

When CHADSVASC is not enough

young woman (47) , vet

2007: syncopal and pre-syncopal episodes.

Only after several registration of Holter ECG she had the documentation of short (a few minutes) paroxysmal atrial fibrillation.

She was classified in class 1 for the CHA₂DS₂Vasc score (only for female sex) and she was treated with aspirin.

Case report

- November 2010: the patient developed a contemporary heart attack with ST inferior elevation (STEMI) with atrial fibrillation and an acute ischaemic stroke with right hemiplegia and dysarthria, facial central paresis

(National Institutes of Health Stroke Scale NIHSS score 11)

Case report

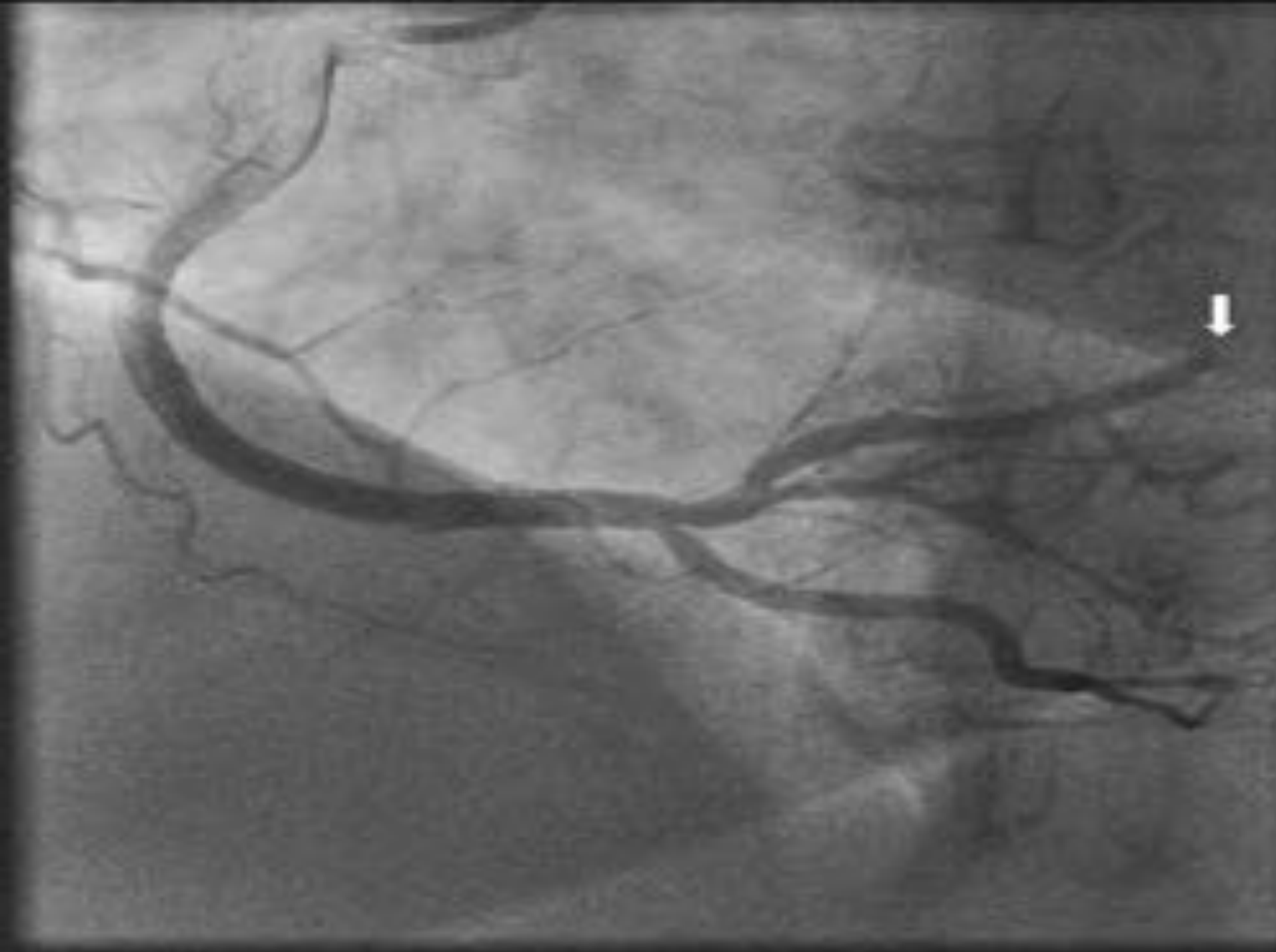
- In the emergency room the echocardiogram revealed inferior ipokinesia and absence of left atrial or ventricular thrombosis. Good left ventricular systolic function.
- CT scan excluded an aortic dissection or intracranial haemorrhage .



CT
scan

ECG





STROKE UNIT

- She was admitted and in the stroke unit and she was treated with i.v. tPA 0.9mg/kg over 1 hour (the procedure was started at 4.30 h after symptom onset) with improvement of neurological and ischaemic framework. (NIHSS score 9)

Cerebral ischemia

- The acute cerebral ischemia was located in the area of circulation of the left cerebral artery. Atrial fibrillation continued.
- A CT scan of the head post thrombolysis did not show any hemorrhagic transformation.

Follow up

- After 1 week with Low Molecular Weight Heparin patient started –autonomal decision- the anticoagulant therapy with **dabigatran** etexilate (150 mg BID) for stroke prevention in atrial fibrillation. She had a near total recovery in motor function and language.
- She remained in permanent atrial fibrillation and she is still treated with dabigatran. Subsequent investigations have ruled out the presence of foramen ovale and genetic tests for thrombophilia were negative. The left ventricular function has regained normality.

Myocardial infarction can also occur secondary to an ischemic insult in the absence of overt coronary artery disease, by an imbalance between myocardial oxygen supply and/or demand termed type 2 MI, which embodies a myriad of diseases.

List of **reported causes of myocardial infarction from coronary embolism in the absence of coronary atherosclerotic disease**

Cardiomyopathy

Rheumatic heart disease with mitral stenosis

Left ventricular aneurysm

Iatrogenic – injection of thrombus during coronary arteriography

Infectious endocarditis

Marantic endocarditis

Thromboemboli from prosthetic valve

Fibromyxoma of the aortic valve

Atrial myxoma

- Editorial

Embolic Myocardial Infarction as a Consequence of Atrial Fibrillation A Prevailing Disease of the Future

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- 2.Renu Virmani, MD;
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Circulation. 2015;132:241, published online before print June 25 2015,

Prevalence, Clinical Features, and Prognosis of Acute Myocardial Infarction
Attributable to Coronary Artery Embolism / CLINICAL PERSPECTIVE

May 15, 2015.

This is the first study that examines

prevalence

clinical features

prognosis

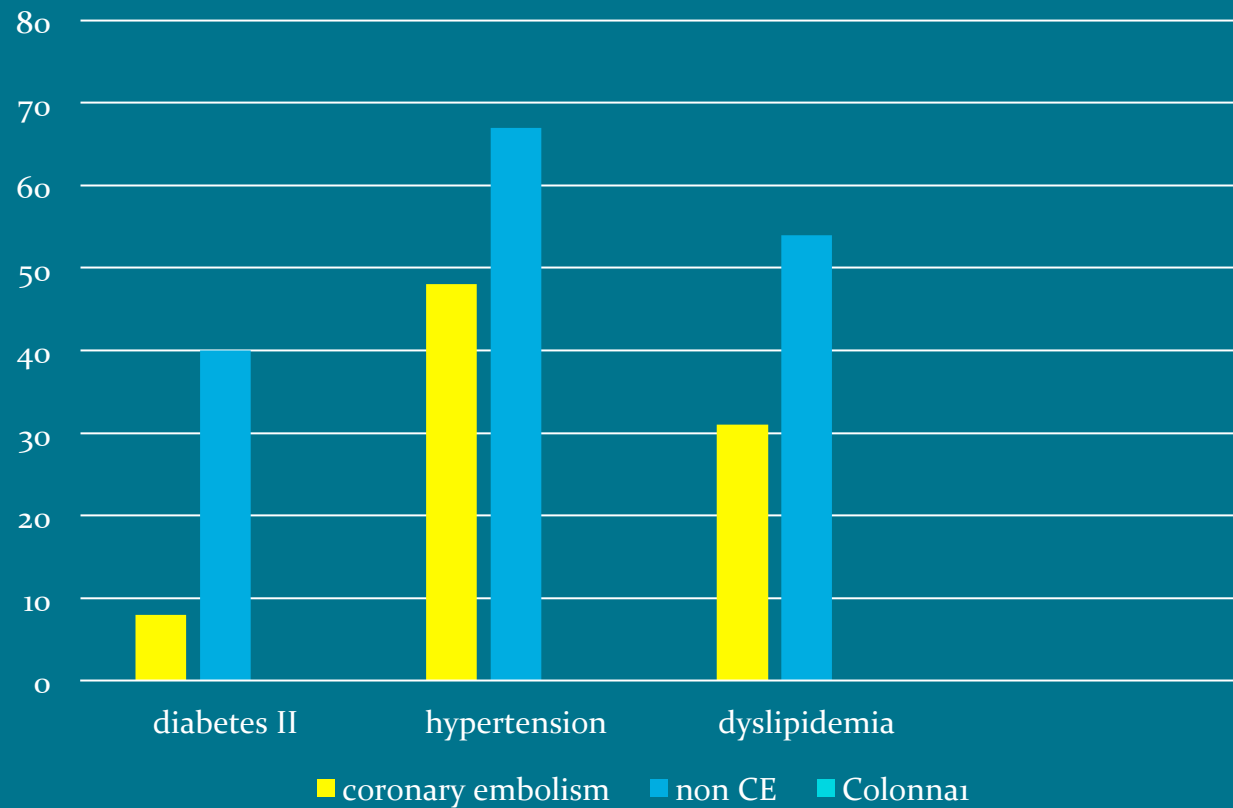
of pts with acute myocardial infarction due to
Coronary embolism.

Prevalence

2,9%

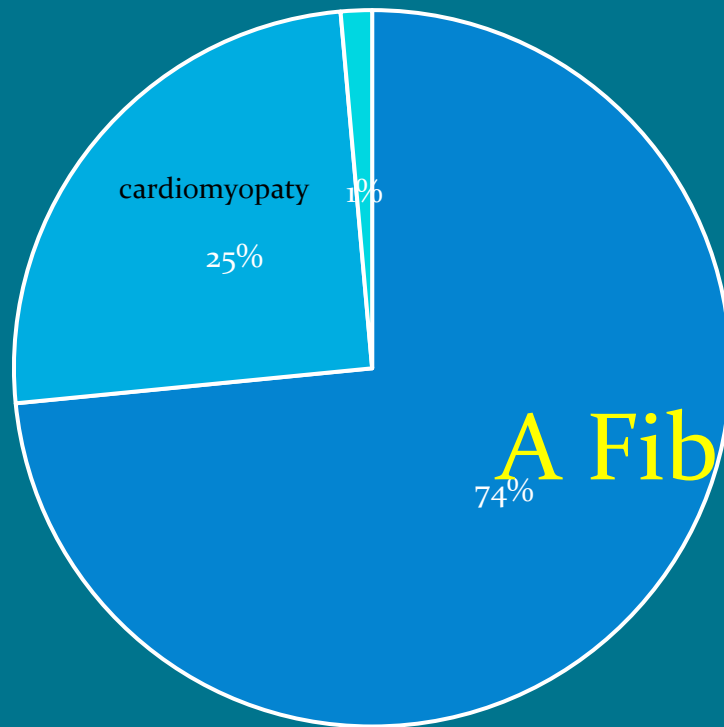
Of 2135 pts consecutively with AMI
from 2001 to 2013
52 classified as having coronary
embolism based on proposed criteria

Mean age: 66 yrs
60% male



60% of Coronary Embolism pts had a low CHADS₂ score of 0 or 1

coronary embolism



■ a fib ■ cardiomyopathy ■ valvular heart disease ■

The Authors **implicate atrial fibrillation (AF) as the most common cause of Coronary Embolism**, 38 of 52 (74%) as compared to non-AF related CE 116 of 1724 (7%) ($p < 0.001$).

Other etiologies included cardiomyopathy ($n=13$, 25%), and valvular heart disease ($n=8$, 15%).

The most common causes were **chronic AF 25 (66%)** and **paroxysmal AF 13 (34%)**.

Coronary Embolism patients **had a lower prevalence of hypertension, diabetes mellitus, dyslipidemia, smoking** and a lower number of major risk factors for CAD than non-CE AMI patients.

Over half of the Coronary Embolism patients (18/30) with non-valvular AF had CHADS₂ scores of 0 or 1

In this study only 15 pts were treated with oral anticoagulants

Prognosis

Recurrent thromboembolic episodes were identified in 10.4% of patients during follow-up (49-months) and were therefore more likely to benefit from treatment with oral anticoagulants. Long-term outcomes between the Coronary Embolism and non-CE cohorts by Kaplan-Meier showed a significantly **higher incidence of all-cause death** (hazard ratio [HR], 3.82; 95% confidence interval [CI], 2.06 to 6.48; $p < 0.001$) and **cardiac death** (HR, 5.39; 95% CI, 2.38 to 10.6; $p < 0.001$) in Coronary Embolism group relative to the non-CE.

Although, thromboembolism in the absence of CAD is reported as an infrequent cause of AMI, the consequences are potentially life threatening. **Distal embolization is clinically recognized by coronary angiography as “small peripheral stops”, which are otherwise indicators of poor prognosis .**

The overall incidence and prevalence of “non-atherosclerotic” embolic events resulting in myocardial ischemia and infarction remains unknown, as for the most part, embolic infarcts are clinically under-recognized.

Proposed criteria for the clinical diagnosis of Coronary Artery embolism : two or more major criteria , or one major plus two minor, or three minor criteria.

MAJOR CRITERIA:

- Angiographic evidence of coronary embolism without atherosclerotic component
- Concomitant cor embolization at multiple sites
- Concomitant systemic embolization without left ventricular thrombus

MINOR CRITERIA:

- <25% stenosis on angiography except for the culprit lesion
- Evidence of an embolic source based on transthoracic echo, TEE, or MRI or CT
- Presence of embolic risk factors: a fib, cardiomyopathy, rheumatic valve dis, prosthetic heart valve, patent foramen ovale, atrial septal defect, cardiac surgery, infective endocarditis, hypercoagulable status

Diagnosis of coronary artery embolism should **not** be made if there is :

- Pathological evidence of atherosclerotic thrombus
- History of coronary revascularization
- Coronary artery ectasia
- Plaque disruption or erosion in the proximal part of the culprit lesion

Acute stroke with concomitant acute myocardial infarction

In our case another problem is
Is appropriate perform
thrombolysis?

(AMI within the previous 3 months is considered a relative contraindication for therapy with alteplase or rtPA for associated increased risk of cardiac rupture secondary to breakdown of the existing fibrin clot within the necrotic myocardium and degradation of collagen).



Cardiogenic Stroke Despite Low CHA₂DS₂-VASc Score

Assessing Stroke Risk by Left Atrial Appendage Anatomy (ASK LAA)

Sotirios Nedios, M.D.; Emmanuel Koutalas, M.D.; Jelena Kornej, M.D.; Sascha Rolf, M.D.; Arash Arya, M.D.; Philipp Sommer, M.D.; Daniela Husser, M.D.; Gerhard Hindricks, M.D.; Andreas Bollmann, M.D., Ph.D.
J Cardiovasc Electrophysiol. 2015;26(9):915-921

The main objective of the study was to analyze the morphology and other LAA characteristics in AF patients with **low CHA₂DS₂-VASc score (≤ 1)** in order to identify their association with prior thromboembolic events, using a case–control study.

Univariate analysis showed that TE patients had a higher incidence of superior LAA takeoff (i.e., higher than the left superior PV; 28% vs. 4%, $P = 0.002$) and a higher incidence of hyperlipidemia (40% vs. 17%, $P = 0.028$), while LAA morphologies, inferior takeoff, and other LAA characteristics were similar between groups. Logistic regression revealed that a superior LAA takeoff (OR: 9.1, 95% CI: 2.1–38.6, $P = 0.003$) was the only independent predictor of TE.

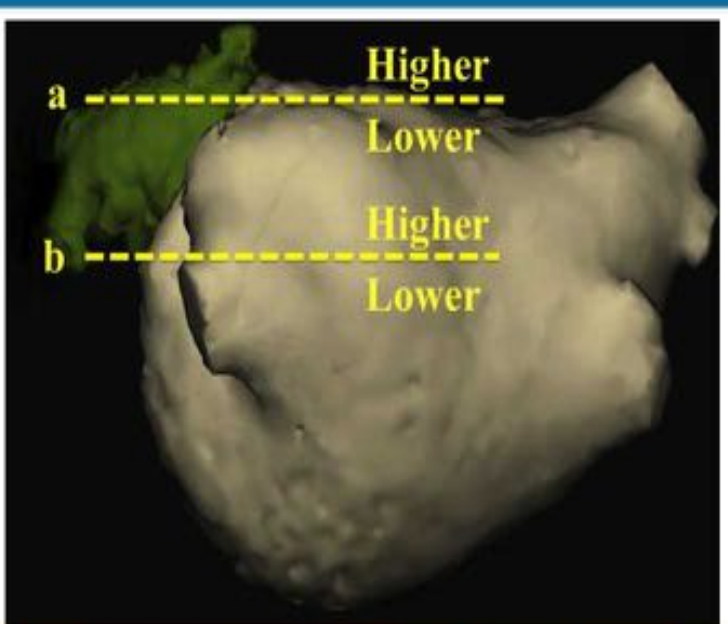


Di Biase L, Santangeli P, Anselmino M et al. Does the left atrial appendage morphology correlate with the risk of stroke in patients with atrial fibrillation?: results from a multicenter study. **J Am Coll Cardiol.** 2012 Aug 7;60(6):531-8.

The authors hypothesize that complex internal anatomy of cauliflower and windsock type LAA leads to increased blood stasis and thrombogenesis when compared simple LAA anatomy of chicken wing and cactus type LAA. As SCI has been shown to predict symptomatic strokes, the authors suggest that therapies directed at reduction/prevention of SCI may prevent early cerebral damage in AF patients.

Systematic analysis of the characteristics and quantitative parameters of LAA in low-risk patients with and without TE allowed to carefully assess the impact of LAA anatomy. A high superior LAA-takeoff is associated with a 9-fold higher risk of thrombogenesis.

In conclusion: the LAA anatomy might be useful for predicting strokes and guide anticoagulation management of patients with AF and low $\text{CHA}_2\text{DS}_2\text{-VASc}$ score.





cactus



cauliflower



chicken wing



windsock





A. Wing & Hook



B. Wing & Hook



C. Wing & Hook



D. Double Wing



E. Double Wing



F. Wing & Finger



G. Wing, Hook
& Knob



H. Double Hook
& Wing



I. Double Knob, Hook
& Arrowhead



J. Finger, Wing,
Hook & Knob



A. Hook



B. Seahorse



C. Finger (with knob)



D. Knob



E. Wing



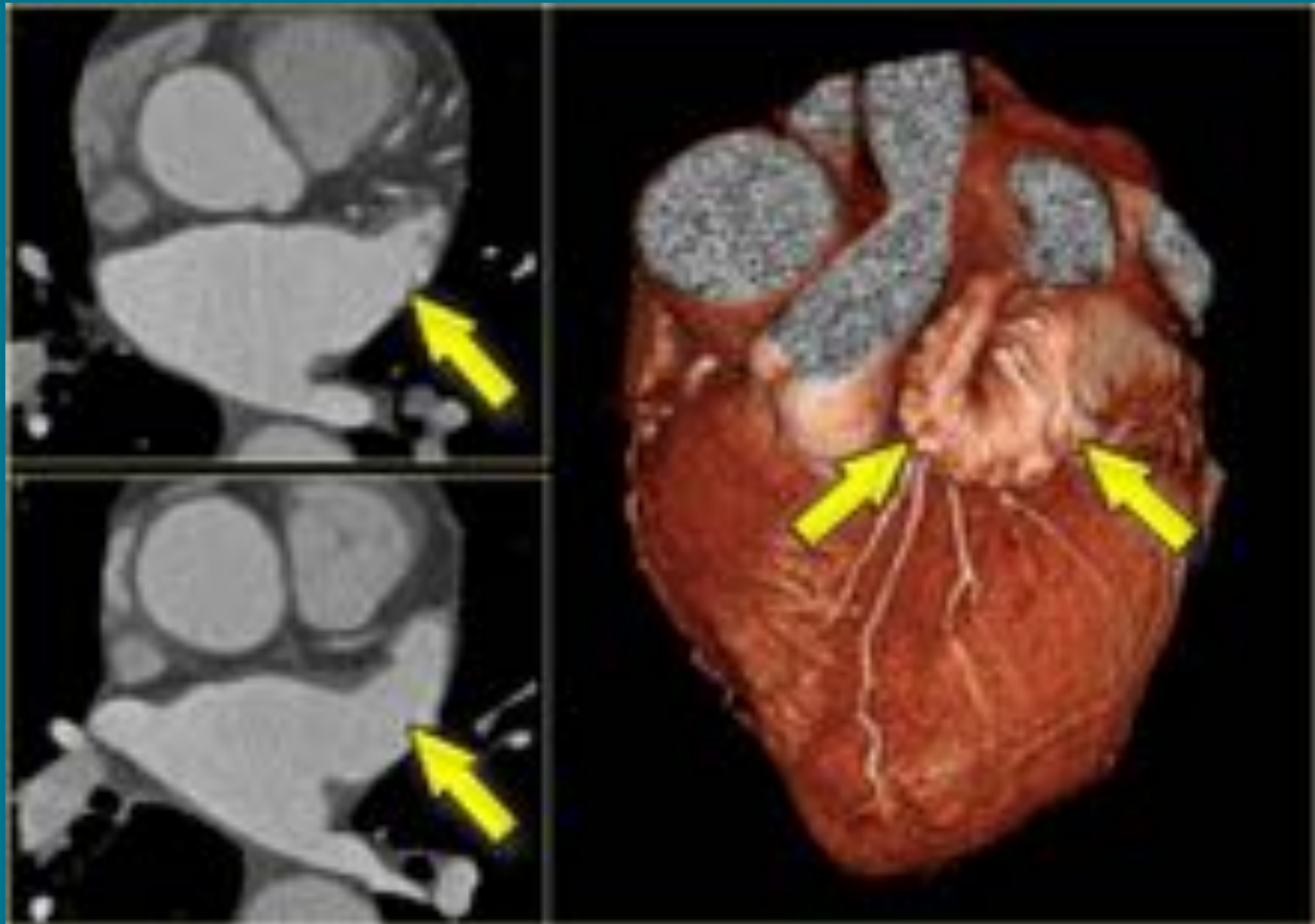
F. Sprial

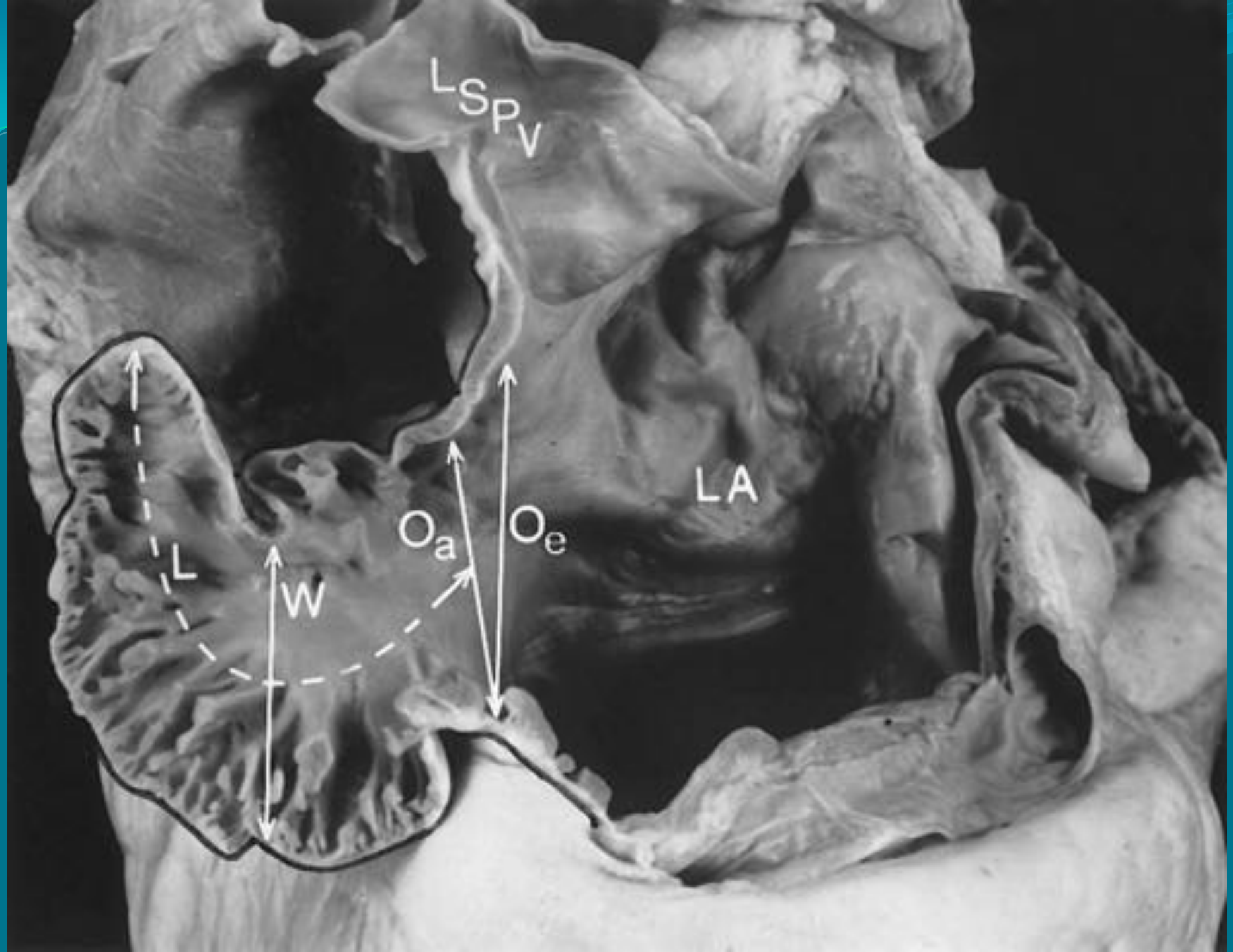


G. Flame



H. Arrowhead





The limited reported incidence of coronary artery emboli, particularly in nonatherosclerotic CAD may result from several levels of protection provided by the **swift current flow around the coronary ostia**, and/or caliber differences between the aorta and the coronary arteries, and the acute angle at which the coronary artery originates from the aorta.

The position of the coronary ostia behind the valve cusp during systole may also guard the coronary arteries from the central stream of the systolic blood flow.

Not only are the coronary arteries relatively protected from emboli, compared with other organs, but the frequency of multiple emboli from the left atrial appendage also appears lower :documented in 8 of 32 (25%) patients with AF associated Coronary Embolism. It is therefore, likely that Coronary Embolisms are under-diagnosed, for reasons including a failure to distinguish embolism from thrombosis, under-reporting limited to only the most dramatic cases, or a failure to make a systematic search for small emboli in the distal and intramural branches of coronary arteries .

CONCLUSION:

The left arterial appendage (LAA) is an important cardiac structure with embryologic, anatomic, and functional differences in reference to the left atrium (LA). Most intracardiac thrombi originate in the LAA during and after AF.

Our case illuminates the role of the left atrial appendage in the occurrence of thromboembolic events in a patient with low CHA₂DS₂VASC score (<1). Therefore the LAA anatomy might be useful for predicting stroke risk and guide anticoagulation management of pts with AF despite conventional clinic low stroke risk.



a multicenter retrospective study in which 359 AF patients undergoing catheter ablation were screened. LAA morphology was assessed by cardiac magnetic resonance (MR) or computed tomography (CT) and classified into 1) cactus LAA 2) chicken wing LAA 3) wind sock LAA and 4) cauliflower LAA.

Major LAA Types

The Wind Sock Type LAA is an anatomy in which one dominant lobe of sufficient length is the primary structure



The Chicken Wing Type LAA is an anatomy whose main feature is a sharp bend in the dominant lobe of the LAA anatomy at some distance from the perceived LAA ostium



The Broccoli Type LAA is an anatomy whose main feature is an LAA that has limited overall length with more complex internal characteristics

