April 2018 Tracings

Questions?
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81 Year Old Man

April 2018

Tracing 1

- (AVDIS) + AV dissociation, mean V-rate = 65 [Now - -
  Present]
- (RBBB) = Right bundle branch block [Remains] - - -
- (SDiLA) + Nonspecific Lateral region ST depression - -
  [Now Present]
- (LAE) - [Now Absent] Left atrial enlargement
  - ABNORMAL ECG -

- PR 138
- QRS 460
- QTc 478
- P IND
- QRS 50
- T -13
The computer interpretation is partially correct, but it has ignored important points. P waves are regular at 120/min., but arise in a low atrial focus. It is evident that not all that the waves are conducted and, thus, there is “some degree” of A-V block -- probably 2:1 transmission. An accelerated junctional focus appears at a rate of 70/min. and remains in command until the end of the tracing. The ladder diagram depicts the problem. When the AV bridge is depolarized, there is a lengthy recovery (R-P) resulting. The prolonged refractory interlude prevents transmission of the majority of the atrial impulses. However when a P wave is late enough after the QRS complex (R-P interval of 0.60 sec.) conduction occurs. This unusual, but important variety of A-V dissociation has been termed “block -- acceleration dissociation”, and may be due to digitalis excess.
Tracing 2

84 Year Old Woman

Your observations include?
Except for sinus bradycardia, the aberrantly conducted APC, and possible anterior M.I., the 12 lead tracings is rather boring!! The evident interest is -- what are the high-frequency “spikes”???
Measuring carefully, you will note that they recur regularly at a rate of +/- 120/min and have nothing to do with the underlying P-QRS.

Answer: This nice lady forgot to turn off her “TENS” unit (transcutaneous electrical nerve stimulator) before her EKG was recorded. Axiom: “Beware the pernicious patient”
P.S. If you attempted to interpret this tracing as some kind of malfunctioning cardiac pacemaker --- FOR SHAME!!!!
Tracing 3

Kindly make some observations on this tracing. Careful analysis can allow you (ALA Sherlock Holmes) to make an exact diagnosis.
Elementary!!

1. There is excessive precordial voltage due to LVH.

2. The short Q to onset of the T wave is indicative of hypercalcemia.

The combination of hypercalcemia with LVH is an obvious tipoff to a patient with coexisting pheochromocytoma and hyperparathyroidism, and allows the EKG judgment that the patient has "multiple endocrine adenomatosis -- Type 2" (Sipple syndrome). The observation demands careful examination for another feature of the syndrome: medullary carcinoma of the thyroid. This patient had bilateral pheos, thyroid CA and parathyroid hyperplasia!

Stay alert!!!
70 year old woman

Tracing 4

Do you agree with computer?
We will all make errors of omission and commission, but the computer commits both with this tracing. The sinus impulses are conducted with LBBB and are wedded to bogeminal VPCs. The morphology of these simulates “RBBB” with right axis deviation, and prompts the computer to “consider RVH.”
94 Year old woman

Tracing 5

Agree? Anything to add?

--- AXES ---
P 57
QRS 206

PR 151 (NSR). Normal sinus rhythm, rate 67
QRS 124 (BIFBP). Bifascicular block: RBBB & LFPB
QT 410
QTc 433

- ABNORMAL ECG -
The computer is not programmed to exclude the RBBB contribution from frontal plane axis determination. It provides an axis of 206º and interprets the right axis deviation as due to left posterior fascicular block.

In reality, the “pre-blocked axis” is 0º. Importantly, the prominent initial R waves in V1-2 and the q waves in V3-6 are consistent with a posterolateral myocardial infarction.
Evidence of Ant. M.I.?

How might you erase it?
The upper tracing shows low QRS voltage in both frontal and horizontal planes. Absence of R waves in V1-3 is consistent with anterior M.I.. Could this be artifactual?

If the decreased voltage is due to hyperinflation pulmonary disease, the heart would be trailing the low-lying diaphragm. Thus, appropriate precordial electrode position would be “too high” for the cardiac position.

The lower tracing was recorded three interspaces below the normal position and eliminates the concern about an anterior M.I. Locally, this exploration is called “precordial mapping”.

Evidence of Ant. M.I.?

How might you erase it?