Nuclear Cardiology

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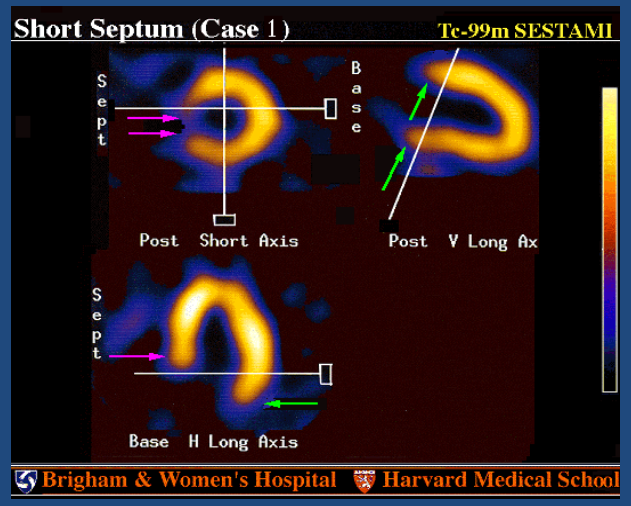
Varying Spectrum of Normal SPECT Images

- Most apical short-axis slices:(without ventricular cavity): inhomogeneous uptake, Coffee bean appearance
- Apical thinning
- Septum:
  - Uptake: Septal < lateral wall
  - Less basal inferoseptal uptake in male patients
  - Short Septum



The “11 o’clock” defect

- linear defects extending through the myocardium in the short-axis tomograms at approximately :
  - 11 o’clock and
  - 7 o’clock.

- precise cause of these defects is not known.

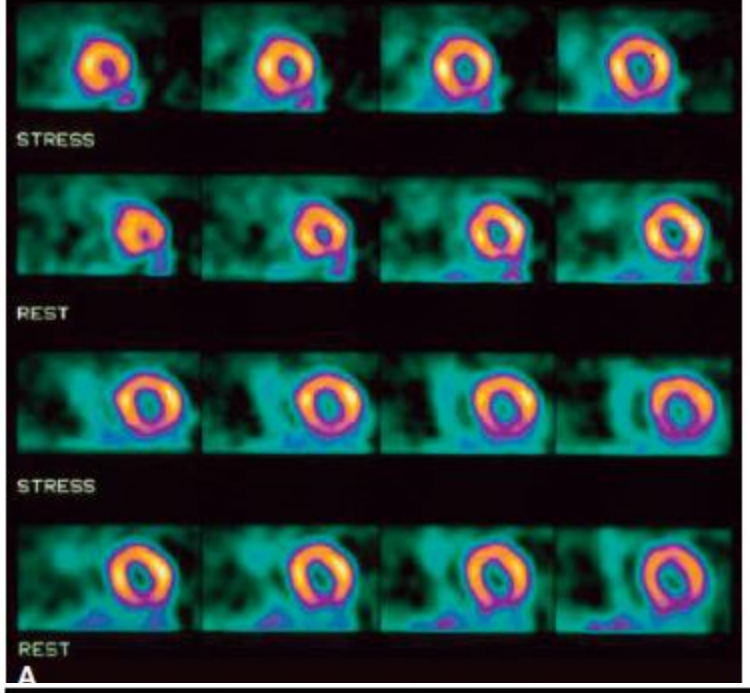
- almost invariably located adjacent to **the insertion points of the RV myocardium** as it joins the septum
  - they are most likely either anatomic variants or
  - related to attenuation by the right ventricle myocardium and/or blood pool.

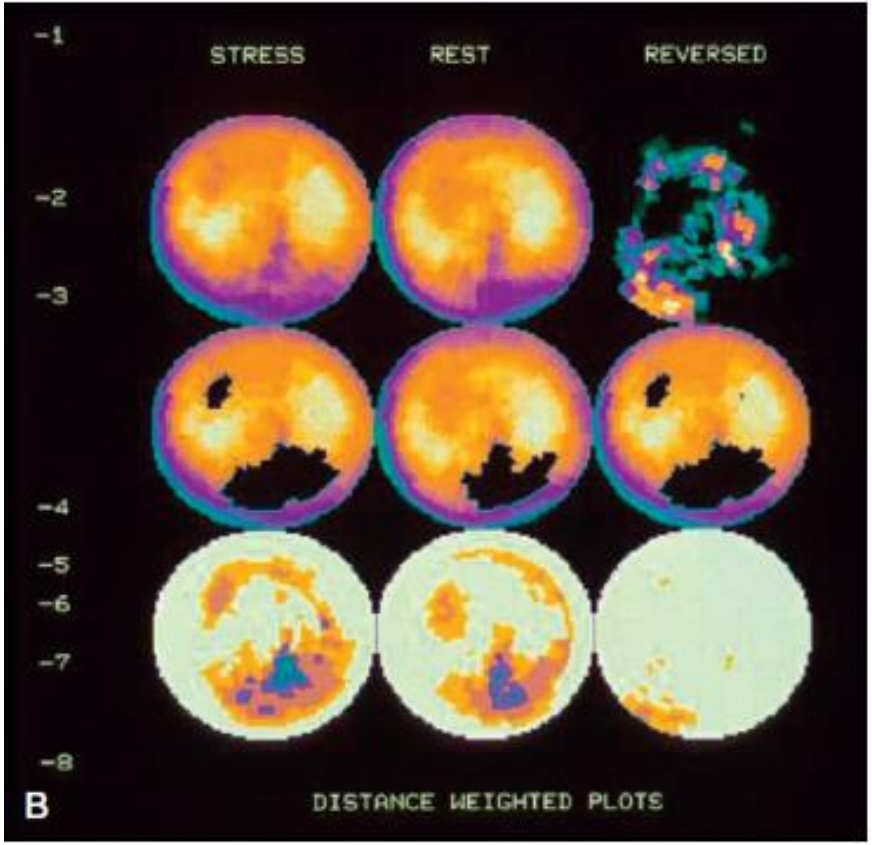
Artifacts on SPECT images

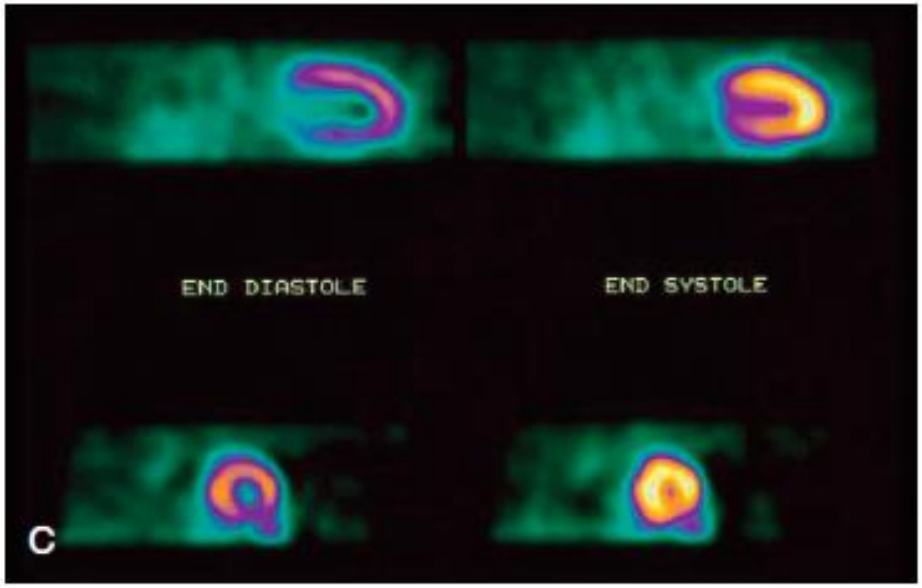
- Soft Tissue attenuation
  - DA
  - Breast A
  - Lateral Chest wall fat attenuation
- Upward Creep
- Patient Motion
- COR and Uniformity
- Orbit related Artifacts
- Caused by reconstruction and display
- Intense Subdiaphragmatic activity
- Artifacts on Polar maps

Breast Artifacts

- average body habitus:
  - **anterolateral wall**
- **large, pendulous breasts,**
  - **the breasts lie adjacent to the lateral chest wall: lateral attenuation artifact.**
- **very large, pendulous breasts:**
  - **may inferior or inferolateral defects.**
- very large breasts,
  - the breast tissue may overlie the entire LV.
  - attenuation artifact may be diffuse and less discrete or may primarily involve the apex.







Technetium-99m-sestamibi stress and rest single-photon emission computed tomography (SPECT) tomographic slices (A) and polar coordinate plots (B) demonstrate an apparent mild relative decrease in tracer concentration in the inferior wall. Gated end-diastolic and end-systolic midventricular short-axis and vertical long-axis tomograms (C) demonstrate normal inferior wall motion and wall thickening, thus favoring an attenuation artifact rather than inferior infarction as a cause of the fixed defect.

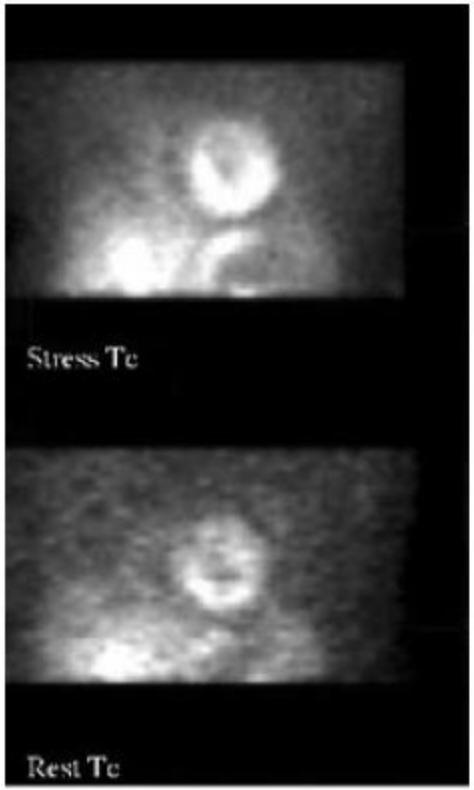
- **Quantitative analysis in cardiac SPECT ???**
- **Left mastectomy:**
  - inferior perfusion defect incorrectly identified by quantitative analysis in women
  - This quantitative error can be circumvented by comparison of the patient's data to the normal male file instead

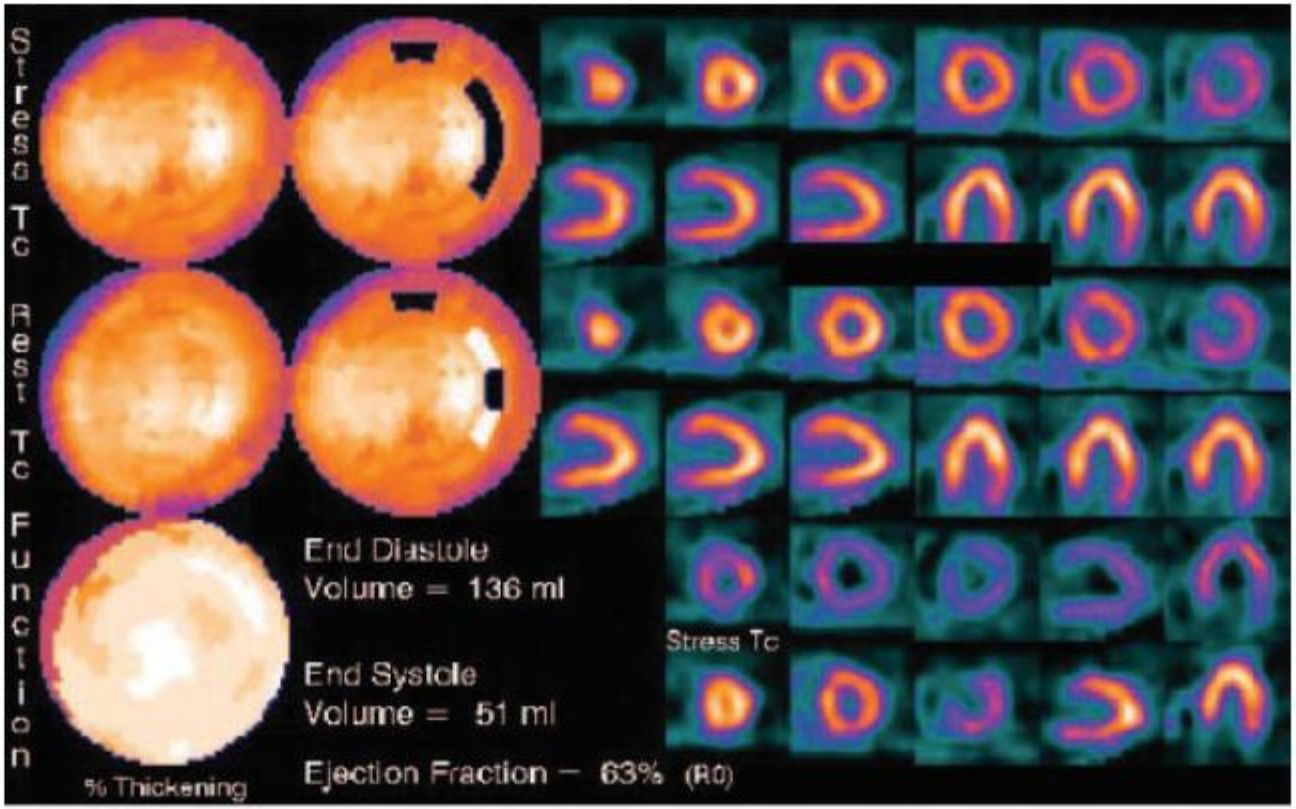
Lateral Chest-Wall Fat Attenuation

- In obese patients, there may be a considerable accumulation of fat in the lateral chest wall

- In obese individuals, lateral chest wall fat
  - not only attenuates photons emanating from the myocardium but also increases the distance between the heart and the scintillation detector.

  - attenuation artifact is usually more diffuse, often involving the entire lateral wall of the LV.





In planar projection images it is apparent that this female patient is very obese, with a considerable distance between the lateral border of the left ventricle and the left lateral chest wall. (b) Stress and rest tomographic images demonstrate a moderate decrease in tracer concentration in the basal half of the posterolateral wall of the left ventricle. As compared to the septum, the lateral wall is less intense. This is an abnormal scan finding, since the lateral wall should normally be more intense than the septum Gated tomograms demonstrate normal wall motion and wall thickening of the entire left ventricle, including that of the posterolateral wall. Quantitative analysis of wall thickening displayed in polar plot format similarly demonstrates normal posterolateral wall thickening, favoring lateral wall soft tissue attenuation artifact rather than scar as a cause of the fixed posterolateral defect.

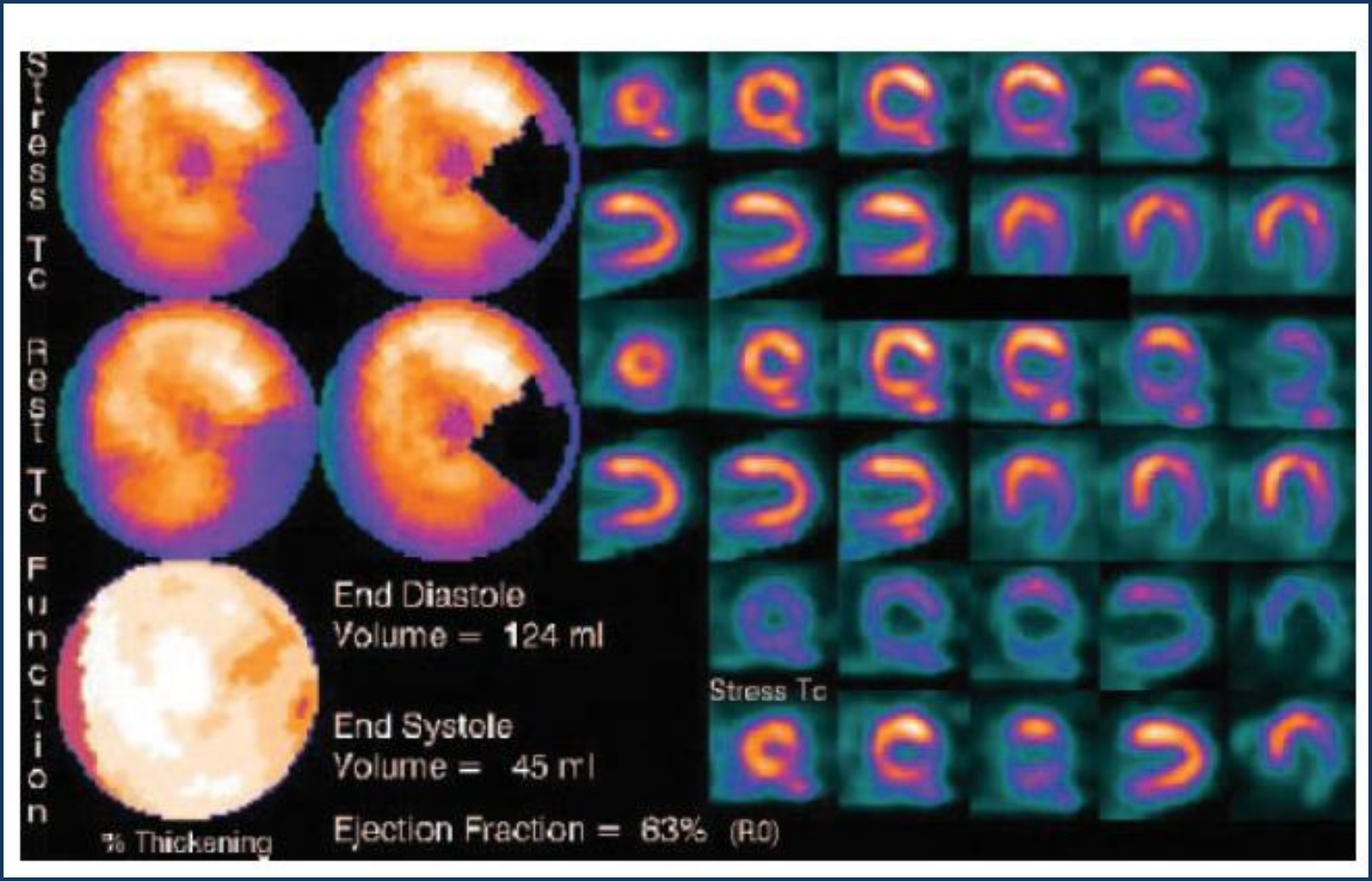
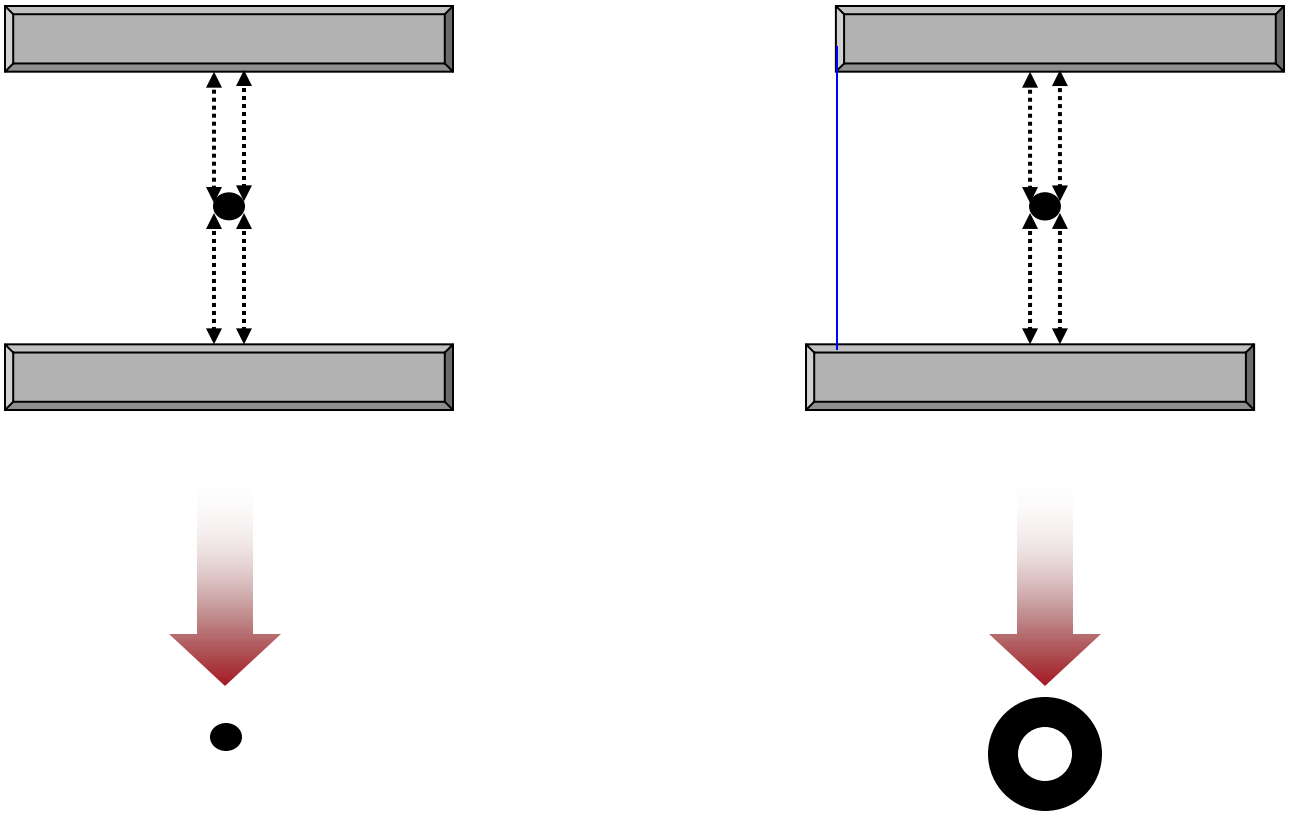


Figure 7.5 Stress and rest tomographic images demonstrate a marked fixed defect involving the basal half of the posterolateral wall of the left ventricle. Gated tomographic images demonstrate markedly decreased wall thickening of the fixed defect. However, posterolateral wall motion is relatively well preserved, most likely accounted for by the anterior translational motion of the heart during systole. Quantitative analysis of wall thickening displayed in polar plot format demonstrates markedly decreased posterolateral wall thickening, consistent with myocardial scarring rather than attenuation artifact as a cause of the fixed posterolateral defect.

Left arm attenuation

- The arm should be positioned as posteriorly as possible
- Nevertheless, even with these precautions, **lateral wall attenuation artifacts** are frequently encountered
- These attenuation artifacts may be particularly severe because not only soft tissue but also bone attenuates photons.
- It is particularly important to determine that the position of the arm is identical in the stress and rest images.

COR offset



- **The center of rotation (COR) parameter is a measure of the alignment of the opposite views (e.g., posterior versus anterior or right lateral versus left lateral) obtained by the SPECT system.**
- **The COR must be accurately aligned with the center of the acquisition matrix in the computer**

- **If the COR is misaligned, then a point source would be seen as a**

“donut” on the image .

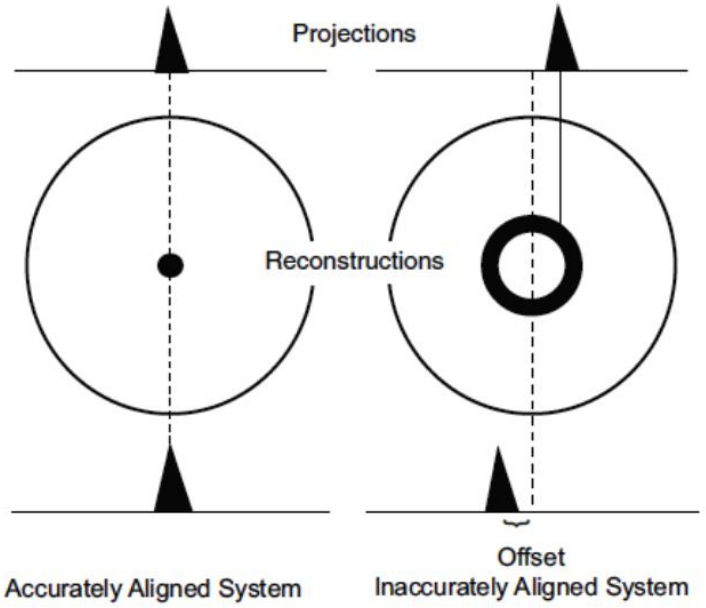
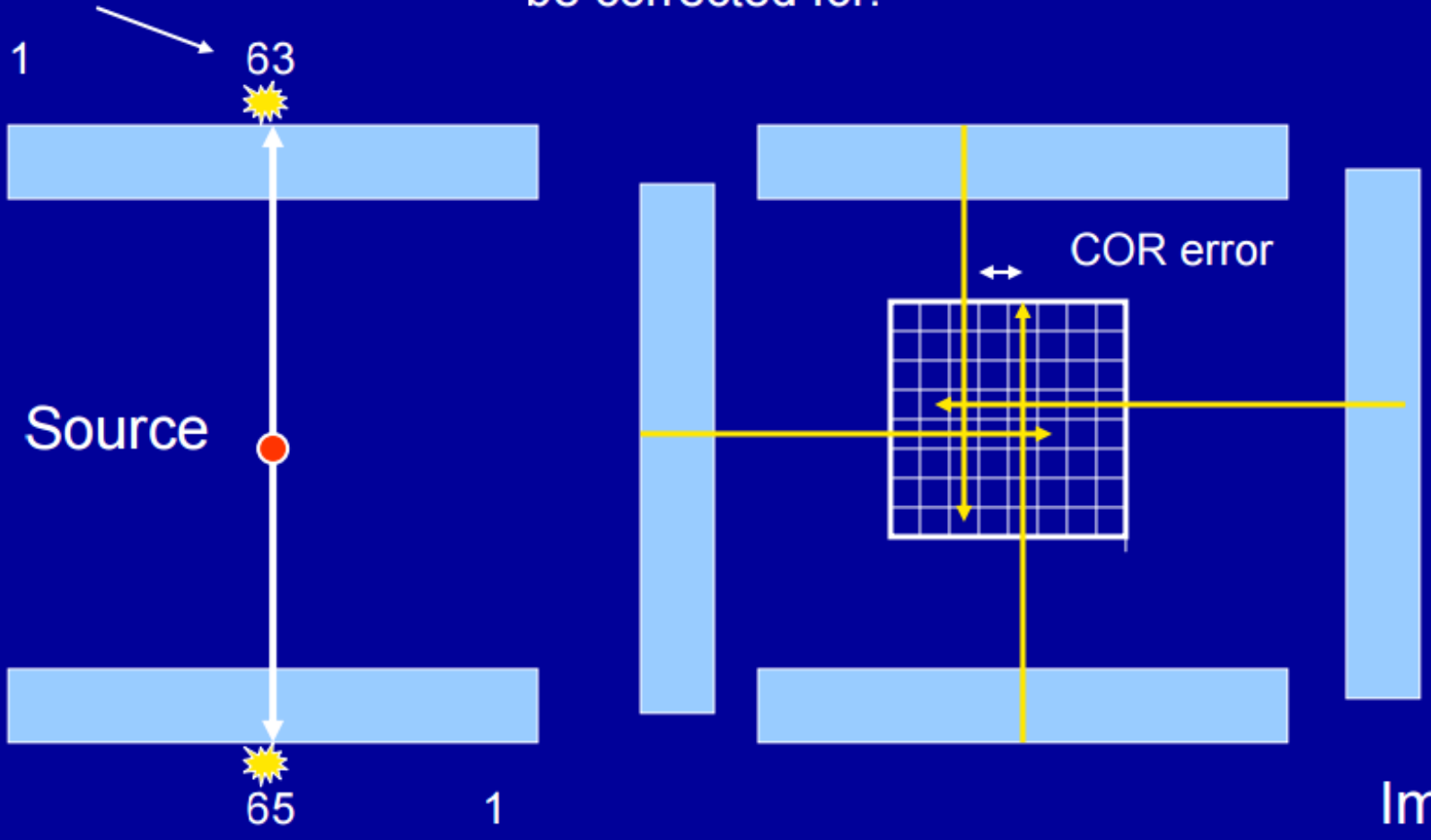


FIG. 12.24. An illustration of the effect of a misaligned center of rotation. A “donut”-shaped image appears from inaccurately aligned center of rotation. (From Todd-Pokropek A. The mathematics and physics of emission computerized tomography (ECT). In: Esser PD, Westerman BR, editors. *Emission Computed Tomography*. New York: Society of Nuclear Medicine; 1983; 3.)

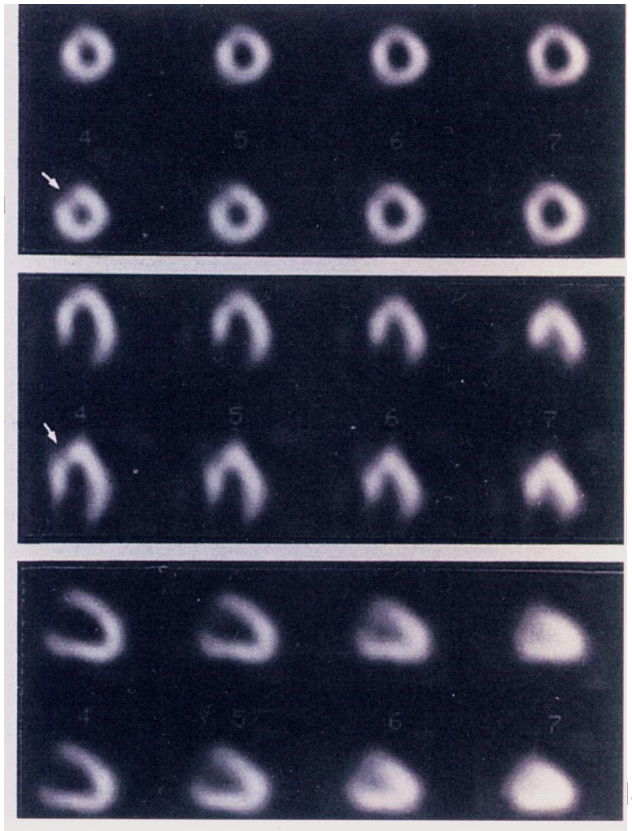
Pixel number recording signal 128x128 matrix

Offsets between physical center of rotation and center of image matrix must be corrected for.



COR offset

- **Smearing or hurricane sign** near the apex best seen on the Hor. Long axis.
- **Degradation of image resolution**



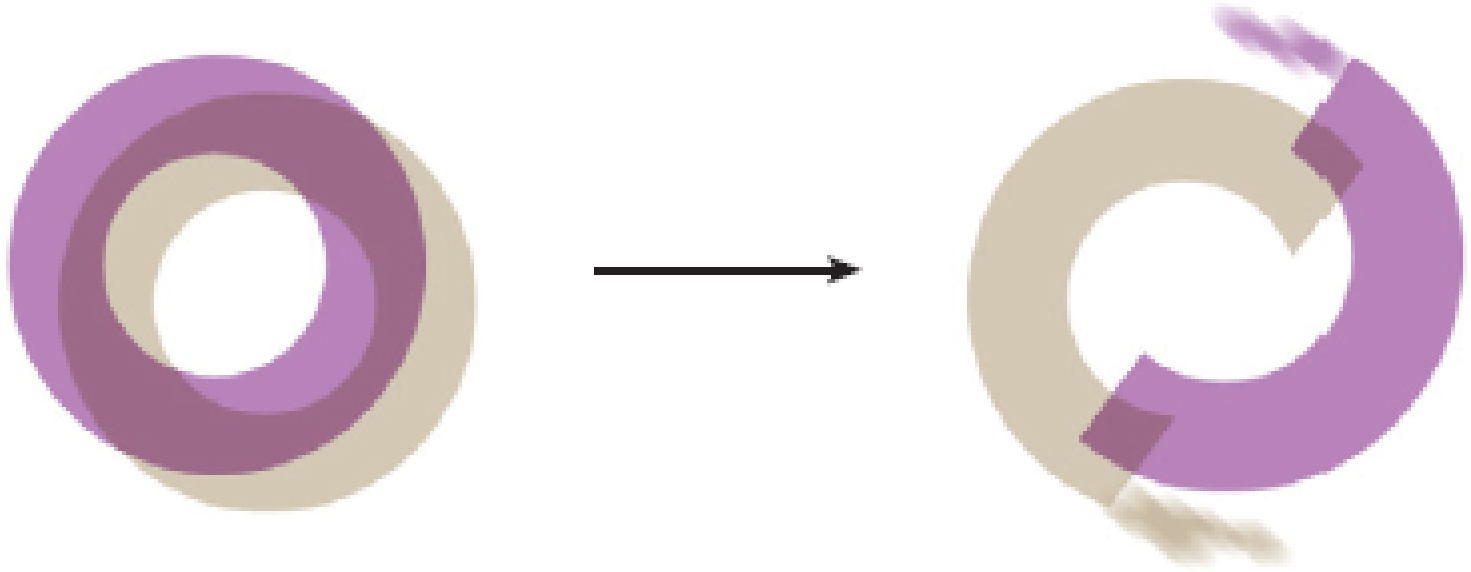
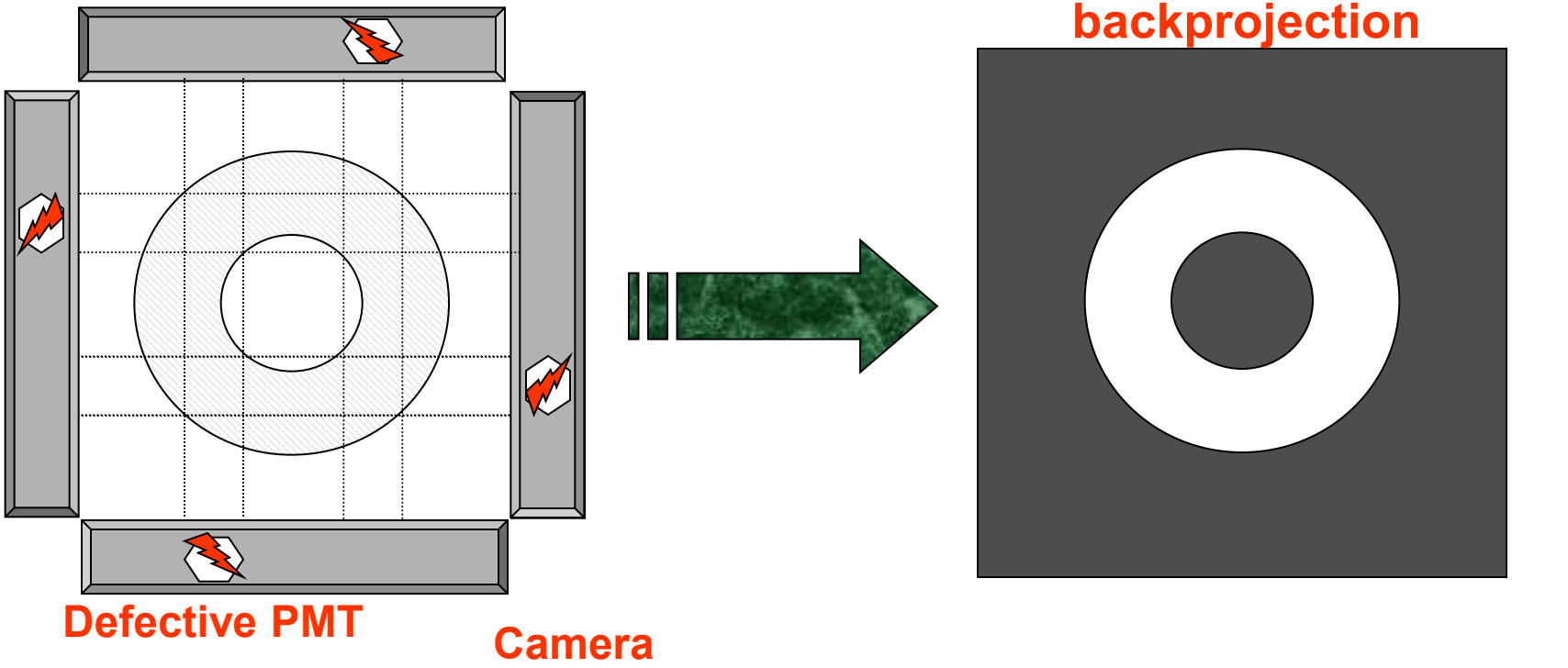


Figure 5-2 Schematic representation of camera center of rotation error and resultant artifactual myocardial perfusion defects. Defects are in opposite sides of the "heart," and "tails" of activity are present extending from the edges of the defects. *(Reproduced with permission from Iskandrian AE, Verani MS [eds]: Nuclear Cardiac Imaging: Principles and Applications, 3rd ed. New York: Oxford University Press, 2003.)*

Flood Field Nonuniformity



Flood Field nonuniformity

- **Ring artifacts**
  - Photondeficient rings may be apparent in

- **Quality control**
  - acquired the first thing in the morning each working day.
  - if ring artifacts appear in clinical SPECT images, it may be necessary to reacquire flood field images in the middle of the day.

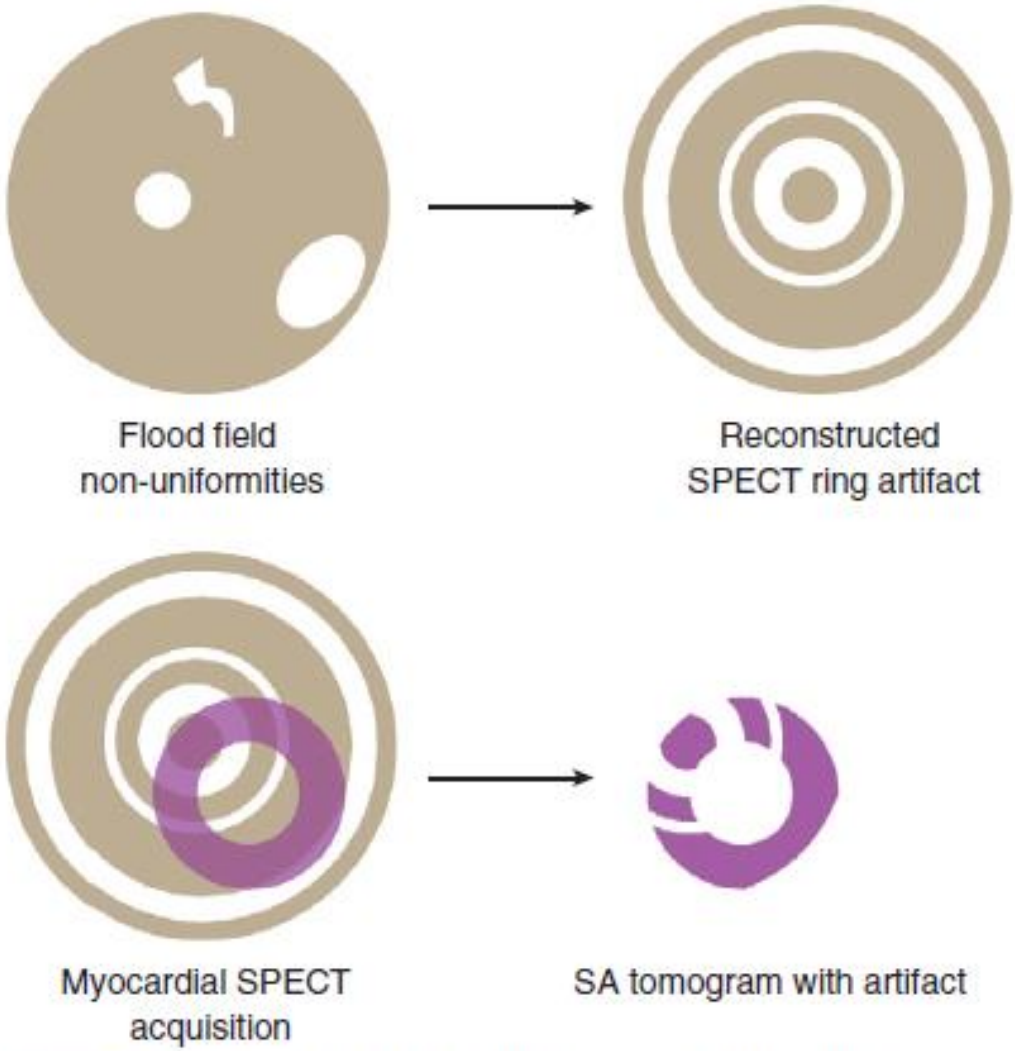
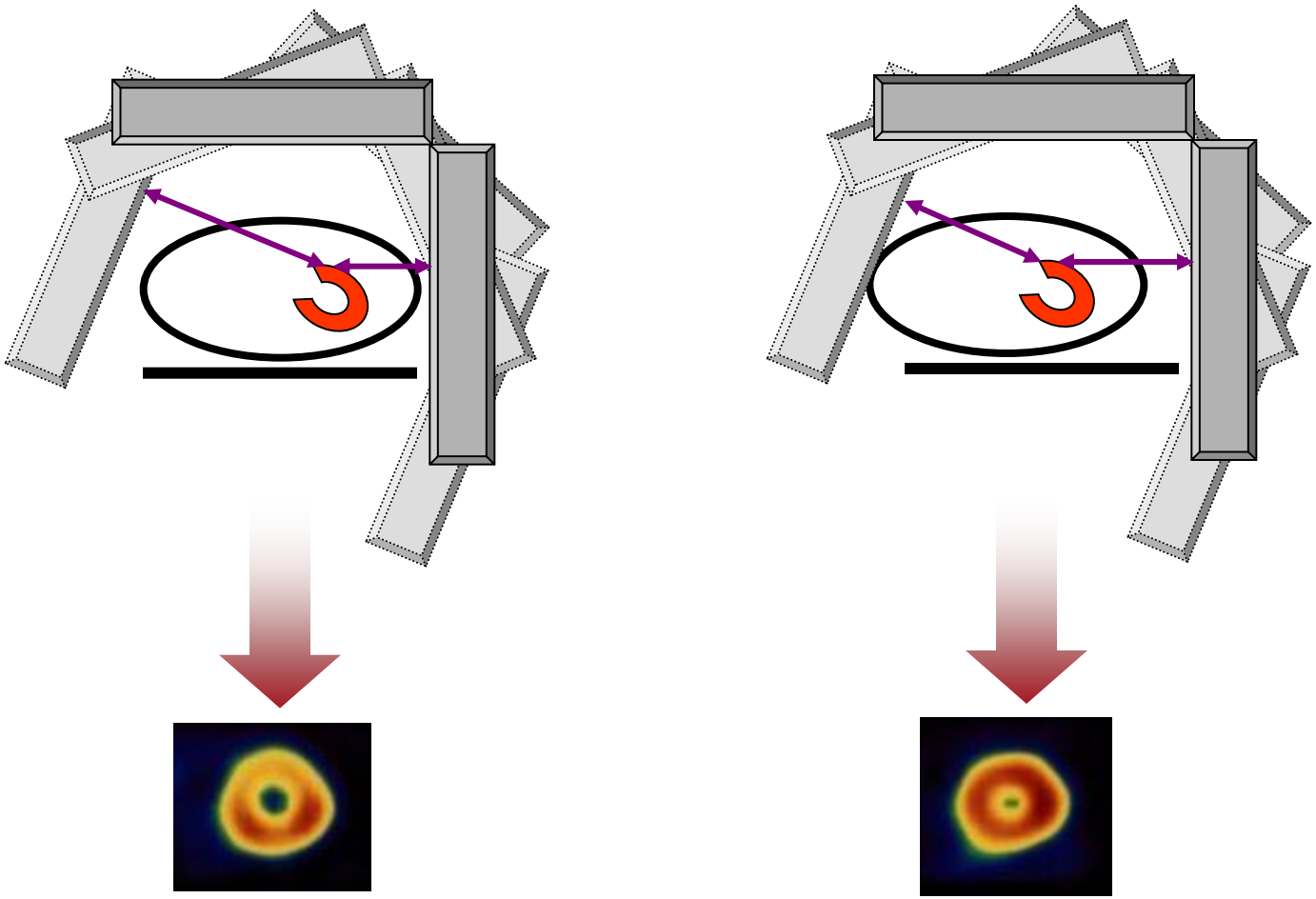


Figure 5-1 Flood field nonuniformity resulting in ring artifacts and artifactual myocardial perfusion defects. *(Reproduced with permission from Iskandrian AE, Verani MS [eds]: Nuclear Cardiac Imaging: Principles and Applications, 3rd ed. New York: Oxford University Press, 2003.)*

Orbit-related artifacts



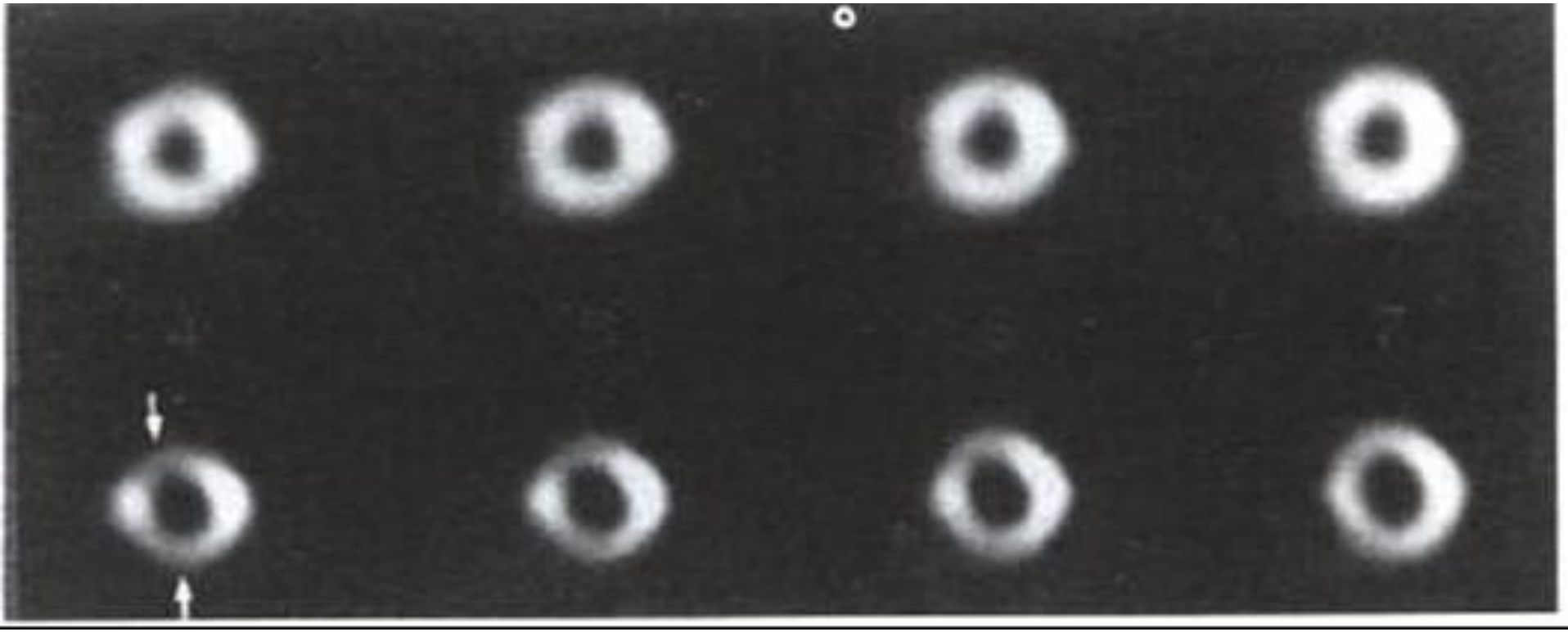
Orbit-Related artifacts

- **Heart is eccentric in the chest**
  - Circular orbit
    - Varying distances from the target organ
      - Varying in resolution
      - May : 180-degreees diametrically opposed defects on the short axis slices.

Orbit-Related artifacts

- **Avoiding:**
  - **360-degree acquisition**
  - **Moving patient laterally over the table**

- **Depending on the degree of eccentricity of the heart and body habitus:**
  - may be disturbing in the first apical short-axis slices



EXTRA-CARDIAC ACTIVITY

- **1.** FBP & Normalization (scaling) problems.

- **2.** Scatter of photons from the area with high activity area to the myocardium, leading to false myocardial counts.
- **3**. overlying or close proximity extra-cardiac activity the inferior or lateral walls are impossible to read due to such overlying or close proximity extra-cardiac activity

THE RAMP FILTER ARTIFACT

- This filtering process minimizes count density adjacent to an intense object,

THE RAMP FILTER ARTIFACT

- when the Ramp filter is applied to intense tracer concentration adjacent to the heart; there may be an artefactual decrease in count density in the inferior wall of the LV

- produce cold defects due to reconstruction artifacts

- may produce fixed, reversible, or reverse-distribution inferior artifacts.

THE RAMP FILTER ARTIFACT

iterative reconstruction :

proposed as a means to avoid the Ramp filter artifact.

SCATTERED ABDOMINAL VISCERAL ACTIVITY

- Thereby true (inferior) perfusion defects may be obscured.
- The activity scattering into the inferior wall may cause focal hot spots.
  - remote areas with relatively decreased activity may be misinterpreted as true perfusion defects
- If scatter into the inferior wall is more marked at rest than with stress, the relative increase in inferior count density from rest to stress can easily mimic inferior reversibility

Patients with hypertrophic, ischemic, or nonischemic cardiomyopathies

may develop reversible or fixed perfusion defects in the septum or elsewhere, even in the absence of CAD

- **These defects are likely due to**
  - **supply-demand imbalances,**
  - **metabolic abnormalities,**
  - **MBF regional differences,**
  - **and microscarring**

Myocardial Hypertrophy

- Diffuse myocardial hypertrophy often
  - results from
    - systemic hypertension or
    - increased volume overload
    - Increaed pressure overload a(with valvular heart disease).

- Such hypertrophy results in a generalized increase in myocardial uptake

Regional Myocardial Hypertrophy

- may be a localized increase in count density:
  - most often in the septum, papillary muscle, or apex

- appear as a localized increase in image count density
  - regions adjacent to and distant from the "**hot spot**" will appear to have relatively decreased count densities.

Regional Myocardial Hypertrophy

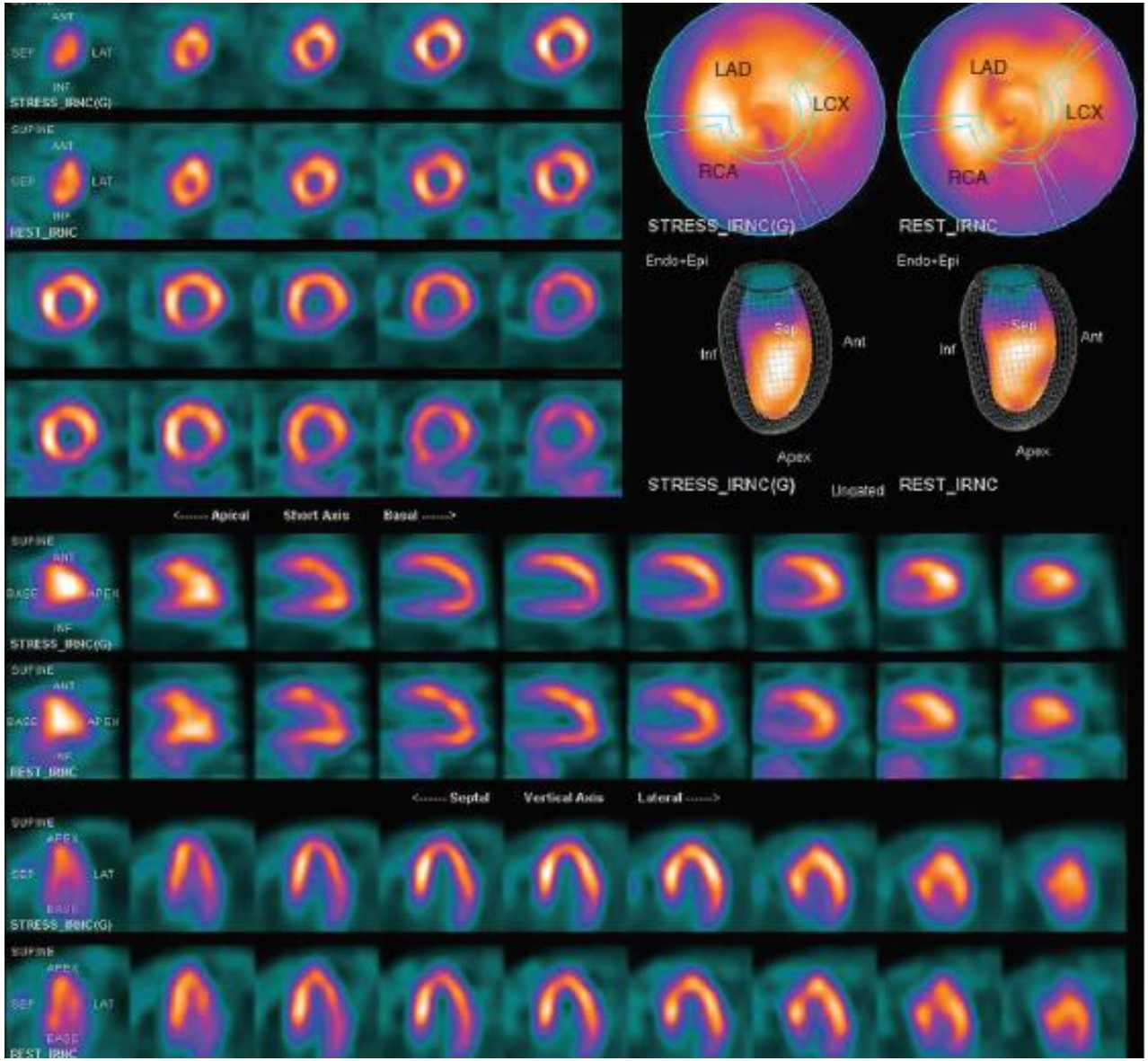
- **Increased septal count density** may be present in patients with idiopathic hypertrophic subaortic stenosis (IHSS),

- **HTN: common: a relative increase in septal-wall count**

- longstanding HTN, a significant decrease in the lateral-to–septal wall count-density ratio as compared to that in normotensive controls has been reported. (LCX territory)

- usually “fixed” defect

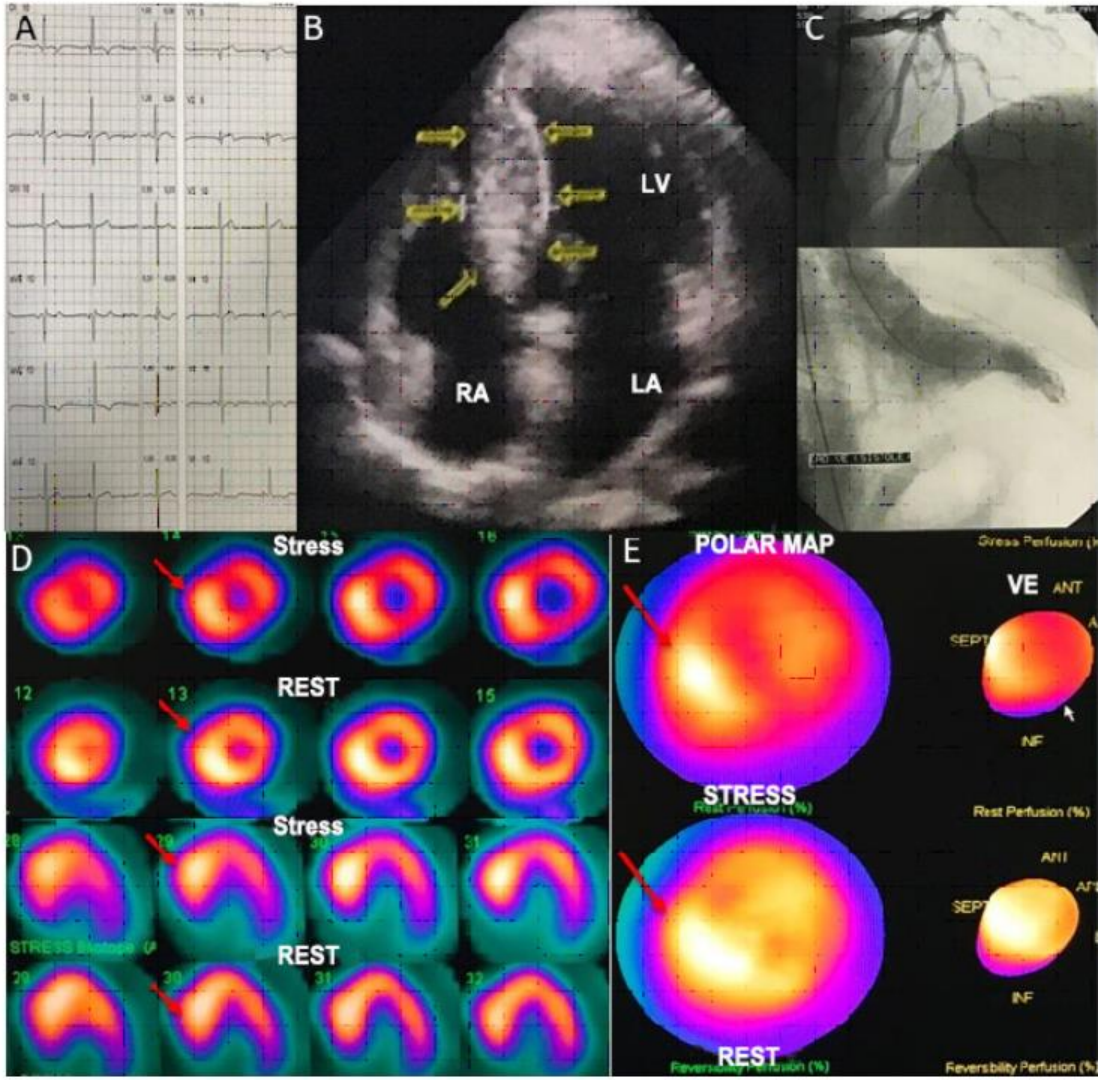
- occasionally the septum may appear **slightly less intense in resting images**, rendering the defect partly reversible and raising a clinical suspicion of ischemia.



longstanding HTN:

Septal hypertherophy

In this patient with longstanding systemic hypertension, there is a relative increase in count density in the septum in technetium-99m-sestamibi stress and rest tomograms.Because images are normalized to the region of greatest count density (the septum), the remainder of the ventricle,particularly the lateral wall, appears abnormal.

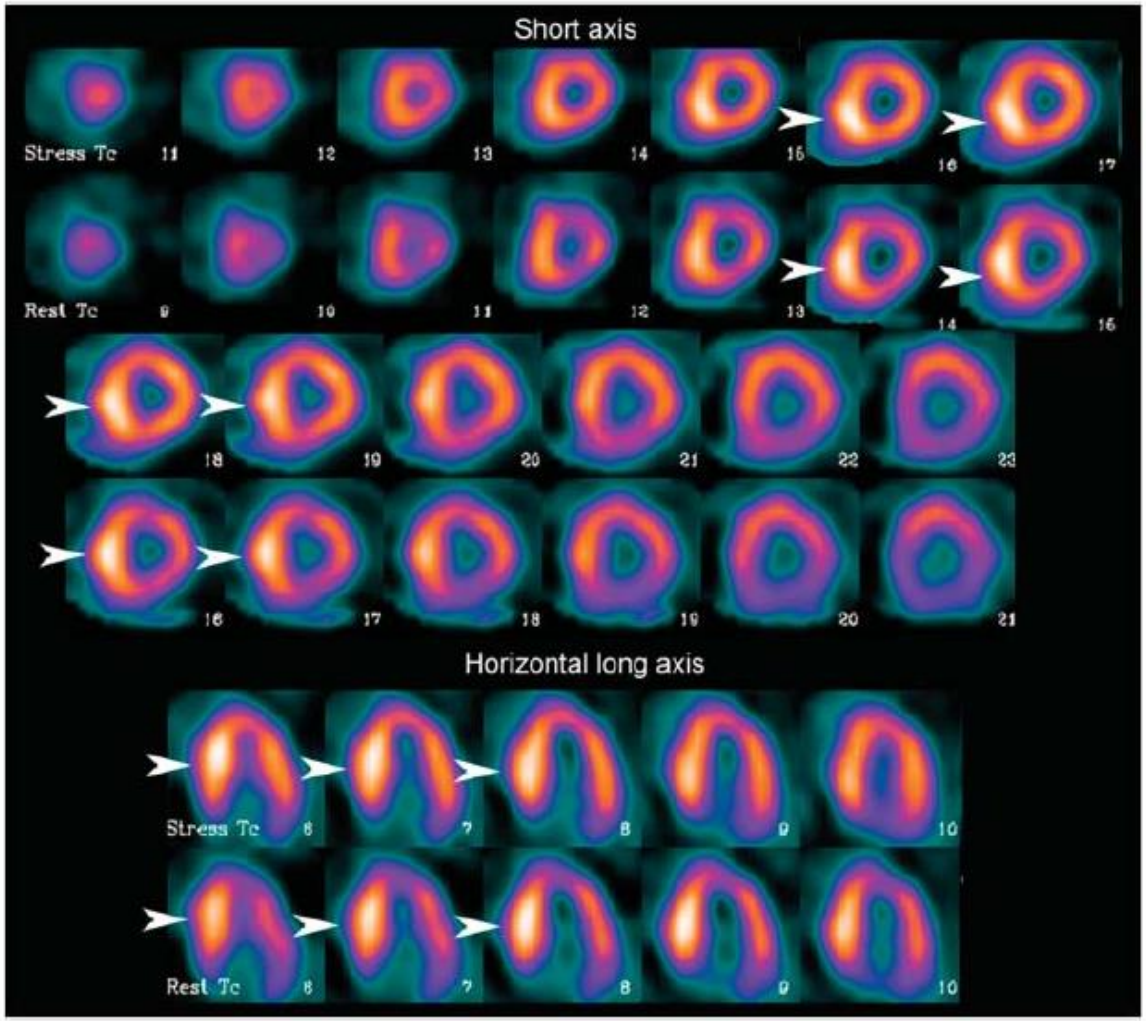


Patient with hypertrophic cardiomyopathy.
(A) ECG: signs of hypertrophy and ventricular repolarization disorders.

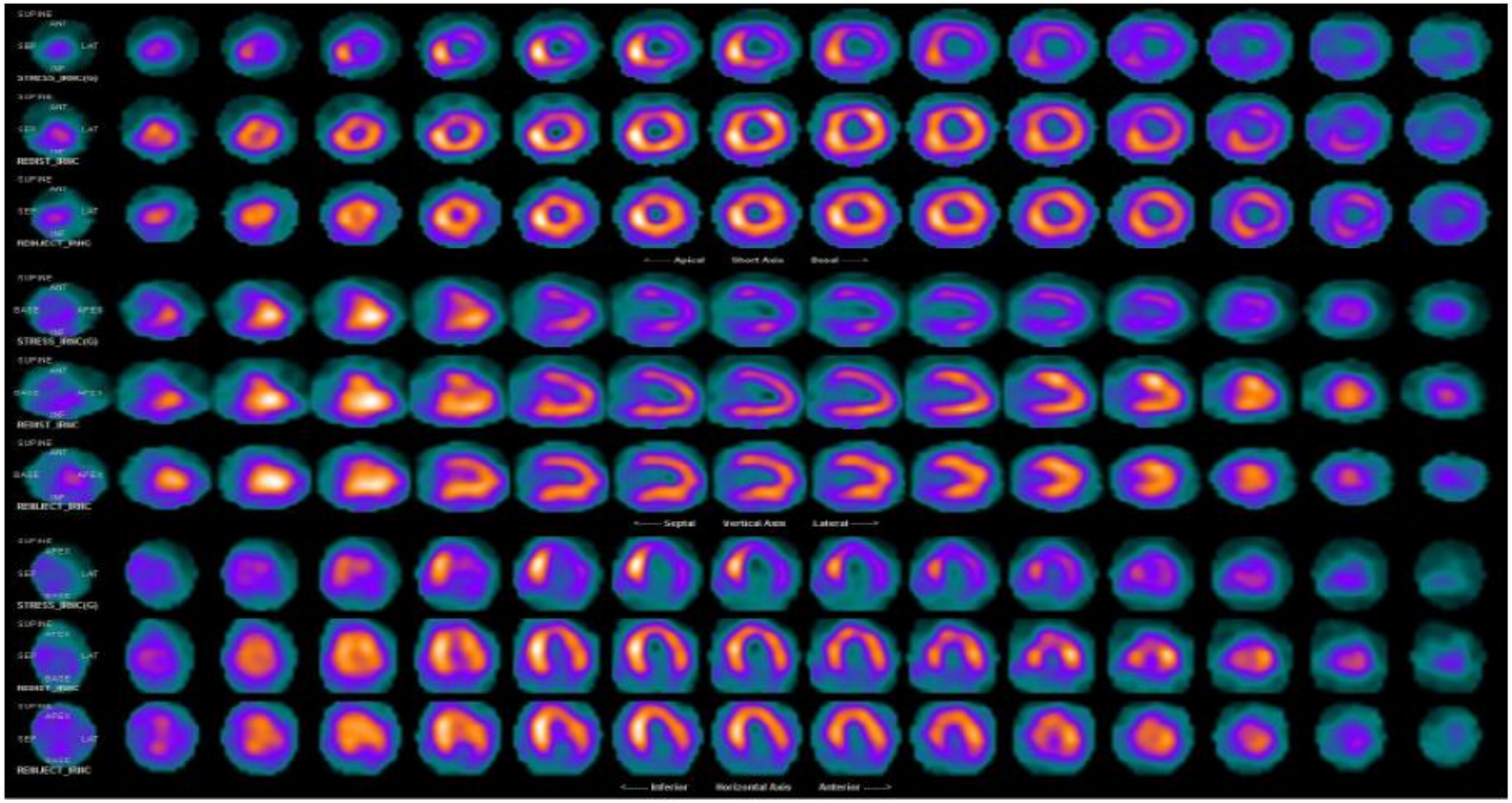
(A) Echocardiogram: apical 4-chamber view of severe septal hypertrophy (yellow arrows).
***(B) Cardiac catheterization: no obstruction** and ventriculography with the sign of "ballerina foot," which suggests ventricular hypertrophy.*

(A) MPI: hyperconcentration of the radiopharmaceutical drug in the septal wall (arrows) and relative reduction in the other walls, especially after stress.

(D) Polar map, after stress and at rest, shows greater "brightness" (arrows) in the radiopharmaceutical uptake in the inferoseptal wall (local hypertrophy denoting greater myocardial perfusion). Lower concentration (less perfusion) of the radiopharmaceutical drug in the other walls, especially in the polar map of stress.



perfusion images from a 35-y-old man with hypertrophic cardiomyopathy. There is marked increased uptake throughout the septum, which, because of the normalization to the hottest pixel, results in apparent widespread decreased perfusion to the remaining walls of the LV.



MPI: *with tallium-201. Patient* ***with hypertrophic cardiomyopathy****, 42 years old, female, presenting moderate drop in left ventricular ejection fraction in a recent scan. Previous echocardiography (previous year) revealed normal left ventricular ejection fraction. On the images after stress (top rows), increased perfusion in the septal wall and perfusion defect in the other walls, with virtually complete perfusion improvement in redistribution images (middle rows) and reinjection (bottom rows). Coronary angiography did not show any obstructive epicardial coronary lesions.*

Regional Myocardial Hypertrophy

- **Papillary muscle hypertrophy** may occur in patients with LV hypertrophy
- Papillary muscle hypertrophy occurs commonly in patients with concentric myocardial hypertrophy, producing a localized "hot spot" in the lateral wall of the left ventricle.

creating localized hot spots in the

anterolateral and

inferolateral walls.

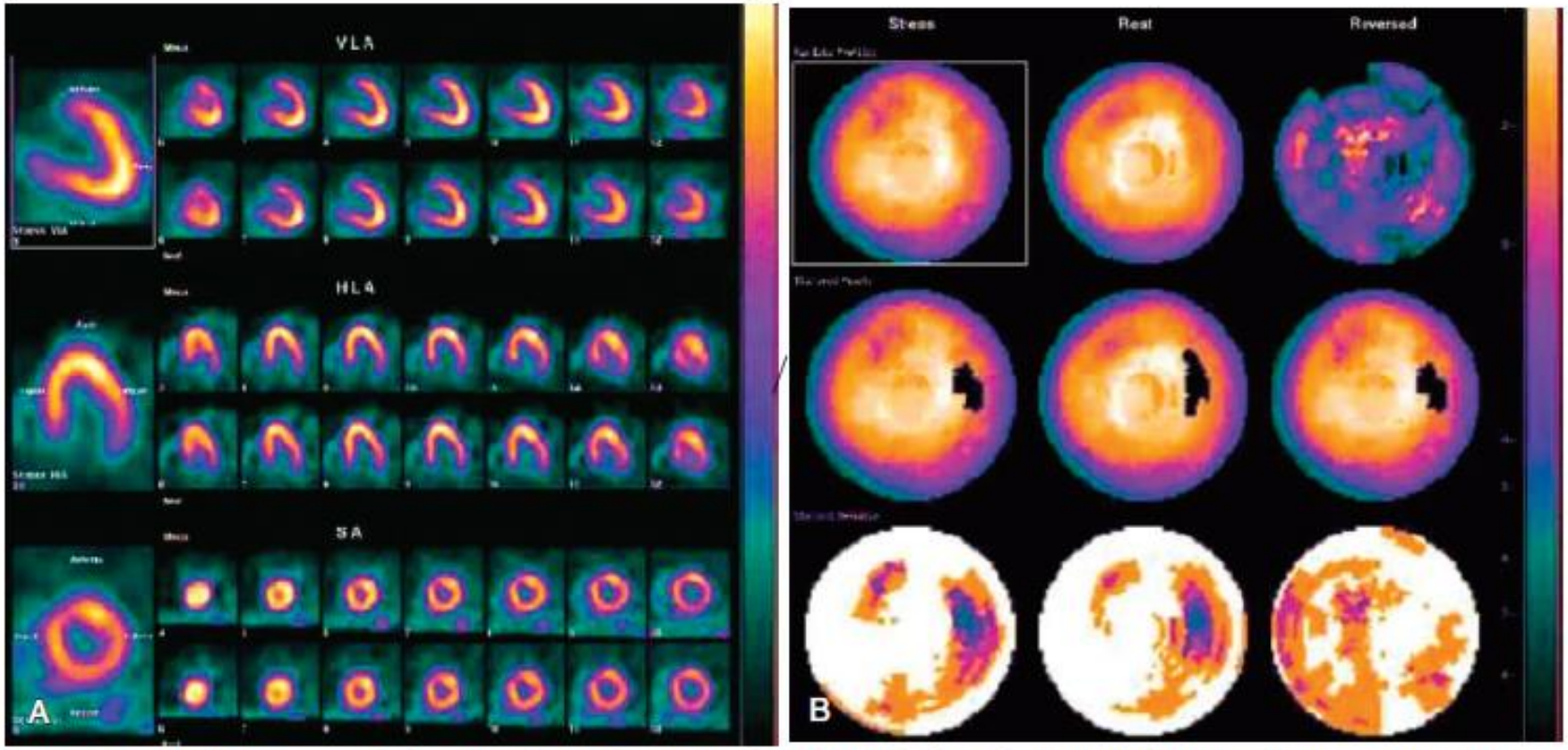
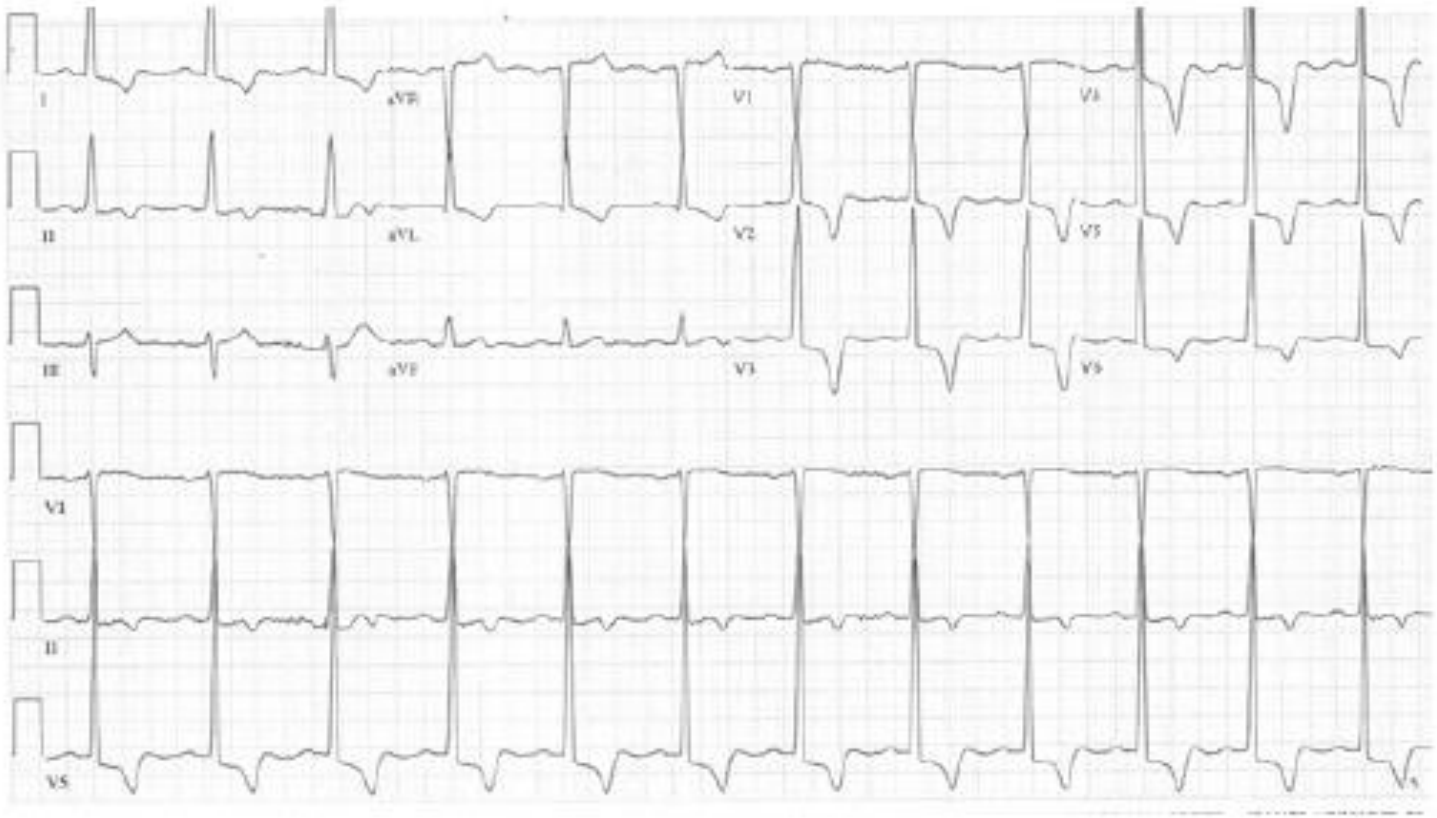


Figure 5-20 In this patient with hypertrophic cardiomyopathy with marked involvement of the apex, there is an increase in apical count density noted in tomographic slices (**A**). Polar plots, which are normalized to the apex in this particular case, demonstrate a relative decrease in count density in the more peripheral aspects of the left ventricle (**B**). *(Reproduced with permission from Iskandrian AE, Verani MS [eds]: Nuclear Cardiac Imaging: Principles and Applications, 3rd ed. New York: Oxford University Press, 2003, p 103.)*

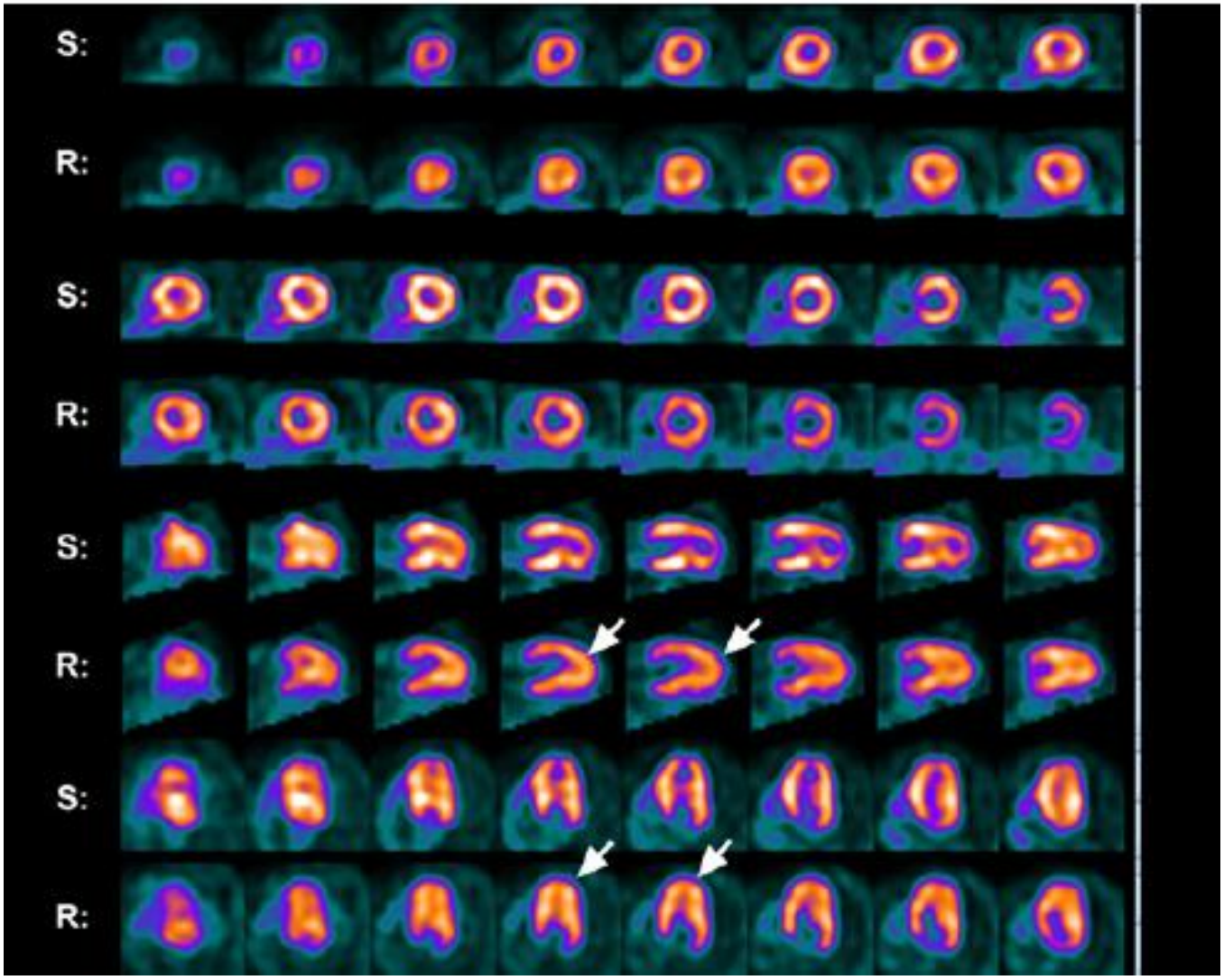
localized **apical hypertrophy**, in which case there may be an **apical hot spot** with a consequent relative decrease in count density in the **more basal aspects** of the LV



Giant T-wave inversions in the precordial and high lateral leads, without septal Q waves

Exercise stress testing revealed no ischemia or arrhythmia.

- 52-year-old man
  - Syncope
  - no chest pain
  - no palpitations, dizziness, presyncopal aura, or post-ictal confusion
  - Non-smoker



LV hypertrophy (confined to the apical portion of the ventricle and more apparent at rest), and reversibility in the apex of the ventricle

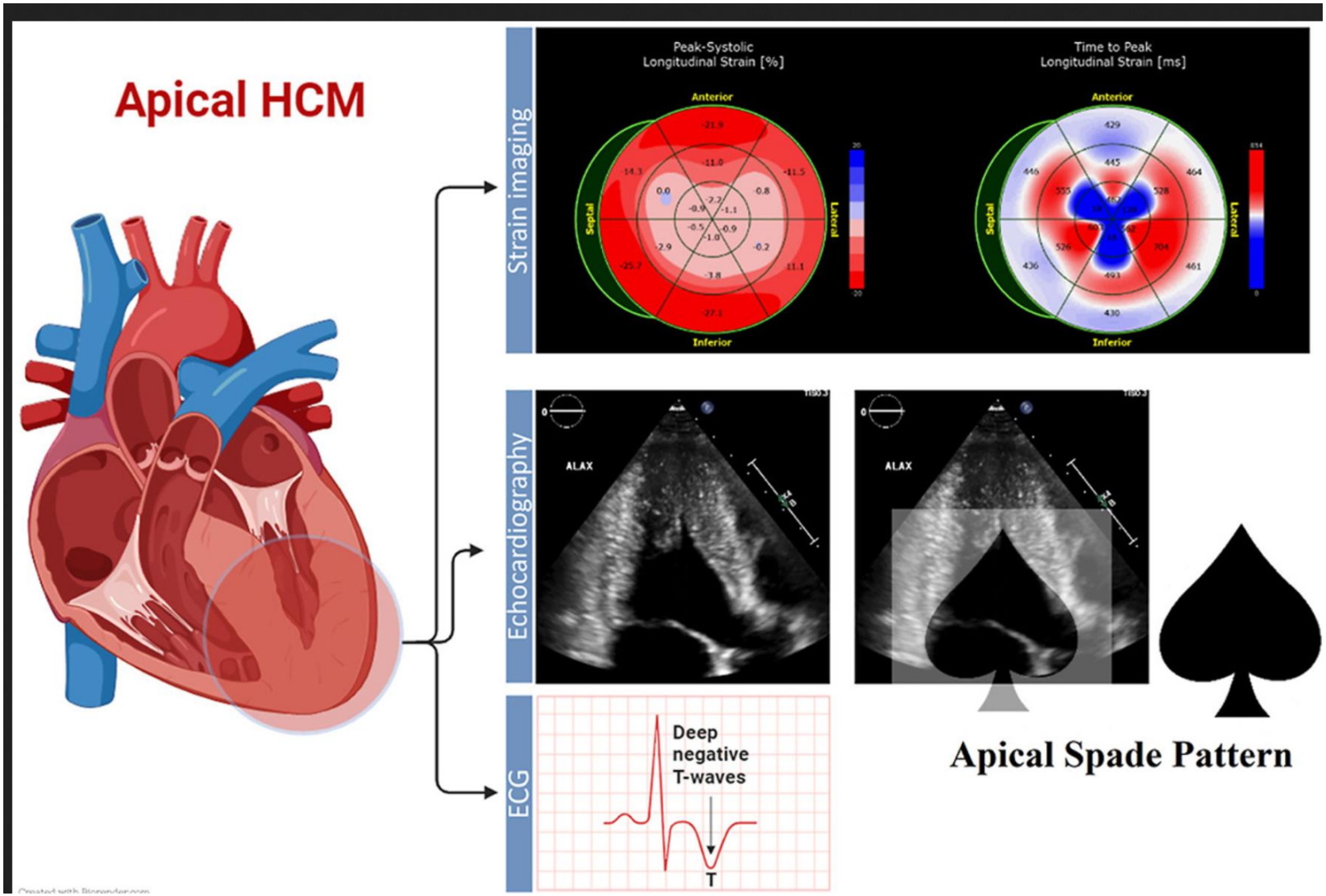
apical HCM can mimic symptomatic obstructive coronary artery disease, many patients undergo
cardiac stress testing for possible ischemia.

Significant myocardial ischemia, probably secondary to closure of the epicardial coronary vasculature during systole.
-subendocardial ischemia and thereby lead to the rather paradoxical enlargement of the apical cavity during stress SPECT imaging. Stress echocardiography can be useful in correlating images in such cases

-Another possible explanation of the apical perfusion defect in our patient could be the demand–supply mismatch during stress

Two-dimensional transthoracic echocardiography with microbubble contrast revealed marked hypertrophy of the apical third of the LV and complete annihilation of the apical portion of the LV cavity during systole

Echocardiography usually reveals an “ace of spades” appearance of the ventricle,



fixed or ischemic perfusion defects in HCM can be related to:

- epicardial coronary stenosis

in patients with HCM in the absence of coronary artery disease and is associated with abnormal MP

  - Small vessel disease, microvascular dysfunction & arteriolar architecture distortion
  - Reduced coronary flow reserve
  - Intramuscular course of the coronary vasculature and/or
  - Mismatch between myocardial oxygen supply and demand from the increased myocardial mass and ventricular overload conditions.

- Blunted myocardial blood flow during stress is the functional consequence of microvascular dysfunction and can be detected as regions of hypoperfusion with various imaging techniques

    - **PET: subendocardial ischemia**

- Myocardial ischemia caused by microvascular dysfunction is an important pathophysiologic component of hypertrophic cardiomyopathy (HCM),
  - promoting myocardial fibrosis,
  - adverse left ventricular remodeling, and
  - impacting on clinical course and outcome in HCM patients.
- replacement fibrosis by repetitive bouts of microvascular ischemia contributing to myocyte cell death

- presence of regional perfusion defects could have prognostic implications

- Study: 91 children with HCM: underwent SPECT evaluation:

– Myocardial perfusion defects: 70 children (76.9%)
  - Fixed perfusion defects: 22
  - reversible defect:48

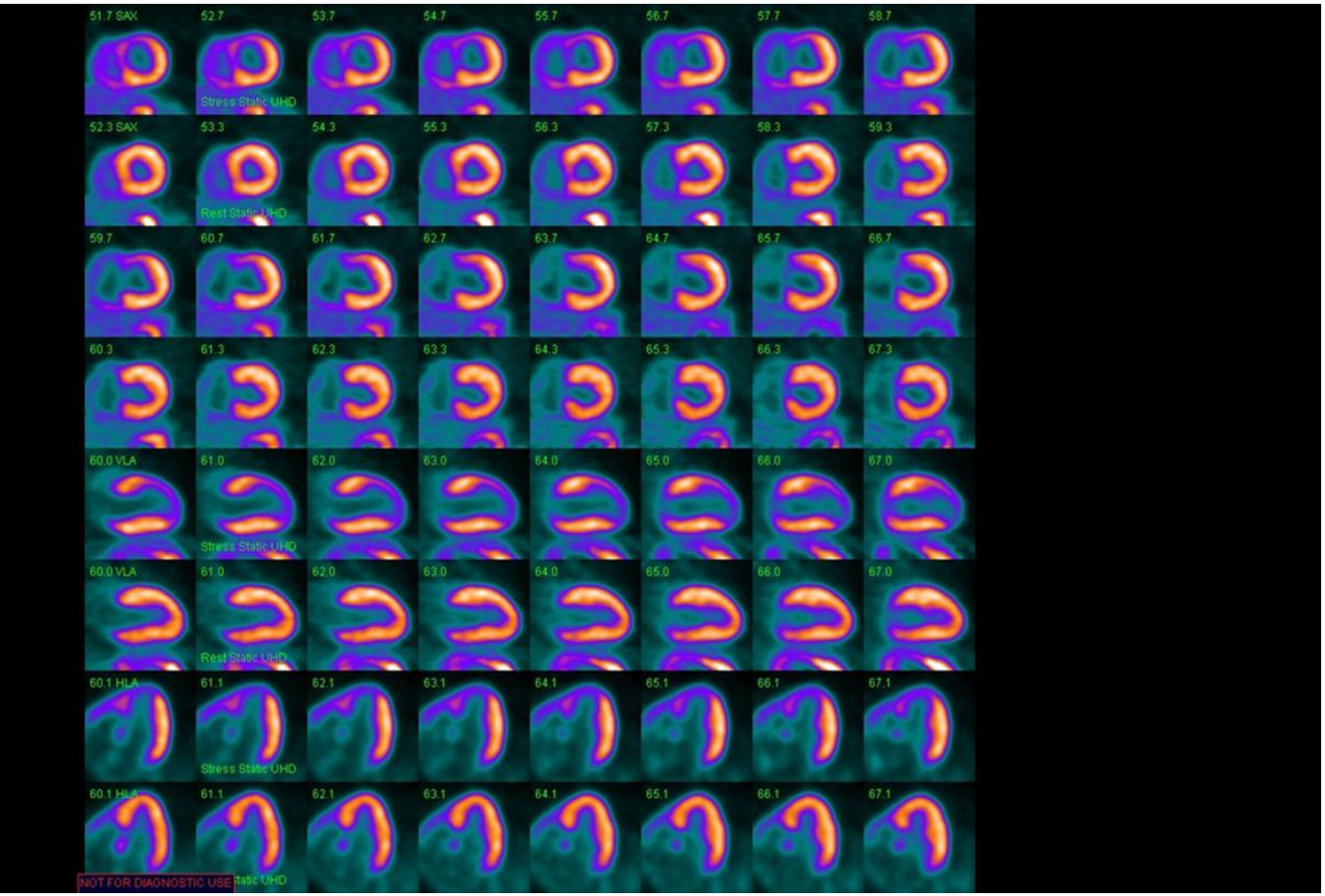
– During follow-up at a median of 8.3 years in children with myocardial ischemia, clinical endpoints occurred more often (47 vs. 5; $p = 0.02$) and more patients reached a clinical endpoint (28 [40%] vs. 3 [14.3%]; $p = 0.036$).

– In children with myocardial ischemia, myocardial fibrosis was observed with greater frequency.

– Myocardial perfusion defects: an important predictor of adverse clinical events and risk of death in children with HCM.

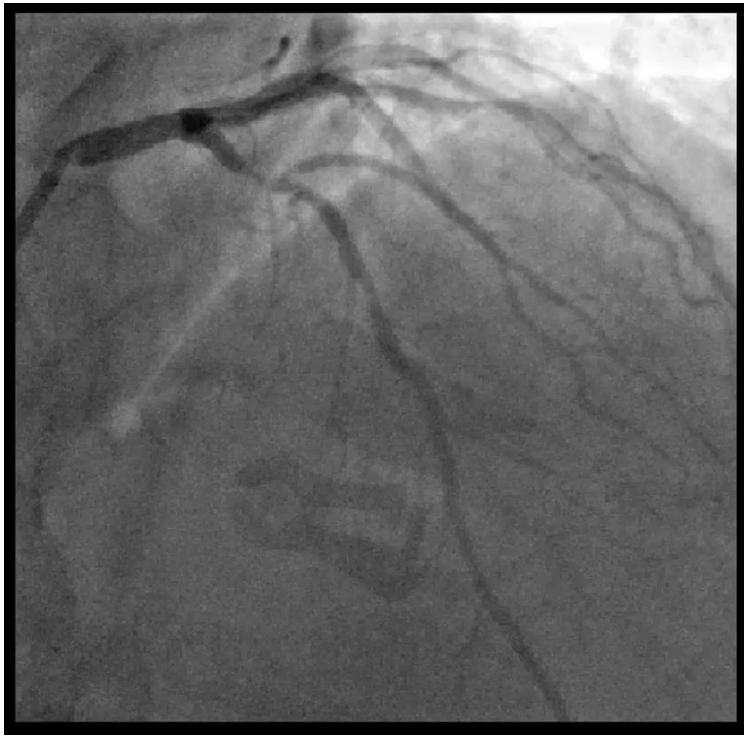
– Myocardial ischemia in HCM patients frequently correlates with myocardial fibrosis.

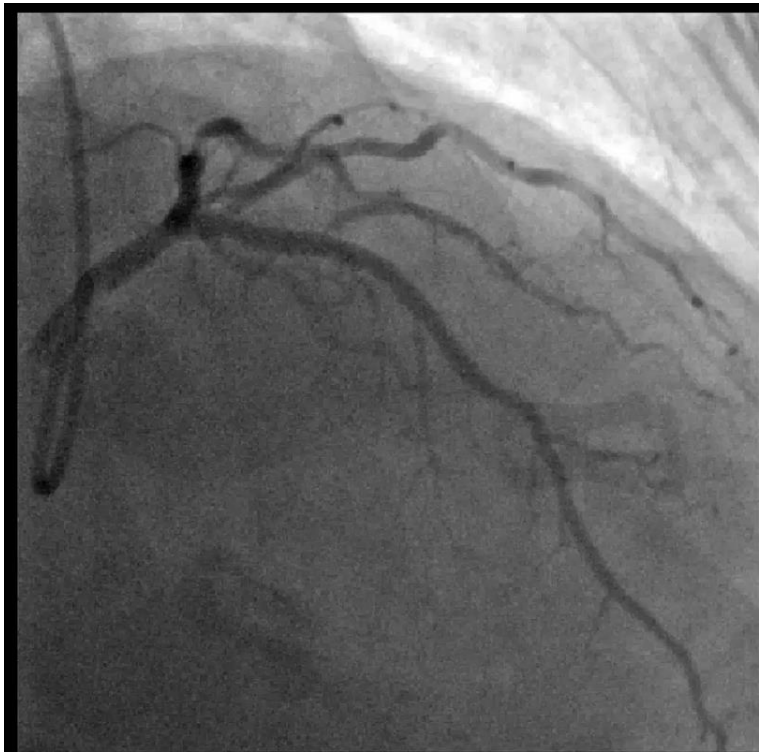
– It is thought that MP disorders and subsequent fibrosis may act as arrhythmogenic substrates.



- **A 58-year-old man with a history of hypertrophic cardiomyopathy (HCM) presented with tightness in the chest and occasional chest pain.**
- **The patient had previously been treated with alcohol septal ablation for a left ventricular outflow tract (LVOT) obstruction secondary to HCM.**
- **The patient also had several risk factors for coronary artery disease including high BMI , hypertension, and diabetes. In view of the clinical possibility of coronary artery disease coexisting with hypertrophic cardiomyopathy, the patient was referred to a MPI study**

large reversible perfusion defect in the anterior wall and apex as well as an adjacent septum reflecting inducible ischemia in the left anterior descending (LAD) territory. The fixed perfusion defect in the upper and basal septum reflects alcohol injection related to the ablation performed previously for relief of left ventricular (LV) outflow obstruction secondary to hypertrophic cardiomyopathy. Significant TID reflects the severity of the LAD territory ischemia with complete reversibility of the ischemic zone demonstrated by normal LAD territory uptake and normal LV cavity size at rest

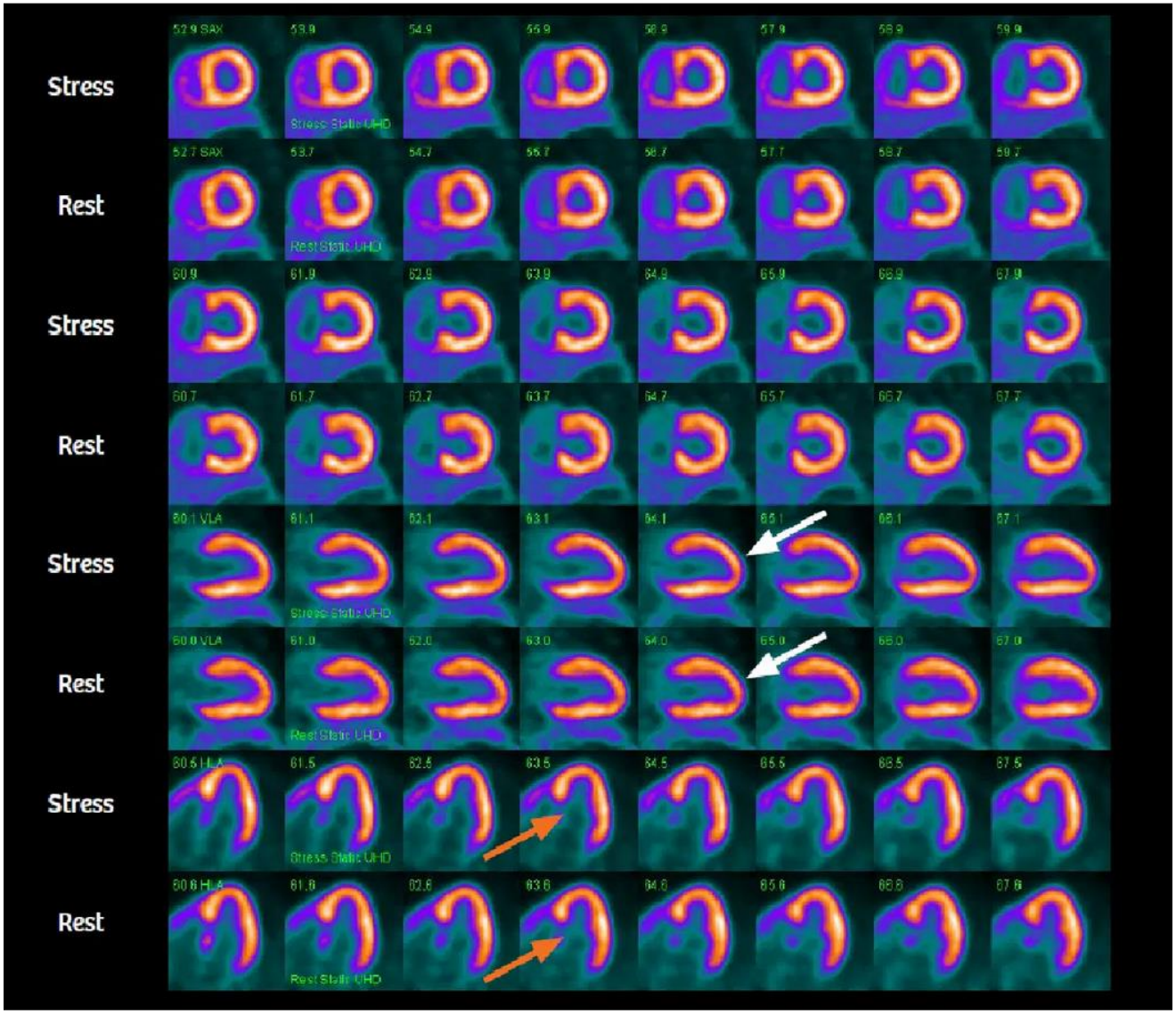




In view of the significant LAD stenosis demonstrated on the coronary angiography , the patient underwent stenting of the LAD lesion. Coronary angiography identified additional plaques in the mid- and distal LAD and left circumflex, which were not considered flow limiting and therefore intervention for the plaques was not performed. Angiographic images acquired immediately following stent placement showed a normal LAD flow restored . A follow-up MPI study was performed one year post-percutaneous coronary intervention (PCI) as the patient was admitted with troponin negative chest pain. The study was performed to assess the significance of plaque disease elsewhere, to ensure stent patency, and to rule out microvascular ischemia.

Follow-up MPI study shows normal perfusion in the LAD territory, which reflects patent LAD stent. a gross reduction in TID was seen. The basal septal fixed perfusion defect related to alcohol ablation of the upper septum for the LVOT obstruction relief appears similar to that seen in the initial MPI study.

Overall: a successful stenting of proximal LAD stenosis in this patient with coronary artery disease co-existing with hypertrophic cardiomyopathy.



FDG PET Studies

- Situations such as increased energy demand due to myocardial hypertrophy, inflammatory response due to infiltration of inflammatory cells involved in the pathogenesis and myocardial ischemia by microangiopathy,

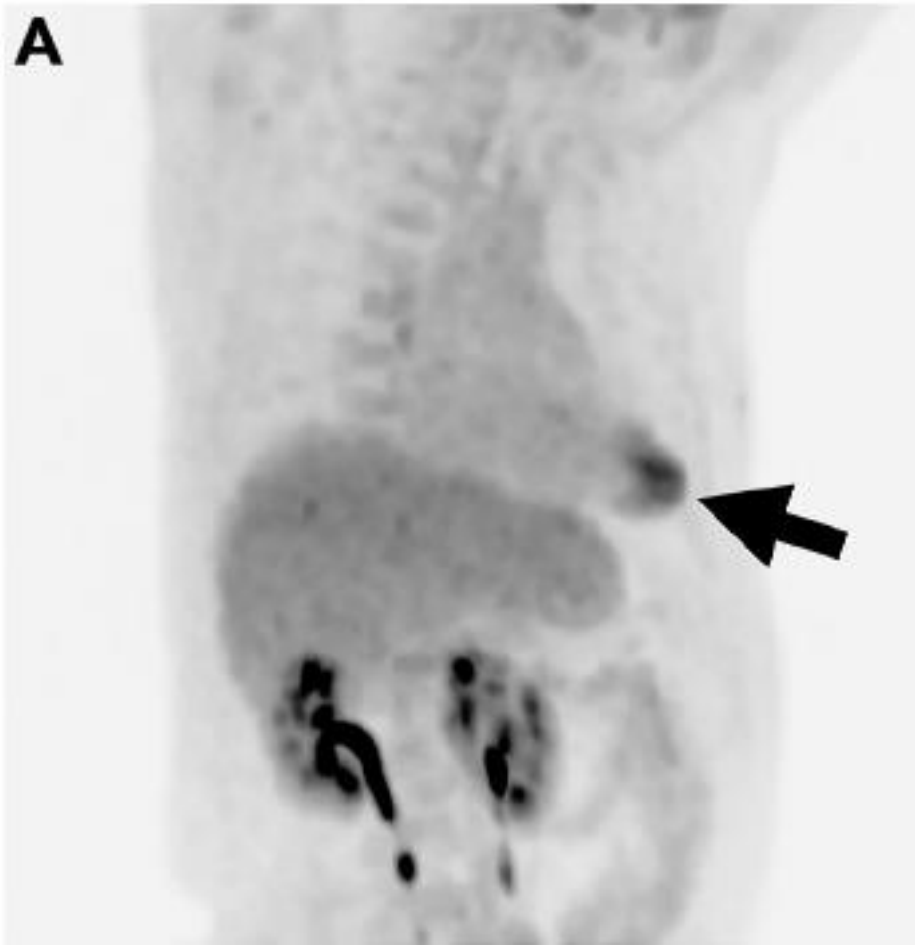
may lead to the up-regulation of hypoxia-inducible factors that trigger a change in metabolism in favor of glycolysis, by inducing the transcription of glucose membrane transporters.

FDG PET Studies

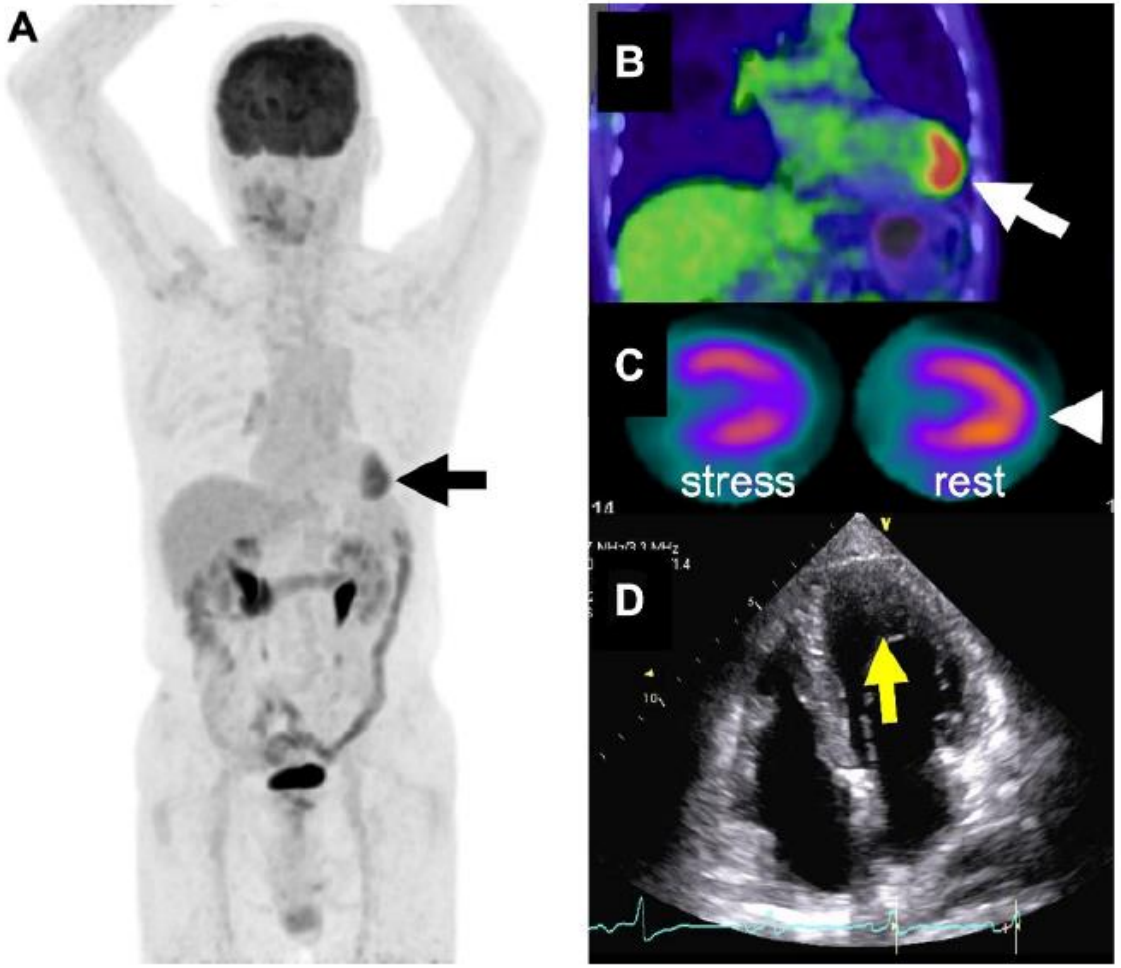
- abnormal 18F-FDG concentration at the cardiac apex in a series of 18F-FDG PET cases, found that the ace of spades pattern observed on the echocardiogram of patients with apical HCM suggests that the intensity of glycolytic activity in this region could be associated with the progression of myocardial hypertrophy, detected by electrocardiographic abnormalities, apical akinesia and abnormal coronary flow reserve (in SPECT images), also allowing to conclude that

concentration of 18F-FDG is more extensively observed in the myocardium with dysfunction

Incidental spade-shaped FDG uptake in the left ventricular apex suggests apical hypertrophic cardiomyopathy



Isolated spade-shaped uptake around the apex (SSUA). PET/CT images (**a, b**) show spade-shaped uptake around the apex (*black* and *white arrows*).
Spade-shaped tracer uptake around the apex at rest SPECT (*arrowhead*) with reduced coronary fow reserve at stress is shown (**c**). Two-dimensional apical four-chamber echocardiography
shows apical hypertrophy (*yellow arrow*, **d**)



Arrhythmias and Gating Errors

– **If the degree of the regular beat rejection varies during the SPECT acquisition,**

- Therefore, projection images will vary in count density. When viewed in endless loop cinematic format, the projection images will appear to “flash.”

- The most serious effect of arrhythmia is a decrease in image count density due to these “discarded” cardiac cycles.

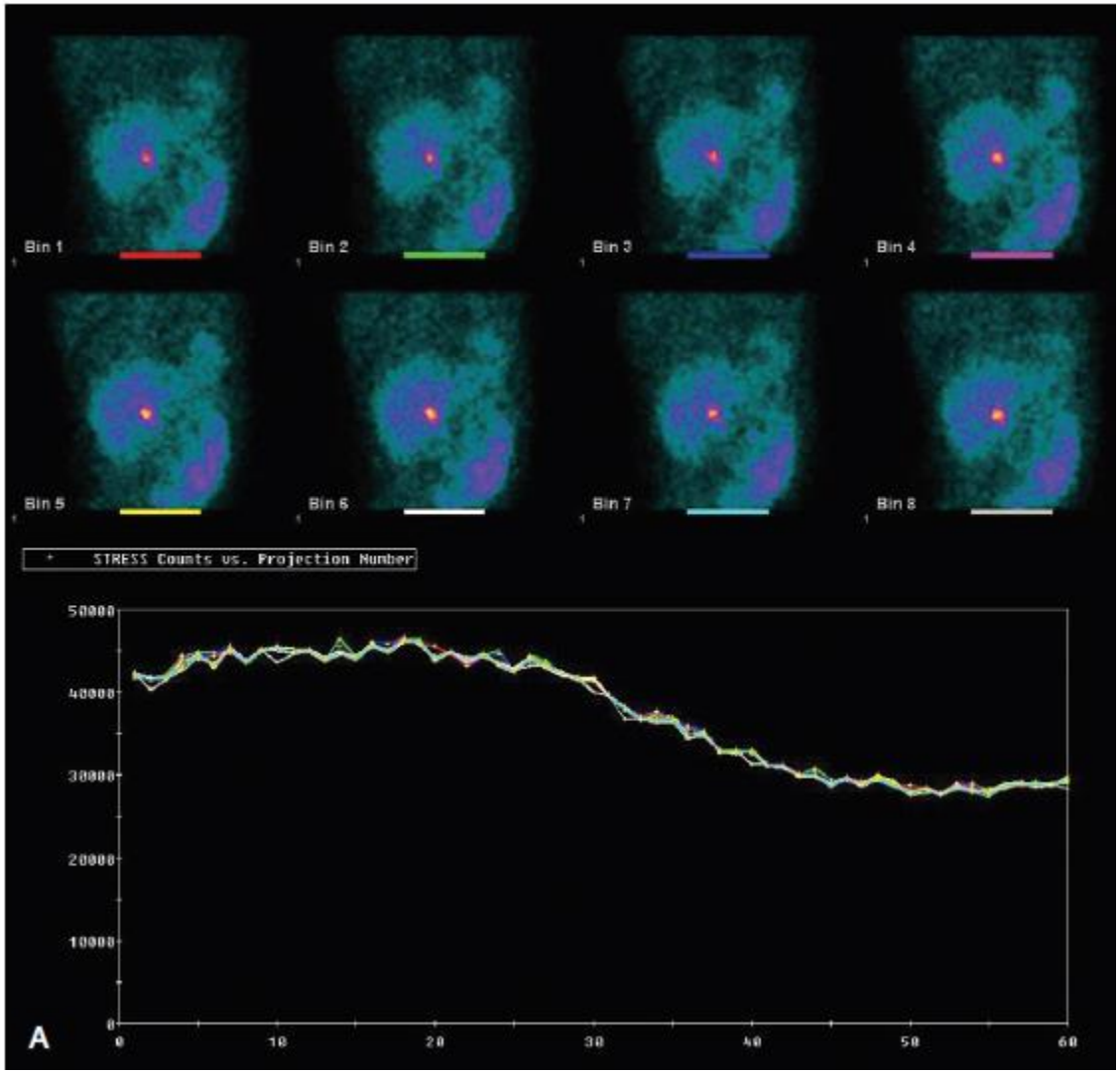
Arrhythmias and Gating Errors

- However, only with severe arrhythmias associated with AF and frequent PVC are clinically significant perfusion artifacts encountered.

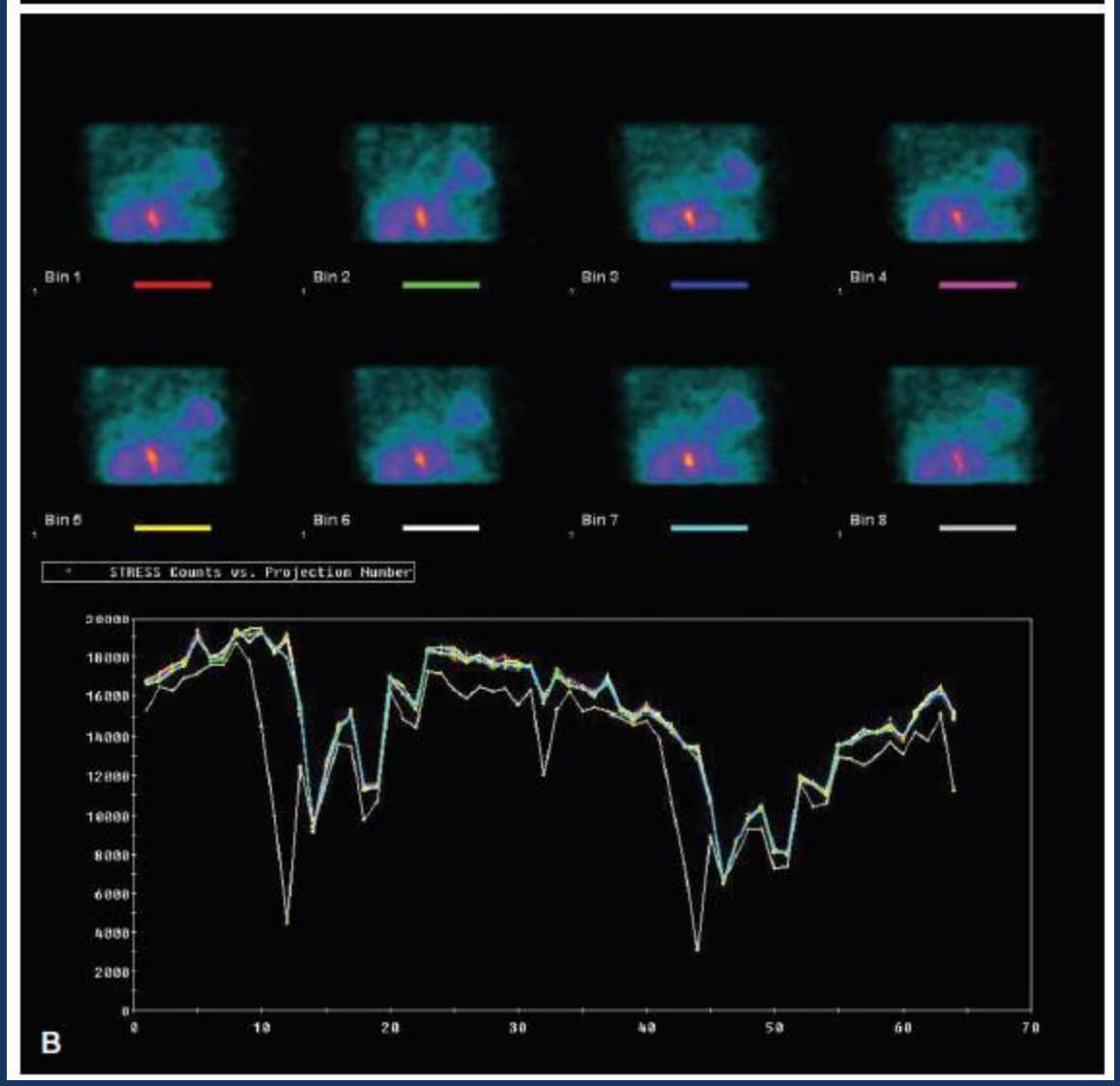
Arrhythmias and Gating Errors

- Recently, some manufacturers have established a “9th bin” wherein rejected cardiac cycles are stored temporarily and excluded from reconstruction of the gated tomograms, but added back into the summed, nongated images

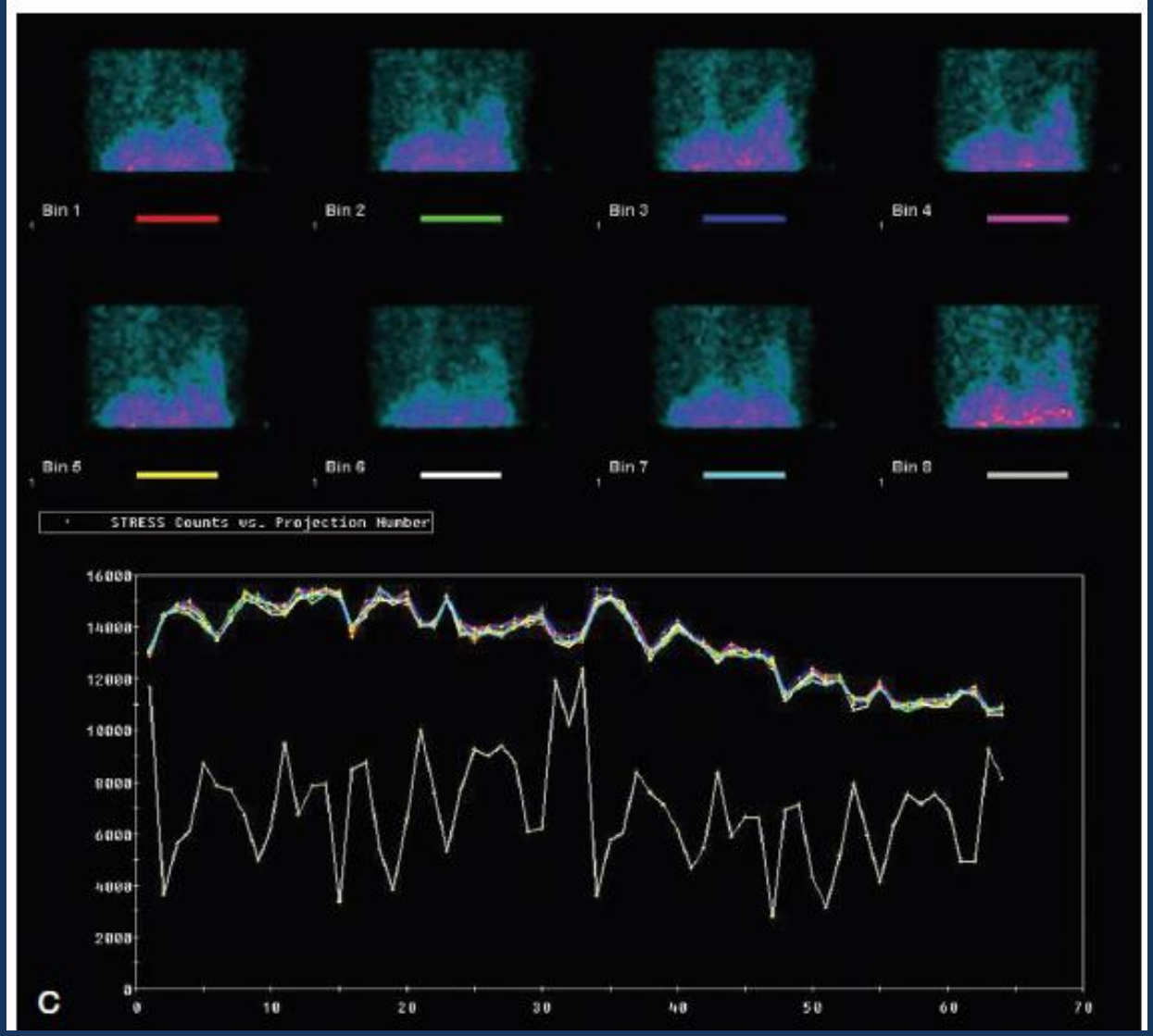
- Alternately, other manufacturers allow simultaneous acquisition of gated and nongated images so that arrhythmic beat rejection will not result in low count-density ungated images.



A, Count density is plotted on the y axis for each of the eight “bins” of an 8-frame per cardiac cycle gated poststress myocardial perfusion scan for the 64 camera stops in the 180-degree SPECT imaging arc of a 90-degree-angled, two-detector scintillation camera. In this patient with no arrhythmia and a very regular R-R interval, the curves are superimposed for each of the eight gated “bins.”



In this arrhythmic patient, cardiac cycles are rejected during camera stops 8 to 24 and simultaneously during stops 40 to 56 for the other scintillation detector. This is manifested as a decrease in counts acquired/accepted at those stops.



In this patient, all cardiac cycles were accepted during the SPECT acquisition. However, owing to variation of the R-R interval of the accepted beats, there is a decrease in count density in the 8th gated “bin.”

Case Presentation

A 79 Y/O female patient :

- Epigastric pain
- Agitation and unrest
- Admitted
- TP +
- No risk factor
- Echo: LVEF: 55%, NL LV systolic F, Mild MR & TR

- Admitted for palpitation and arrythmia

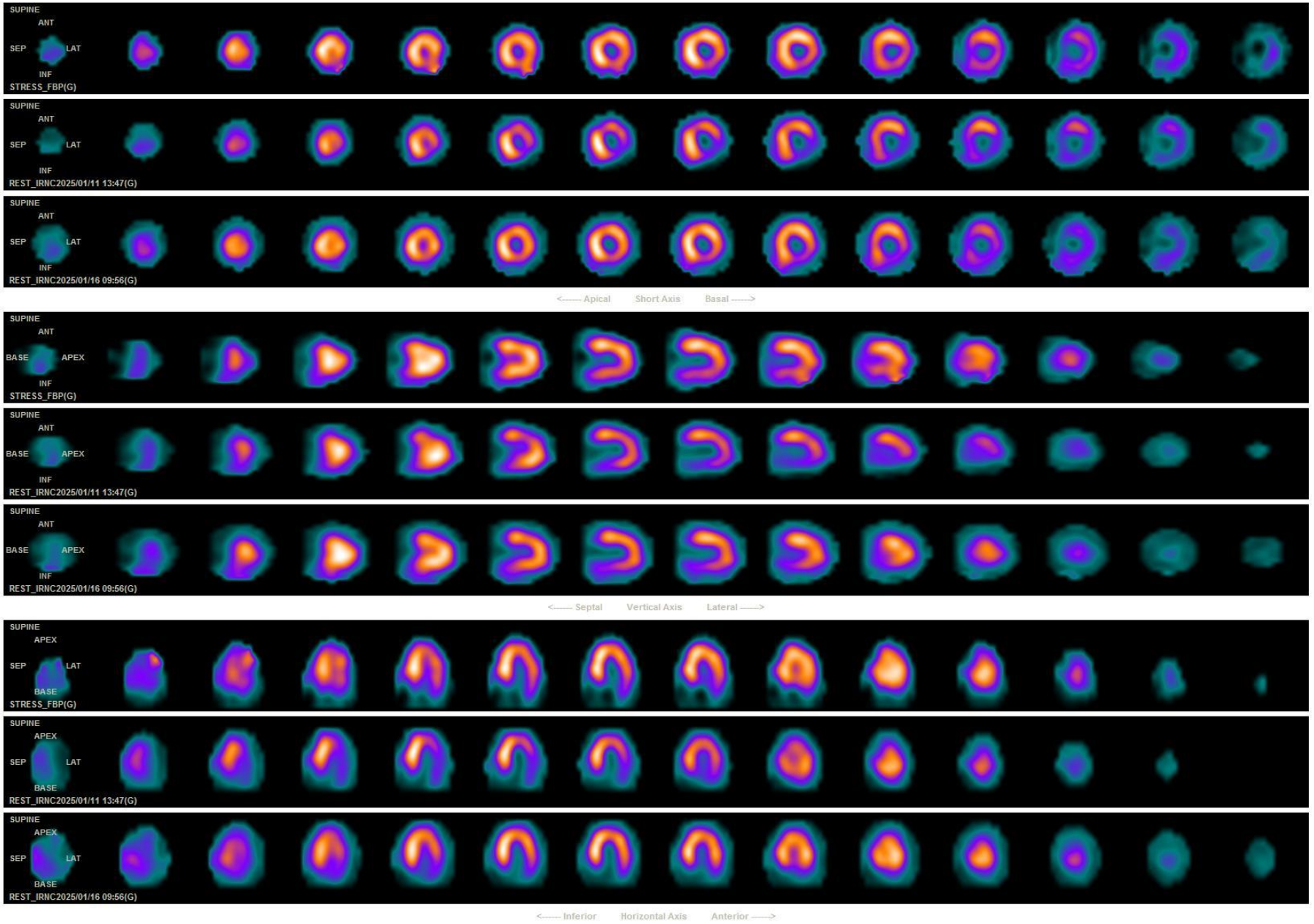
- No Hx of DM and HTN

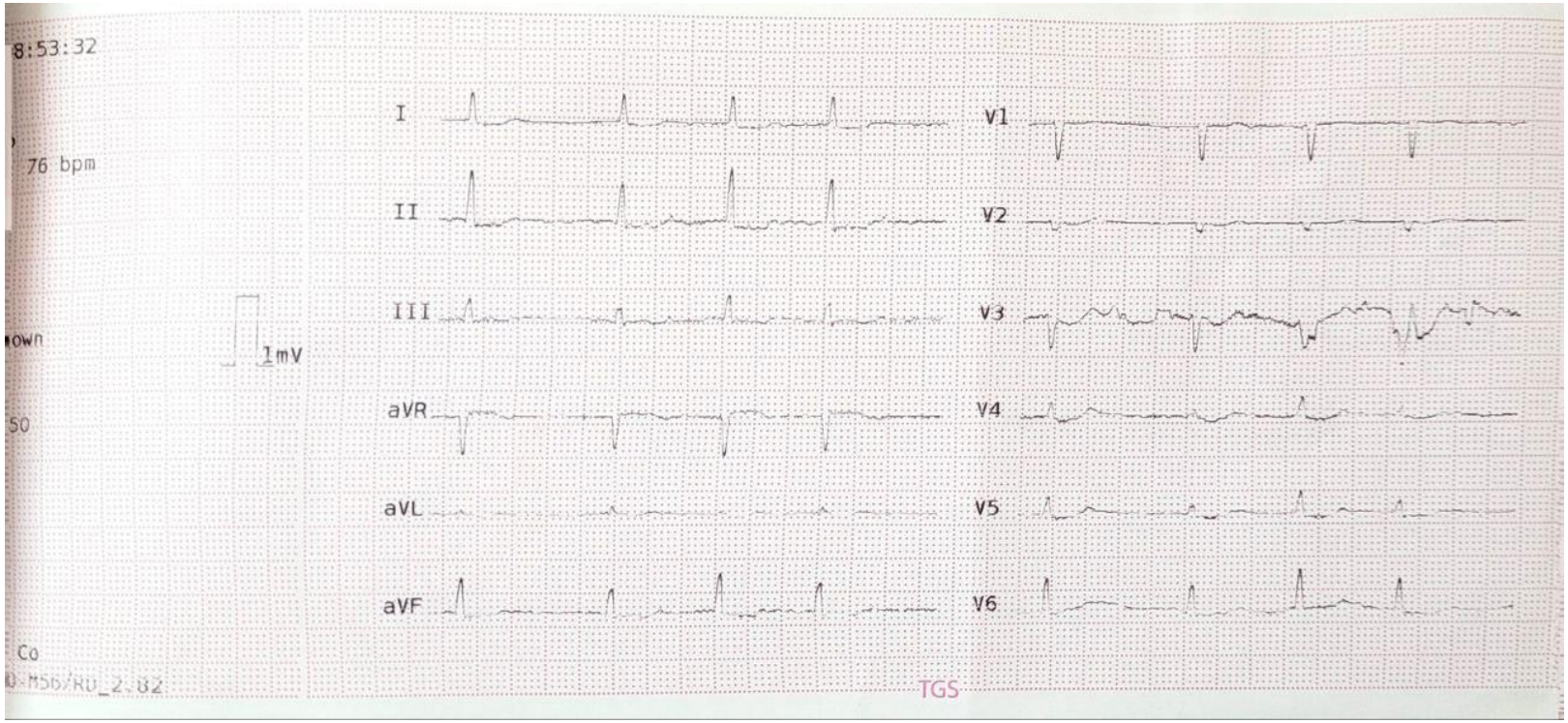
Patient Id: S0310241

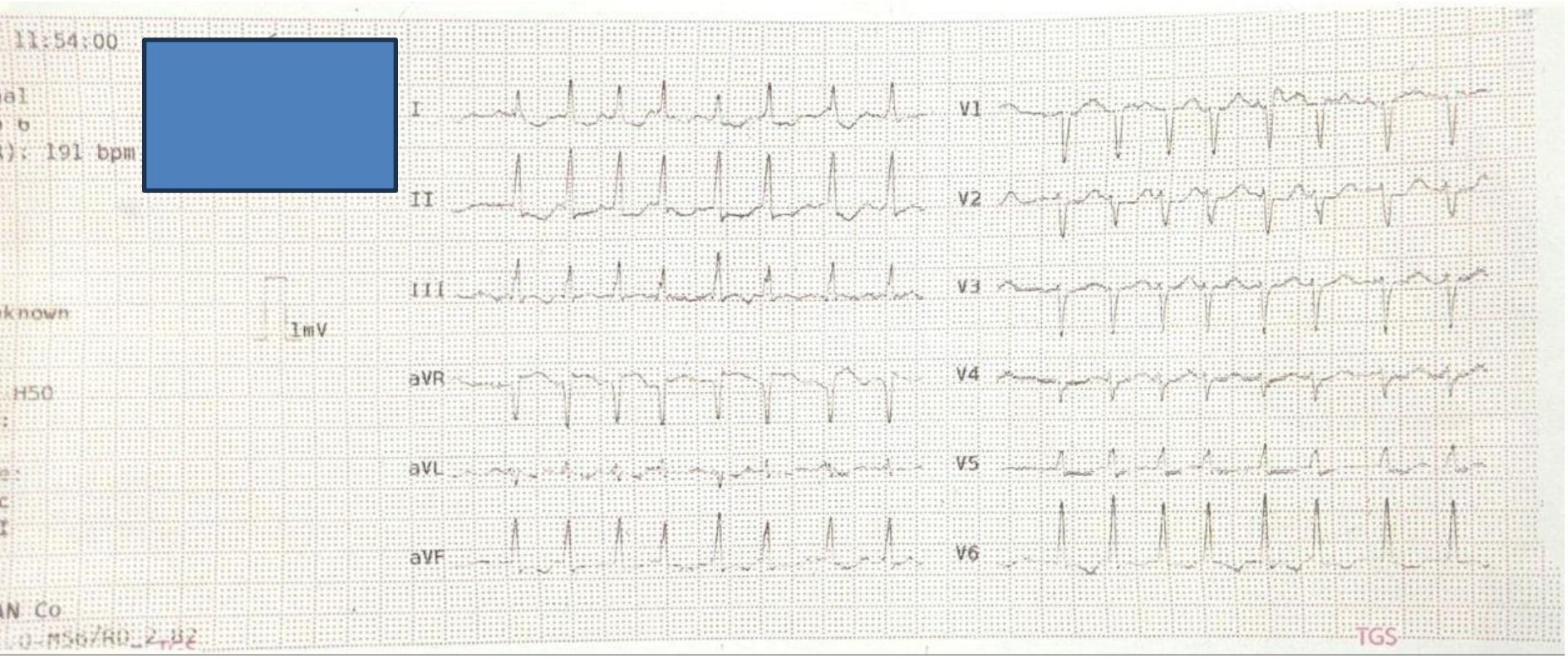
Date & Time: 1/11/2025

GHAEM HOSPITAL

Manufacturer Model: Tandem_Discovery_630




8:53:32
76 bpm
1mV
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
TGS

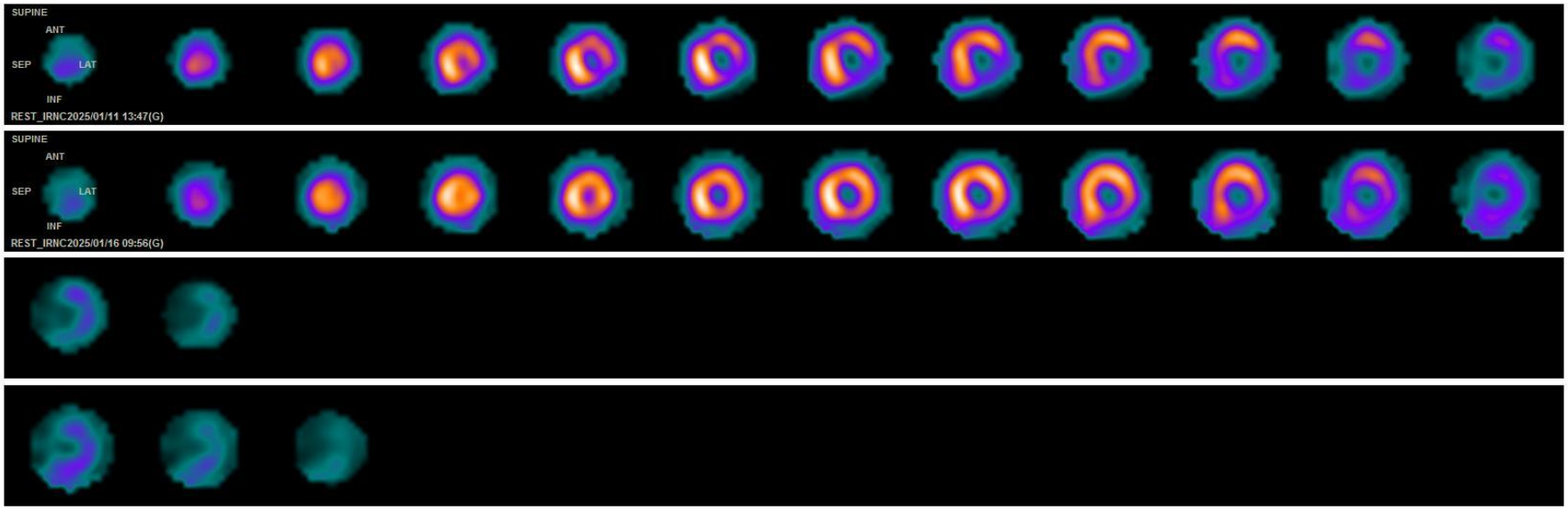

11:54:00
191 bpm
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
1mV
TGS

Patient Id: S0310241

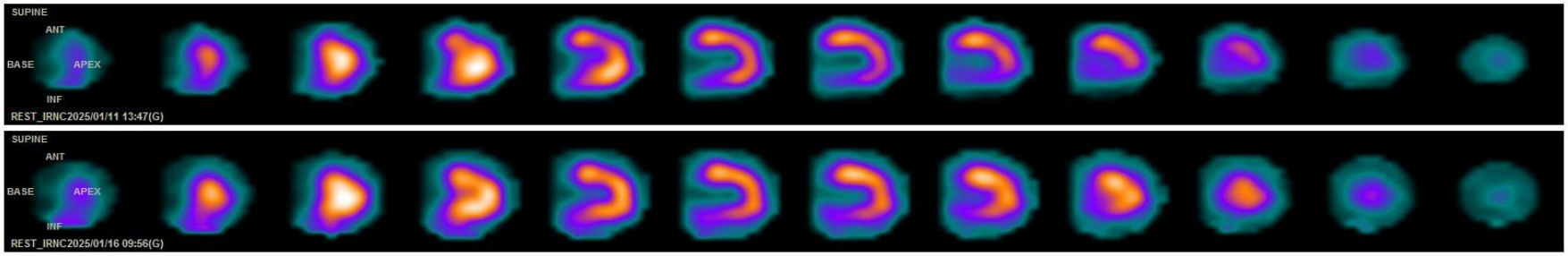
Date & Time: 1/11/2025

GHAEM HOSPITAL

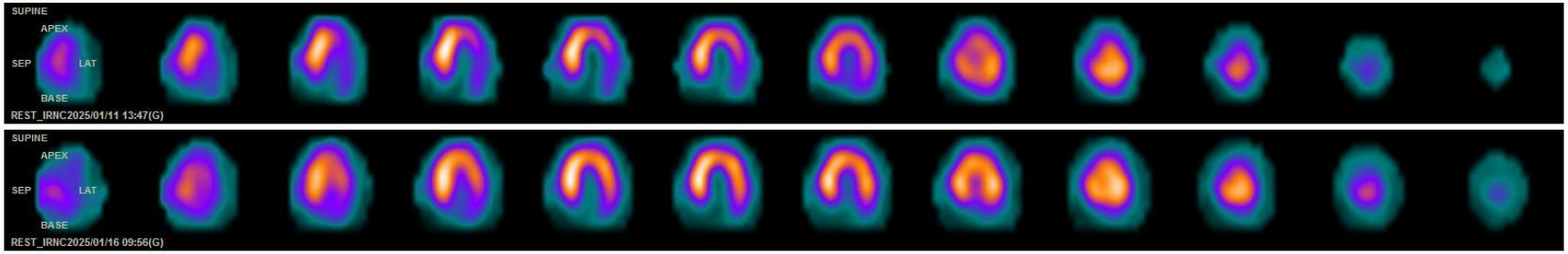
Manufacturer Model: Tandem_Discovery_630



<---- Apical Short Axis Basal ---->



<---- Septal Vertical Axis Lateral ---->



<---- Inferior Horizontal Axis Anterior ---->

To avoid artifacts and to optimize test specificity, both the technologist and interpreting physician must be aware of factors potentially contributing to the creating of artifacts.