 <2mm
  - qR pattern in V1 or V3R
  - rSRprime in V1 with Rprime >10mm
  - also, ST depression and T inversion in V1-V2
Right Ventricular Hypertrophy

Right Ventricular Hypertrophy Type A

Right Ventricular Hypertrophy Type B

Right Ventricular Hypertrophy Type C

Types of RVH by ECG

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<thead>
<tr>
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<th>V1</th>
<th>V6</th>
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<tr>
<td>Normal</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>RVH type A</td>
<td>+</td>
<td>-</td>
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<tr>
<td>RVH type B</td>
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<tr>
<td>RVH type C</td>
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ECG - RVH

- Right axis >110 degrees
- R/S in V1 or V3R >1
- R in V1 >7mm
- S in V1 <2mm
- qR pattern in V1 or V3R
- rSRprime in V1 with Rprime >10mm
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ECG - RVH

- Right axis > 110 degrees
- R/S in V1 or V3R > 1
- R in V1 > 7mm
- S in V1 < 2mm
- qR pattern in V1 or V3R
- rSRprime in V1 with Rprime > 10mm
- also, ST depression and T inversion in V1-V2
ECG - RVH
Biventricular Hypertrophy

- The same ECG meets one or more criteria for both isolated LVH and RVH
- Precordial leads look like LVH but QRS axis in limb leads is > 90 degrees
- Signs of LVH and R>Q in aVR and S>R in V5 and T inversion in V1

Surawicz 2001 Ch. 3. p. 68.
Conduction Abnormalities

- Normal ventricular activation
- LBBB
- LAFB, LPFB
- RBBB
- IVCD

Causes of wide QRS:
- RBBB and bifascicular
- LBBB
- IVCD
- WPW
- Non-His origin beat
- Paced QRS
Ventricular Activation

- **Septal Depolarization**
- **Apical Depolarization**
Ventricular Activation

Frontal Plane
Ventricular Activation
Ventricular Repolarization

Frontal Plane
Ventricular Activation
Sequence of Depolarization
Sequence of Depolarization

Sequence of Depolarization

Chou, 2001, p.12-13
From Durrer 1968
Human heart
Sequence of Ventricular Activation

**SEQUENCE OF VENTRICULAR ACTIVATION**

**PHASE 1**
INITIAL SEPTAL ACTIVATION.
(0.01 SEC)

**PHASE 2**
CONTINUED ACTIVATION OF SEPTUM AND ACTIVATION OF APICO-ANTERIOR PORTIONS OF RIGHT AND LEFT VENTRICLES. (0.02 SEC)

**PHASE 3**
COMPLETION OF SEPTAL ACTIVATION AND ACTIVATION OF MOST, IF NOT ALL, OF RIGHT VENTRICLE AND MOST OF LEFT VENTRICLE.
(0.04-0.06 SEC)

**PHASE 4**
ACTIVATION OF POSTEROBASAL REGION OF LEFT VENTRICLE, BASE OF SEPTUM AND BASE OF RIGHT VENTRICLE. (0.06-0.08 SEC)

Friedman HH, 1971, p. 51.
Sequence of Ventricular Activation

Friedman HH, 1971
Sequence of Ventricular Activation

Friedman HH, 1971
Sequence of Activation

FRONTAL PLANE

TRANSVERSE PLANE
Deriving the Vector from the Scalar

From Grant RP. Clinical Electrocardiography. McGraw-Hill. 1957
Deriving the Vector from the Scalar

From Grant RP. Clinical Electrocardiography. McGraw-Hill. 1957
Deriving the Vector from the Scalar

From Grant RP. Clinical Electrocardiography. McGraw-Hill. 1957
Normal Frontal Plane QRS Loop

Mean maximal vector

Standard Limb Lead Projection

Limb Lead ECG and VCG
Vectorcardiographic Measurements

Vector Terminology

Transverse Plane

Transverse Plane

- The end of the QRS is not precisely at the same point as the beginning, so there is a normal ST segment, especially in the transverse plane.
Transverse Plane

- The end of the T loop is back to the E point
Transverse Plane

Maximum QRS Vector

0.04 sec.

Maximum QRS width

0.02 sec.

Transverse Plane

Terminal deflection (S loop)

Afferent limb

Body (R loop)

Initial deflection (Q loop)

Efferent limb
Left Bundle Branch Block

- Criteria
- Axis
- Atypical features
- Incomplete
- Corrected transposition
- LVH
- RVH
- Infarction/ primary T wave
Left Bundle Branch Block

QRS DURATION GREATER THAN 0.12 SECOND:
MAJOR QRS DEFLECTION (DURATION) UPWARD IN
LEAD I AND L. CHEST LEADS, DOWNWARD IN R. CHEST
LEADS; P AND P–R INTERVAL NORMAL
Left Bundle Branch Block

NORMAL

TRANSVERSE PLANE

RIGHT SAGITTAL PLANE

FRONTAL PLANE
ECG - LBBB

- QRS duration >= 0.12 sec
- Broad (usually notched or slurred) R in aVL, V5, and V6
- Absent Q waves in I, V5, and V6
- Delay in R peak time in V5 and V6 >0.06 sec
- Broad Deep S wave or QS complex in V1
- Repolarization: ST segment and T wave directed opposite to mean QRS
• QRS duration $\geq 0.12$ sec

• Broad (usually notched) R in aVL, V5, and V6

• Absent Q waves in I, V5, and V6

• Delay in R peak time in V5 and V6

• Broad Deep S wave or QS complex in V1

• Repolarization: ST segment and T wave directed opposite to mean QRS

ECG - LBBB

Discordant T wave

0.12 sec
ECG - LBBB

Discordant T wave

0.12 sec
Left Bundle Branch Block

- Axis may shift leftward or not
- Atypical features, RS in V5-6, Q in V1-2
- Incomplete, QRS 0.11 and R peak time 0.06 and no Q in V5 and V6 and I (excessive >0.15 or 0.18)
- Corrected transposition – absent rightward initial force, otherwise normal (due to ventricular inversion)
- LVH: S in V2 plus R in V5>45mm; QRS 0.16; probably anyway
- RVH: probably not
- Infarction/ primary T wave
  - Qs in I and aVL, V5 or V6; notch in S in V3-5; inf Q >30ms and T inversion – all these probably aren’t really any good, also for paced beats
  - Concordant ST elevation is diagnostic, especially lateral
Left Bundle Branch Block

• Intermittent:
  – Acceleration dependent – phase 3 block, voltage or time-dependent block, usual refractoriness
  – Deceleration dependent – phase 4 block, more controversial, spontaneous depolarization
  – Non rate-related
  – Ischemic, autonomic tone variations

• Complete may be relative, and can occasionally be made worse by pacing or premature beats
• Intermittent
  – Phase 3
  – Phase 4

Left Bundle Branch Block

- Automaticity depends on threshold, on maximal polarized potential and on phase 4 slope
- A partially depolarized cell may not propagate an action potential normally

Left Bundle Branch Block

- Automaticity depends on threshold, on maximal polarized potential and on phase 4 slope
- A partially depolarized cell may not propagate an action potential normally
Left Bundle Branch Block

- Intermittent
- Phase 3
- Phase 4

Right Bundle Branch Block

QRS DURATION GREATER THAN 0.12 SECOND:
MAJOR QRS DEFLECTION (DURATION) DOWNWARD
IN LEAD I AND L. CHEST LEADS, UPWARD IN R. CHEST
LEADS; P AND P-R INTERVAL NORMAL
Right Bundle Branch Block
Right Bundle Branch Block

NORMAL

RIGHT BUNDLE BRANCH BLOCK

TRANSVERSE PLANE

FRONTAL PLANE
Right Bundle Branch Block

- QRS duration $\geq 0.12$ sec
- R prime in V1 or V2 larger than R wave
- R prime in V1 usually 0.06 sec wide
- Wide (slurred) S wave in I, V5 and V6, often distinct onset
- Preserved initial forces
- Repolarization: T wave opposite the terminal delay
ECG - RBBB

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- R prime in V1 or V2 larger than R wave
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- Wide (slurred) S wave in I, V5 and V6, often distinct onset
- Preserved initial forces
- Repolarization: T wave opposite the terminal delay
ECG - RBBB

0.12 sec
Right Bundle Branch Block

- Rightward anterior terminal conduction delay
  - QRS 0.12, rsr' in V1 or 2, S>40 ms or S longer than R in V6 and I, normal R peak time in V5-6 but >0.05 sec in V1
- Initial 0.06 sec unaltered, terminal delay at least 0.04 sec, transition often is obvious
- T wave directed opposite the terminal conduction delay
- ST segment unaltered except possibly depressed in V1-2
- Diagnosis of infarction Q waves and lateral ST depression is accurate
Right Bundle Branch Block

- May occur in normal persons, 1.8/1000
- Acute RBBB in anterior MI is worse prognosis
- Associated with hypertension, CAD, RHD, cor pulmonale, myocarditis incl Chagas’ disease, DCM, sclerosis of the cardiac skeleton and degenerative conduction system disease, chest trauma, transplant
- LVH more difficult to diagnose if RBBB
- RVH maybe if R prime in V1 >15 mm, not good sensitivity or specificity
- Incomplete RBBB: usually the RBB is pathologically normal
Left Anterior Fascicular Block

• “…the left bundle branch “system” is composed of a main stem and two main divisions”

Rosenbaum MB. J Electrocard. 1969;2:197
Fig. 1. “Divisional” LBBB and “trifascicular” heart block produced experimentally in the dog. a) Control tracing. b) Pure LAH, after cutting the anterior division of the left bundle branch. c) “Divisional” LBBB after subsequently cutting the posterior division. d) The right bundle branch is also cut, and “trifascicular” complete heart block is produced.

Rosenbaum MB. J Electrocard. 1969;2:197
Left Anterior Fascicular Block

Fig. 2. Pure LPH, “divisional” LBBB and “trifascicular” heart block produced experimentally in the dog. a) Control tracing. b) Pure LPH, after cutting the posterior division of the left bundle branch. c) “Divisional” LBBB, after subsequently cutting the anterior division. d) The right bundle branch is also cut, and “trifascicular” heart block develops.

Rosenbaum MB. J Electrocard. 1969;2:197
Left Anterior Fascicular Block

Fig. 3. Typical, uncomplicated LAH. QRS close to $-60^\circ$ $Q_1 S_{III}$ and QRS interval of 0.08 sec. Notice the deep S wave up to $V_6$ and the lack of Q wave in $V_1 - V_6$. Tracings of this kind are considered by some authors as indicating counterclockwise rotation of the heart on its longitudinal axis because of the presence of $Q_1 S_{III}$ and by others, as indicating clockwise rotation because of the leftward shift of the transitional zone. Actually, neither one is present for both are due to LAH.

Rosenbaum MB. J Electrocard. 1969;2:197
Left Anterior Fascicular Block

Fig. 4A. A case of intermittent LAH. LAH (beats 3, 4 and 5 in lead I-II; 3 and 4 in lead III) appears when the heart cycle shortens, and disappears when it lengthens.

Rosenbaum MB. J Electrocard. 1969;2:197
Left Anterior Fascicular Block

Rosenbaum MB. J Electrocard. 1969;2:197

Fig. 4B. Same case as in 4A. In each lead the first beat is without and the second with LAH. LAH shifts ΔQRS from 0 to −60°; widens the QRS interval from 0.08–0.09 to 0.10–0.11 sec; slightly increases the Q wave in leads aVL and III; diminishes the R wave in V₅ and V₆; and simulates left ventricular hypertrophy in aVL.
Left Anterior Fascicular Block

Rosenbaum MB. J Electrocard. 1969;2:197

Fig. 4B. Same case as in 4A. In each lead the first beat is without and the second with LAH. LAH shifts ÂQRS from 0 to −60°; widens the QRS interval from 0.08–0.09 to 0.10–0.11 sec; slightly increases the Q wave in leads aVL and III; diminishes the R wave in V₅ and V₆; and simulates left ventricular hypertrophy in aVL.
Left Anterior Fascicular Block

Rosenbaum MB. J Electrocard. 1969;2:197

Fig. 4C. VCG from the same case as in 4A-B, with and without LAH. Notice how LAH (right side of picture) produces an initial force pointing inferiorly and to the right, and main QRS forces superiorly and to the left.
Fig. 5A. “Physiological” LAH caused by aberrant ventricular conduction of atrial premature beats (the second beat in each lead), in a healthy 12 year-old boy. LAH shifts the QRS from $+35$ to $-30^\circ$, barely widens the QRS interval from 0.07 to 0.08 sec. and obliterates the Q wave from $V_6$. Besides, it simulates left ventricular hypertrophy in the extremity leads and to a lesser degree in the precordial leads.

Rosenbaum MB. J Electrocard. 1969;2:197
Left Anterior Fascicular Block

Rosenbaum MB. J Electrocard. 1969;2:197
Fig. 6A. “Physiological” LAH caused by aberrant ventricular conduction of atrial extrasystoles (the second beat in each lead), in a 65-year-old man with arterial hypertension and left ventricular hypertrophy. In addition to the superior shift of the main QRS forces, LAH produces signs of “left ventricular strain” in the standard leads.

Rosenbaum MB. J Electrocard. 1969;2:197
Left Anterior Fascicular Block

Fig. 6B. Same case as in 6A. Eight different and progressive degrees of LAH.

Rosenbaum MB. J Electrocard. 1969;2:197
Fig. 7. Transient LAH due to an acute anteroseptal infarction. *Top:* Acute anteroseptal myocardial infarction. AQRS: $+60^\circ$; QRS Interval: 0.08 sec. *Middle:* Two days later LAH has developed. AQRS: $-60^\circ$; QRS interval: 0.10 sec. LAH conceals some of the signs of the anteroseptal infarction. *Bottom:* One day later, LAH has vanished.
Other IVCD

• LAFB: qR in left leads, rS in inferior leads, often PPRWP, persistent S in V5-6, often T wave more upright in inferior leads (discordant), may diagnose concomitant inferior MI if peak of R in aVR is later than aVL (counterclockwise loop), but R in aVR and aVL must be terminal

• AFB can cause false positive voltage for LVH in I and III >25 mm

• LAD present (<-30) in 1.9% of normals
Other IVCD

- LPFB: rS in I and aVL, and qR in III and aVF, QRS axis >90 and <180 and QRS <0.12 sec, left precordium may give RS complex – seldom found in absence of RBBB
- Mid-septal fascicles: uncertain
- Bilateral BBB, alternating BBB
- Bifascicular is RBBB plus either AFB or PFB
- Trifascicular block, add FAV to bifascicular
- Periinfarction block: terminal QRS delay directed toward the infarcted area
- Nonspecific IVCD: wide QRS without RBBB or LBBB
Causes of Left Axis Deviation

Surawicz B et al. P.109, 2001
Left Anterior Fascicular Block

RBBB plus LAFB
Unknown 1
LAE with deep P in V1 and V2 and biphasic in V3
Atrial repolarization in V1 and II
Large voltage
Prominent upright T waves, but inverted U waves in V5 and V6
27 yo man short of breath, with sinus tachycardia, QRS duration 0.10, axis –30, LAE, poor R progression, diffuse T abnormality
45 yo woman wt 250, sinus rhythm rate 90, LAE, QRS 0.15 sec, ?lateral wall MI, PVC’s with triplet or quadruplet
28 yo man, PR 0.15, QRS 0.08, axis 85, LAE but no other problem. Possible MS, possible acute MR, less likely myocarditis or DCM.
32 year old woman from renal clinic. Notice leftward axis. LAE, LVH secondary repolarization changes not fully developed.
52 year old man with criteria for RAE and inferior MI and anterolateral MI and LVH. Clinically probably LA enlargement
44 yo man in MICU. LAE unequivocal. QRS 0.11, probable significant LVH. Normal standard. Notch in P in II, PVC’s ST-T abnormal
54 yo woman. Clear LAE. No LVH and no clear RVH, but QT is long
Unknown 9

46 year old woman with sinus tachycardia and LAE and nonspecific ST and T wave abnormality and QRS duration of 0.09
38 year old woman, 62 inches, 200 lb, read as inferoposterior MI.
Actually RVH without RAE or LAE. This would be type A-B.
49 year old man with chest pain in the emergency department. Sinus tachycardia (not flutter), LAE, RBBB, ?inferior MI, ?posterior MI
58 year old man with LAE and posterolateral MI, not RVH, because in I is a QS not an RS. Later developed RBBB too!
58 year old man with LAE and posterolateral MI, not RVH, because in I is a QS not an RS. Later developed RBBB too!, now all leads at half std
Unknown 13

LAE and IVCD. IVCD looks like RBBB in I, but LBBB in V1, large voltage but no LVH because of IVCD. Anterolateral MI? CHF?
37 year old man, QRS duration 0.10. R peak time in V6 is 0.05 and in V5 is 0.05. LVH? – I think so, but not by strict application because he is less than 40 years old.
45 year old woman with no atrial abnormality, but right axis deviation and classic abnormality in V1 with T inversion – RVH. 4 months earlier the RVH was type A and there was also RAE ?embolism
56 year old man in preparation for outpatient ETT! Sokolow-Lyon criterion of 25 mm I and III and repolarization abnormality and QRS duration of 0.10 and LAE and normal R peak time
64 year old woman with LAE and LVH by Sokolow-Lyon or Cornell and dramatic repolarization abnormality, QRS duration 0.07. Voltage lower 4 years later with QRS duration 0.11.
ABNORMAL ECG

Meds: Unknown

Referred by: SEPDHAM 15597

Unconfirmed

** All leads at half standard **
Half standard. World class LVH. 44 year old man.
35 year old woman, echo 8 months prior mild LVH and mildly low EF. LVH by Sokolow-Lyon, but less than 40 years old. QRS 0.12, R peak time abnormal in V5
90 year old woman, peaked P in II, rightward QRS axis without RVH, RAE in V1, two PAC’s, QRS duration 0.07, T flattening.  ?True?
45 year old man, with lateral wall MI and consequent RAD and large voltage suspicious for LVH and clearcut LAE and nonspecific ST and T probably from LVH, maybe anterior MI too
42 year old woman, dramatic RAE, and axis about 80 and no criteria for RVH. Sinus tachycardia. RAE more with pressure and LAE more with volume load (no RAE in ASD)
10 year old boy; pediatric cardiologist diagnosed RAE, RAD and RVH and the T inversion in V4 is abnormal but not in V1-3.
76 year old man, with sinus tachycardia, RAE, LVH, AFB, doesn’t add up well.
74 year old woman in MICU, echo normal EF, mild LVH, normal LA size, mild RAE. The significant S in I with indeterminate axis is new from one day before.
54 year old man, first degree AV block, LAE, IVCD, possible anterolateral MI and inferior MI and RBBB and RVH, no anterior hemiblock since terminal R in aVF