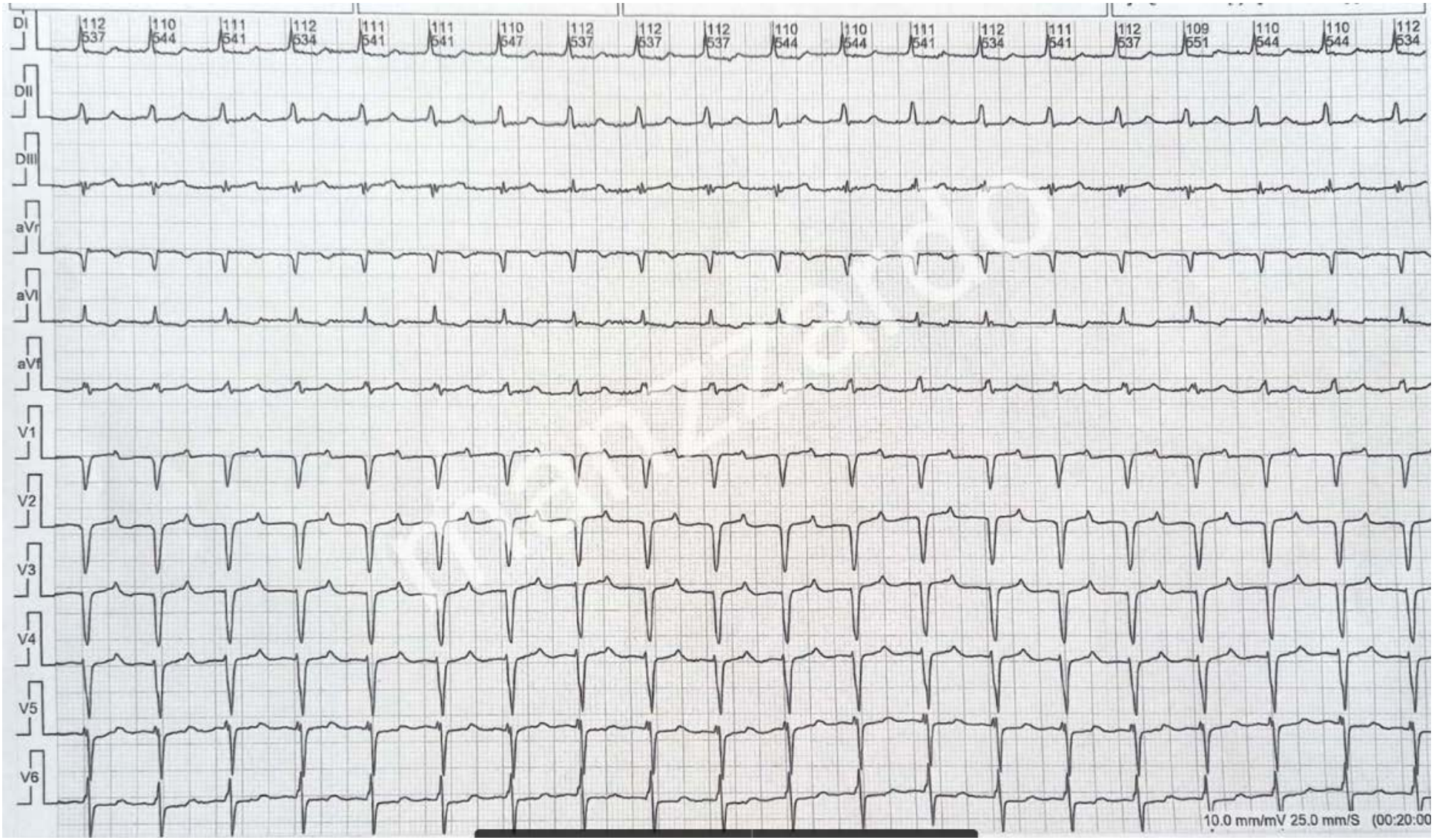
Spanish
Mujer de 81 años de edad, hipertensa, diabética tipo 2, con sobrepeso, y portadora de fibrosis pulmonar que consulta por astenia, palpitaciones, y disnea CF II NYHA desde aproximadamente 10 días.
AP: HTA tratada con enalapril 10 mg y amlodipina 10 mg. Diabetes tipo 2 con dieta y metformina 1 g /día y Fibrosis pulmonar diagnosticada por neumonólogo.
Ex físico, R2 hiperfonética, no ritmo de galope, soplo sistólico 2/6 base sin irradiación, sin soplos carotídeos. No palpo choque de la punta, paciente con sobrepeso.
Pulmones: Roncus en ambas basales, no rales, buena entrada de aire bilateral. No palpo hígado y tiene edemas MMII 2/4 hasta mitad de piernas.
No tengo exámenes complementarios.
El 2° post MSCD muy leve, pues valsalva fue negativa. El resto es la evolución observada.
Post MSC, trastornos de conducción de un ritmo auricular conducido?
O escapes ventriculares? Ver el eje de cada latido.
Espero vuestras opiniones, gracias!
Dr Juan Carlos Manzzardo MD Mendoza/ Argentina

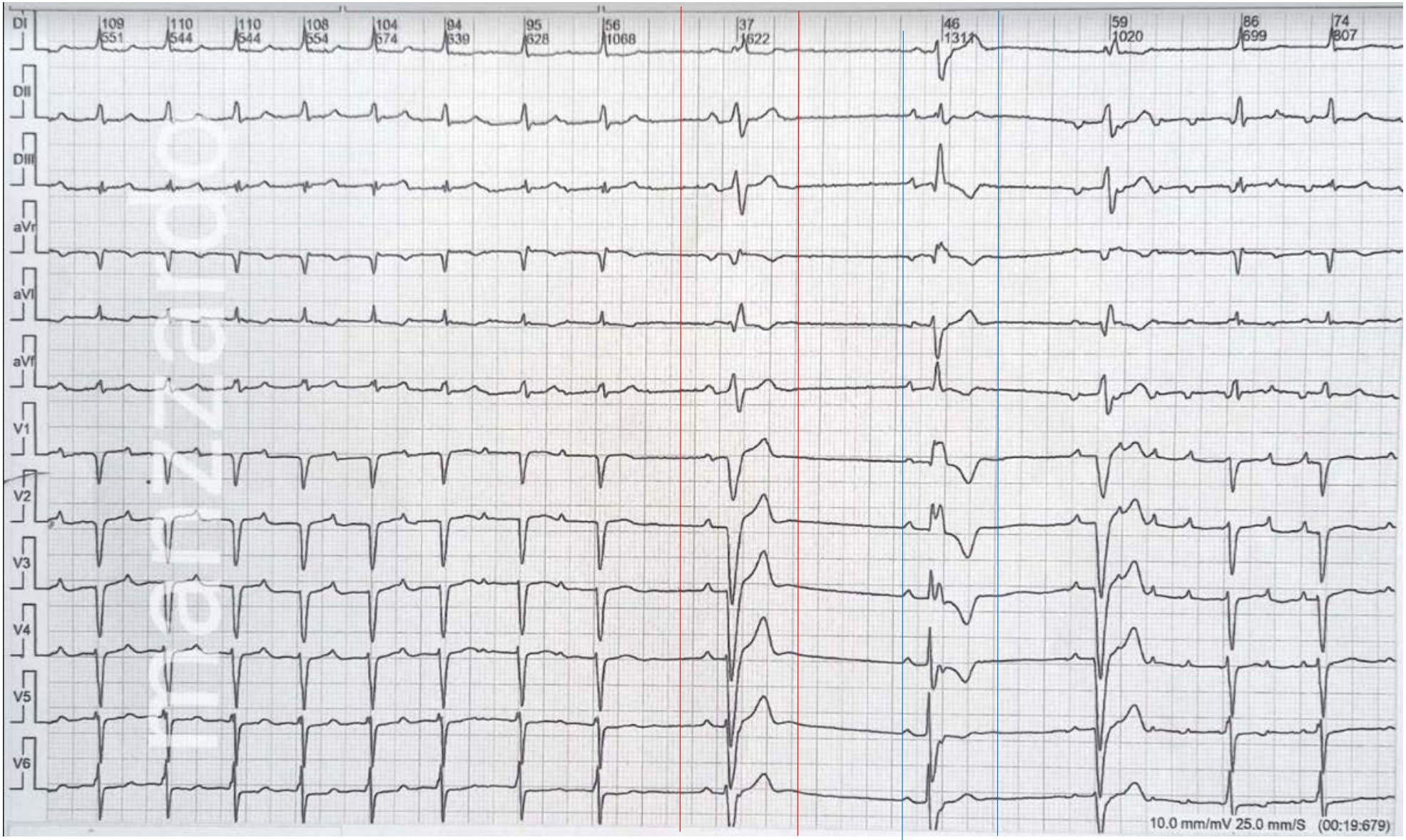
1) ECG basal
2) ECG post MSCD muy leve, pues valsalva fue negativa
3) ECG evolución en apenas minutos mientras permanecía en decúbito supino.
4) ECG evolución en apenas minutos mientras permanecía en decúbito supino.

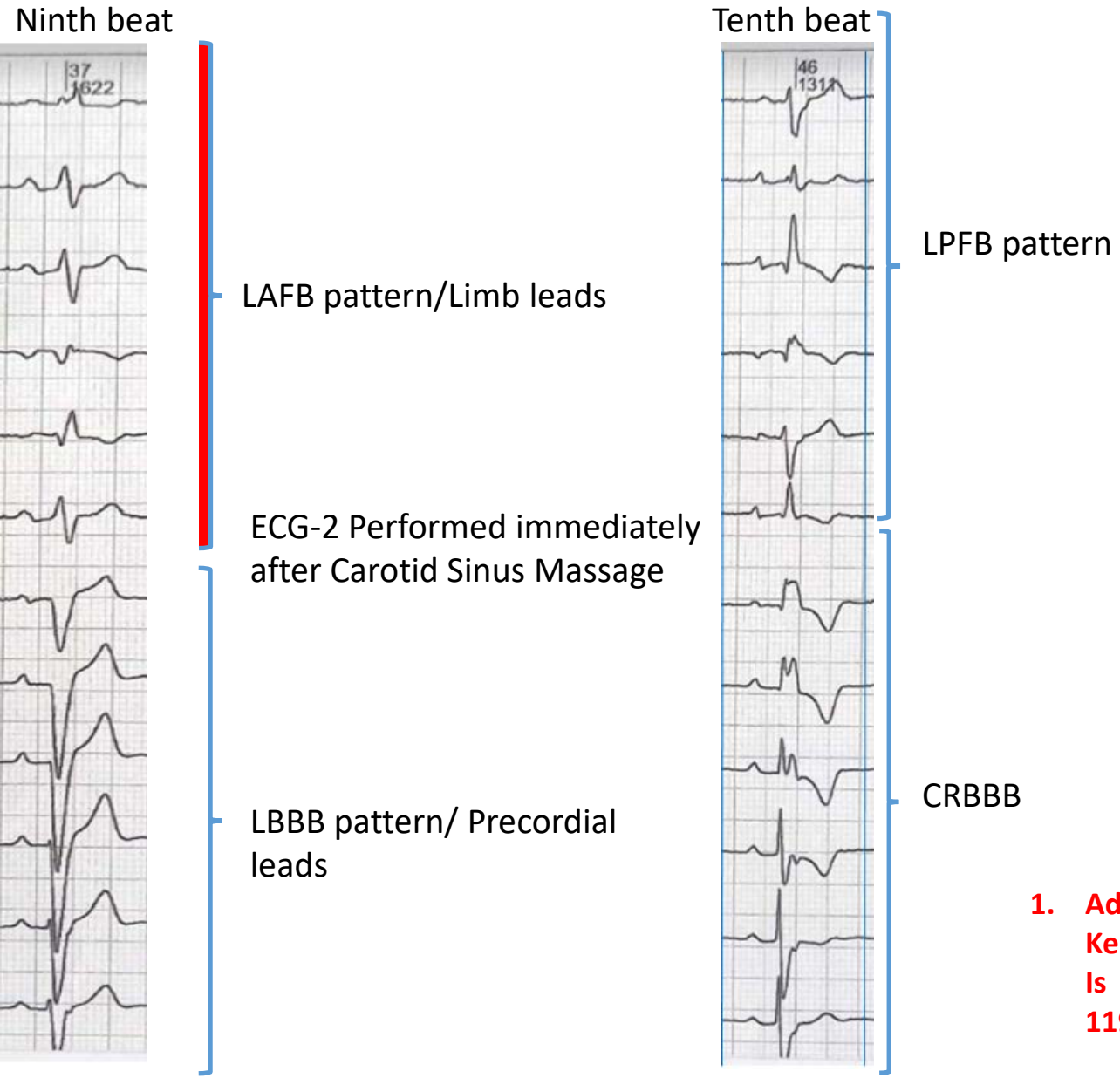
An 81-year-old woman, she has hypertension, type 2 diabetes mellitus, overweight (body mass index (BMI) over 25, and <30), and pulmonary fibrosis
She complain of asthenia, palpitations, NYHA II dyspnea during approximately 10 days.
Her hypertension was being treated with Enalapril Maleate 10 mg + anlodipine besylate 10 mg daily.
Her Diabetes was treated with diet and metformin 1 g/daily.
Physical examination: hyperphonetic R2, no gallop rhythm, 2/6 base systolic murmur without radiation, non-palpable ictus cordis, roncus in both bases, not rales, good bilateral air intake, absence of carotid murmurs.
I cannot palpate the liver and she has 2/4 MMII edema up to the middle of her legs.
I don't have complementary exams.
The 2nd ECG was performed immediately after Carotid Sinus Massage (CSM) which was very mild, since Valsalva was negative. The rest is the observed evolution. Post CSM, conduction disorders of a conducted atrial rhythm? or ventricular escape rhythm? See the QRS axis of each beat.
I await your valuable opinion,
Thank you! ECG-1 Baseline ECG
ECG-2 very mild post-MSC ECG, valsalva was negative
ECG3 evolution in just minutes while in the supine position.
ECG4 evolution in minutes aftere the third in the supine position.

Baseline ECG (1)



ECG-2 Performed immediately after Carotid Sinus Massage





Conclusion Alternating Bundle Branch Block (ABBB) is when both right bundle branch block (RBBB) and left bundle branch block (LBBB) patterns appear **on the same ECG or within a period of hours to days.**

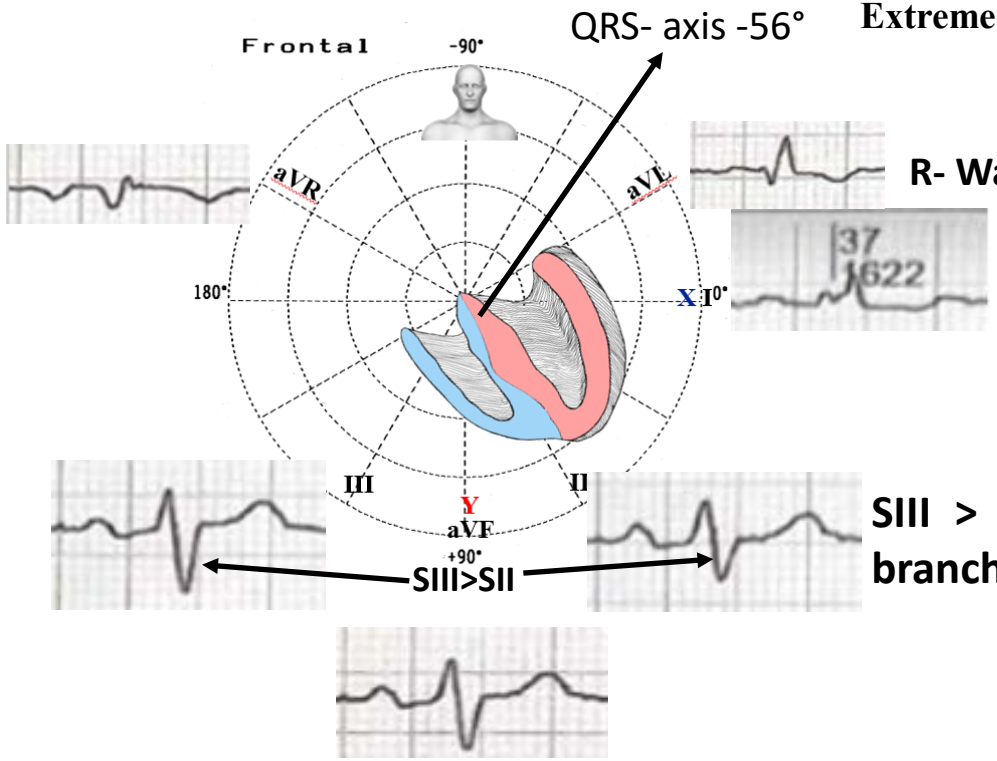
What is the mechanism of the alternating bundle-branch block pattern noted on the ECG?

1. Alternating RBBB and LBBB premature ventricular contractions
2. Alternating phase 3 block in the bundle branches
3. **Alternating phase 4 block in the bundle branches**
4. Interpolated premature ventricular contractions with alternating bundle branch morphologies(1)

1. **Aditya Saini 1, Santosh K Padala 1, Jayanthi N Koneru 1, Kenneth A Ellenbogen 1Alternating Bundle-Branch Block: What Is the Mechanism? Circulation . 2018 Mar 13;137(11):1192-1194. doi: 10.1161/CIRCULATIONAHA.118.033637.**

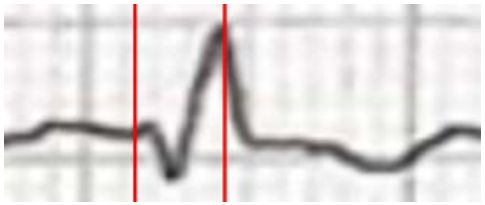
Ninth beat

LAFB pattern/ Limb leads



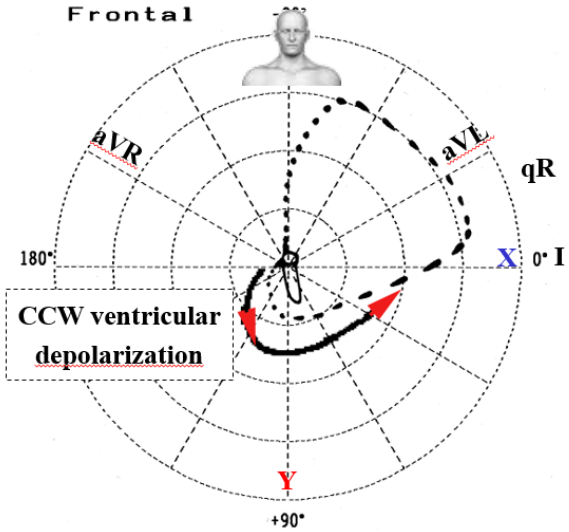
Extreme shift of SÂQRS (beyond -30° up to -90°). Some authors accept - 45°.

R- Wave Peak Time in aVL ≥ 45 ms;

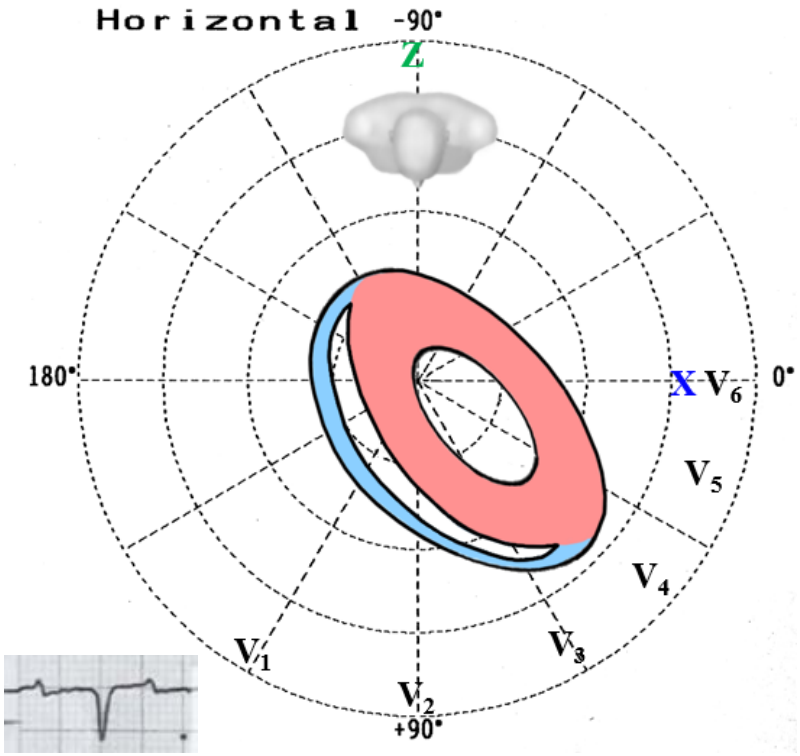


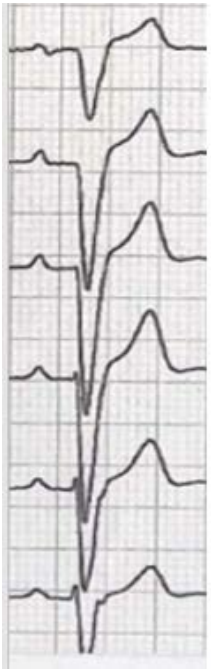
R- WPT

SIII > SII: this criterion differentiates it from RECD of the right branch and SI-SII-SIII syndrome, where SII > SII.

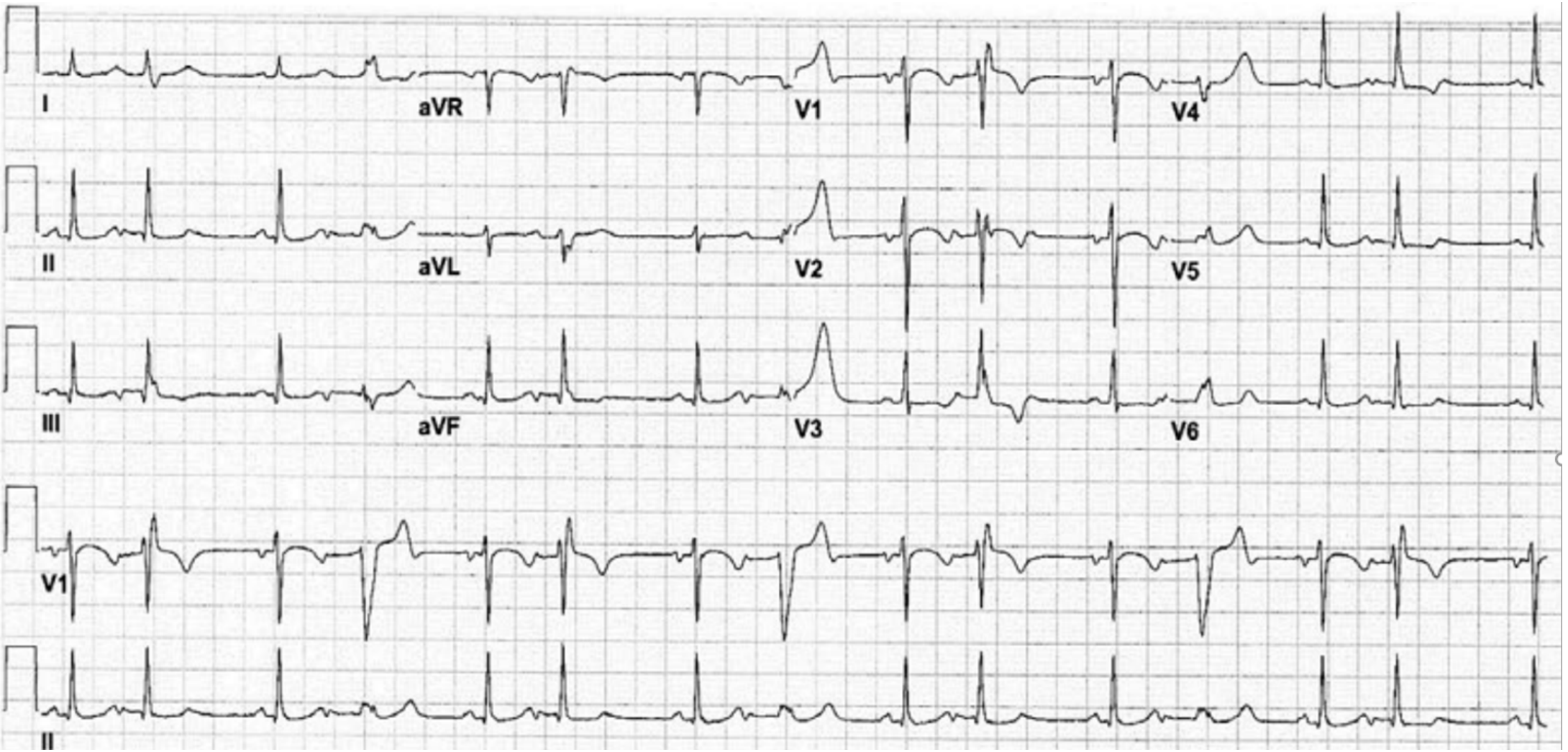


Ninth beat





Similar case from literture



. Twelve-lead ECG showing sinus rhythm with alternating right and left bundle-branch block pattern of ventricular conduction. See diagnosis in the next slide

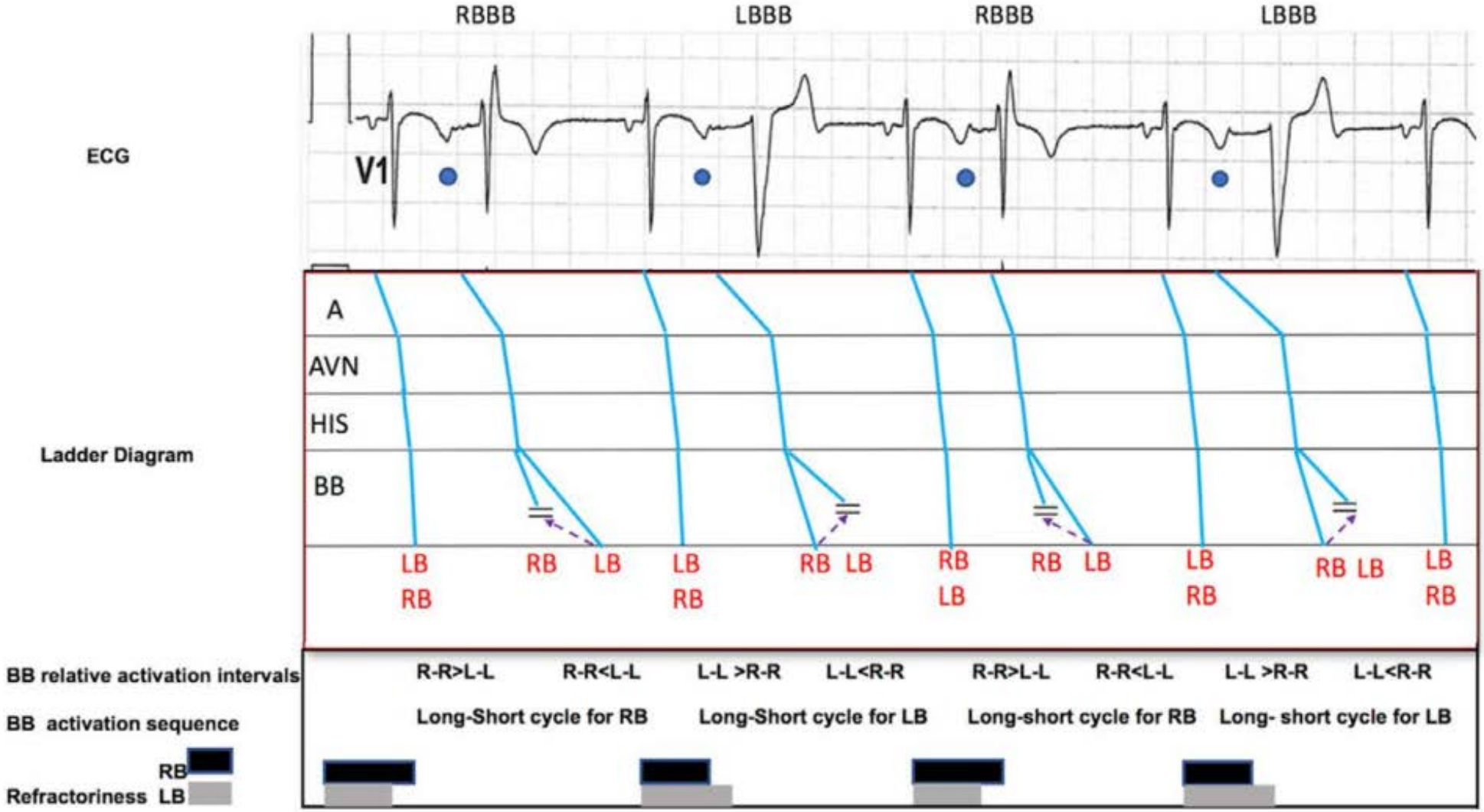
Irregular heart rhythm in this patient is the result of frequent atrial ectopic beats occurring in a bigeminal fashion.

P waves are seen within the preceding T waves during ectopy, and the atrial coupling interval is constant (440 ms), which confirms that these are PACs and not PVC. Each PAC conducts to the ventricles, and the resultant QRS complexes are wide with an alternating pattern of RBBB and LBBB aberrancy. As depicted in the single-lead ECG strip (precordial lead V1 in Figure 2 SILDE 11), the first P wave is a sinus P wave that is conducted normally down to the ventricles, as evidenced by a normal PR interval (120 ms) and narrow QRS duration. What follows next is a PAC (blue dot), which falls within the T wave and is able to conduct to the ventricles with a longer PR interval (200 ms) and RBBB aberrancy (PAC finds right bundle refractory and conducts down the left bundle). It is noteworthy that the block in RB may not necessarily be absolute, but is more likely a result of a relative delay of conduction between LB and RB. The third P wave is a sinus P wave and is conducted normally to the ventricles. The fourth P wave is a PAC occurring at the same coupling interval, but conducts to the ventricle with an even longer PR interval (280 ms) and LBBB aberrancy. This cycle of atrial bigeminy with alternating RBBB and LBBB aberrancy continues over the remaining strip. The ladder diagram (Figure 2 Slide 11) depicts our explanation for the ECG findings.

Aberrant conduction is present when there is an alteration in the QRS contour of supraventricular beats resulting from

impulse transmission during periods of physiological refractoriness or depressed conductivity. This can be acceleration-dependent (phase 3) aberrancy or deceleration-dependent (phase 4) aberrancy. The aberrant conduction in this case is a result of prematurity and is thus acceleration-dependent (phase 3 block). In addition, the refractory periods of the bundle branches are dependent on the preceding cycle lengths. These properties are critical in explaining the variability in aberrancy. Because, under normal circumstances, the refractory period of RB is longer than that of LB, RBBB aberrancy is more common, specifically in patients without conduction system disease. In this case, despite the identical coupling interval of the PACs, there is an alternating shift of relative conduction delay between the bundle branches. The second P wave is a PAC that conducts down the LB (resulting in a RBBB pattern), then via transseptal conduction conceals retrogradely into the RB, resulting in delayed activation of the distal RB. This is followed by a sinus beat that conducts down both the bundle branches without aberrancy. The sinus beat establishes a longer cycle length for the LB than the RB (because the LB was activated earlier on a prior premature beat in comparison with RB, thereby resulting in greater prolongation of the LB refractory period than RB. The subsequent PAC then finds the LB relatively refractory because of the long-short sequence in LB and preferentially conducts down the RB (resulting in a LBBB pattern), then conceals retrogradely into the LB. This pattern of

long-short cycles in the distal His-Purkinje system results in alternating bundle-branch block. This phenomenon of alternating bundle-branch block has been previously reported1–3 and is the result of relative functional delay of conduction in the distal His-Purkinje system during prematurity and does not necessarily indicate the presence of a diseased conduction system.



Concerning the alternating bundle-branch block, and we would like to offer some opinions. The ECG was characterized by an alternating left bundle-branch and a right bundle-branch block. This finding indicated an incomplete block. Consequently, we must consider whether the block is intraventricular. Because this patient had a history of

1. **Type 2 Diabetes: Main** risk factors for coronary artery disease **!!**
2. **Systemic hypertension: Important** risk factors for coronary artery disease!!
3. **Overweight: Androide pattern? She is a woman.** Obesity and overweight are projected to become main risk factors for coronary artery disease (CAD) The waist is measured at the level of umbilicus!! Anthropometric measures have been determined as important measurements for risk assessment of cardio-metabolic diseases, according to the results of large studies such as the well-known Framingham survey (2)
4. **Advance Age**: risk factors for coronary artery disease!!
5. **Pulmonary fibrosis** (secondary to tobacco????): risk factors for coronary artery disease!!

It is important to identify the myocardial damage caused by the PROBABLE CORONARY STATUS. It would be informative to provide the echocardiography for that patient to determine whether the heart endured any structural changes. The study considered the electrophysiological causes of arrhythmia, but Juan Carlos Manzzardo should also consider **the real causes behind it.** Different etiologies require different treatment strategies!!!!.

1. **Aidin Baghbani-Oskouei, MD1 and Mehrzad Gholampourdehaki, MD1, Anthropometric measures and the risk of coronary artery diseaseCaspian J Intern Med. 2020; 11(2): 183–190. doi: 10.22088/cjim.11.2.183**
2. **Preis SR, Massaro JM, Hoffmann U, et al. Neck circumference as a novel measure of cardiometabolic risk: the Framingham Heart study. *J Clin Endocrinol Metab.* 2010;95:3701–10. [**

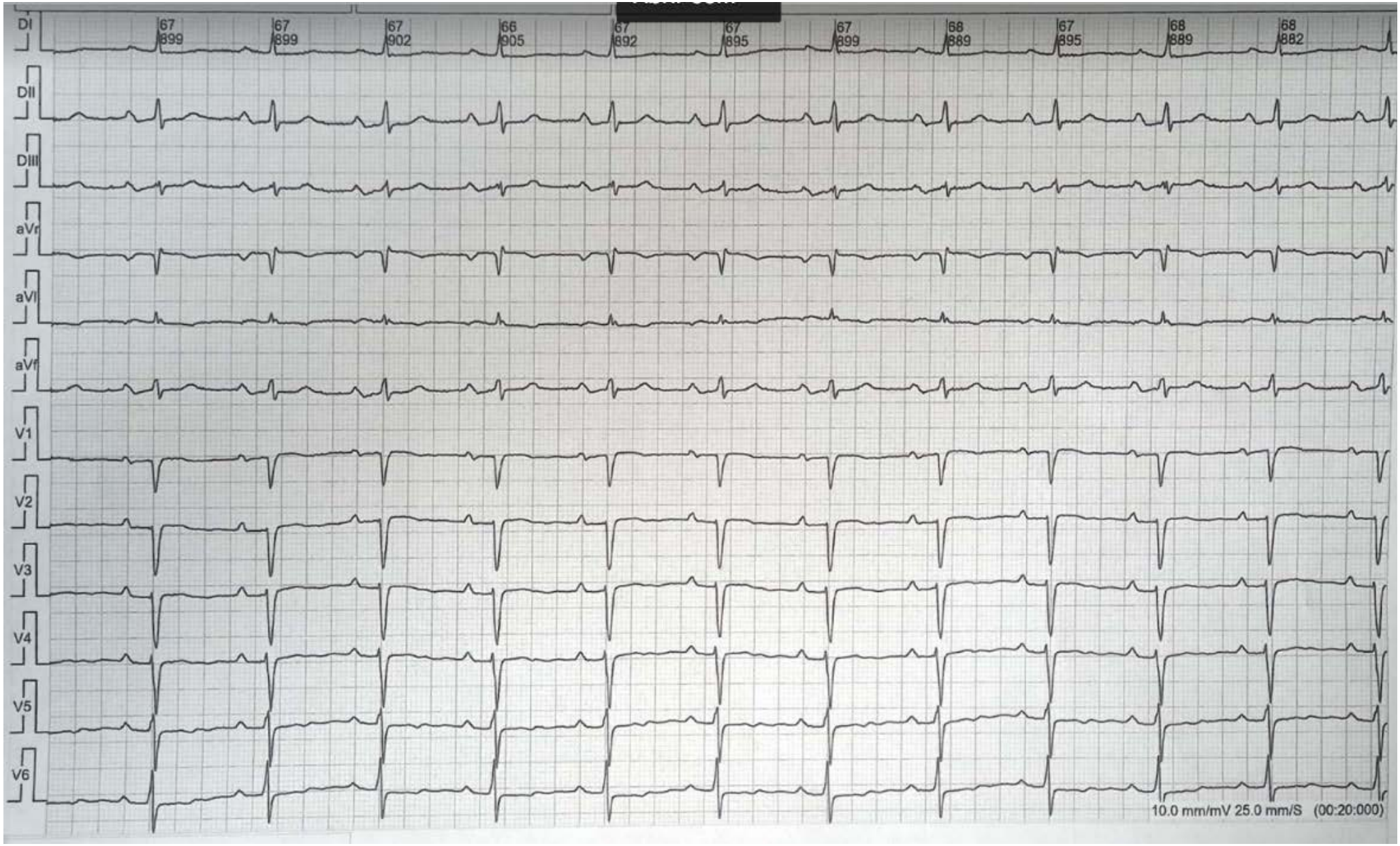
Risk factors for coronary artery disease classify into modifiable and non-modifiable risk factors.

A 2019 article indicated that age, sex, and race captured 63% to 80% of prognostic performance, while modifiable risk factors contributed only modestly. Yet, control of modifiable risk factors led to substantial reductions in CAD events.(1) Non-modifiable risk factors are discussed first:

1. Age: CAD prevalence increases after 35 years of age in both men and women. The lifetime risk of developing CAD in men and women after 40 years of age is 49% and 32%, respectively(2)
2. Gender: Men are at increased risk compared to women.
3. Ethnicity: Blacks, Hispanics, Latinos, and Southeast Asians, are ethnic groups with an increased risk of CAD morbidity and mortality.[(3)
4. Family history: Family history is also a significant risk factor. Patients with a family history of premature cardiac disease younger than 50 years of age have an increased CAD mortality risk. A separate article indicated that a father or brother diagnosed with CAD before 55 years of age, and a mother or sister diagnosed before 65 years of age are considered risk factors.

1. **Pencina MJ, Navar AM, Wojdyla D, Sanchez RJ, Khan I, Elassal J, D'Agostino RB, Peterson ED, Sniderman AD. Quantifying Importance of Major Risk Factors for Coronary Heart Disease. Circulation. 2019 Mar 26;139(13):1603-1611.**
2. **Sanchis-Gomar F, Perez-Quilis C, Leischik R, Lucia A. Epidemiology of coronary heart disease and acute coronary syndrome. Ann Transl Med. 2016 Jul;4(13):256.**
3. **Carnethon MR, Pu J, Howard G, Albert MA, Anderson CAM, Bertoni AG, Mujahid MS, Palaniappan L, Taylor HA, Willis M, Yancy CW., American Heart Association Council on Epidemiology and Prevention; Council on Cardiovascular Disease in the Young; Council on Cardiovascular and Stroke Nursing; Council on Clinical Cardiology; Council on Functional Genomics and Translational Biology; and Stroke Council. Cardiovascular Health in African Americans: A Scientific Statement From the American Heart Association. Circulation. 2017 Nov 21;136(21):e393-e423.**

ECG-3


DI
DII
DIII
aVr
aVl
aVf
V1
V2
V3
V4
V5
V6
67 899
67 899
67 902
66 905
67 892
67 895
67 899
68 889
67 895
68 889
68 882
10.0 mm/mV 25.0 mm/S (00:20:000)

ECG-4 accomplished few minutes after ECG-3