WIDE SUSTAINED QRS TACHYCARDIA: Aberrancy versus Ectopy

Raimundo Barbosa Barros- Fortaleza-Ceará – Brazil

Prezado professor: este é mais um caso que se encontra internado na nossa enfermaria. Masculino, 69 anos, diabético tipo 2, admitido com dor epigástrica, palpitaçôes e cansaço.

Dados clinicos adicionais enviarei posteriormente. O ECO mostra insuficiência mitral importante com hipocinesia difusa e FE baixa e VE e AE dilatados.
Coronariografia: Normal

Pergunta:
- Taquicardia ventricular TV?
- Taquicardia supraventricular com aberrância TSV?

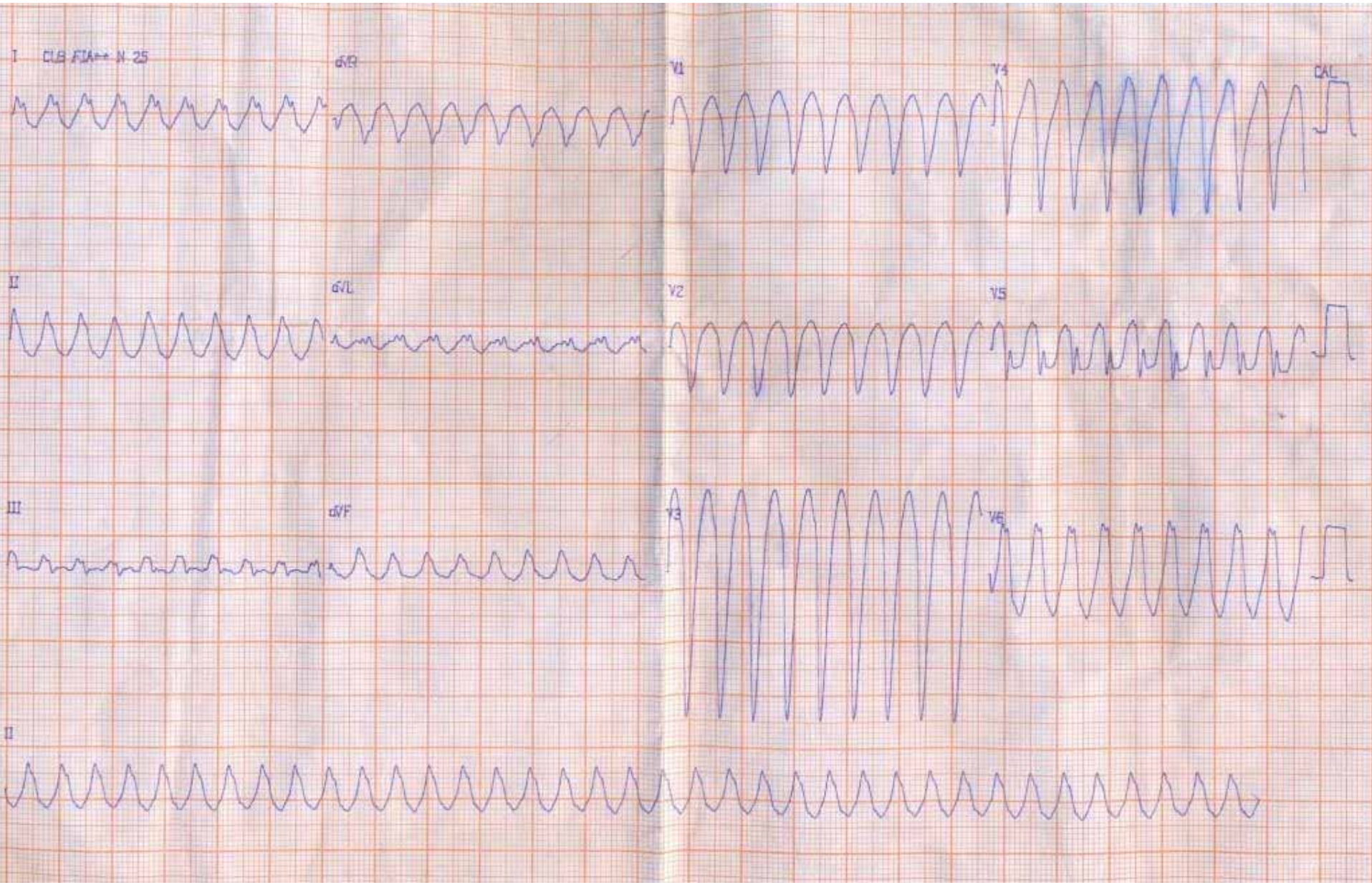
Raimundo Barbosa Barros Fortaleza Ceará

Dear Professor: This is another case admitted in our Hospital.
Male, 69 years, type 2 diabetic. He complained of epigastric pain, palpitations and fatigue.

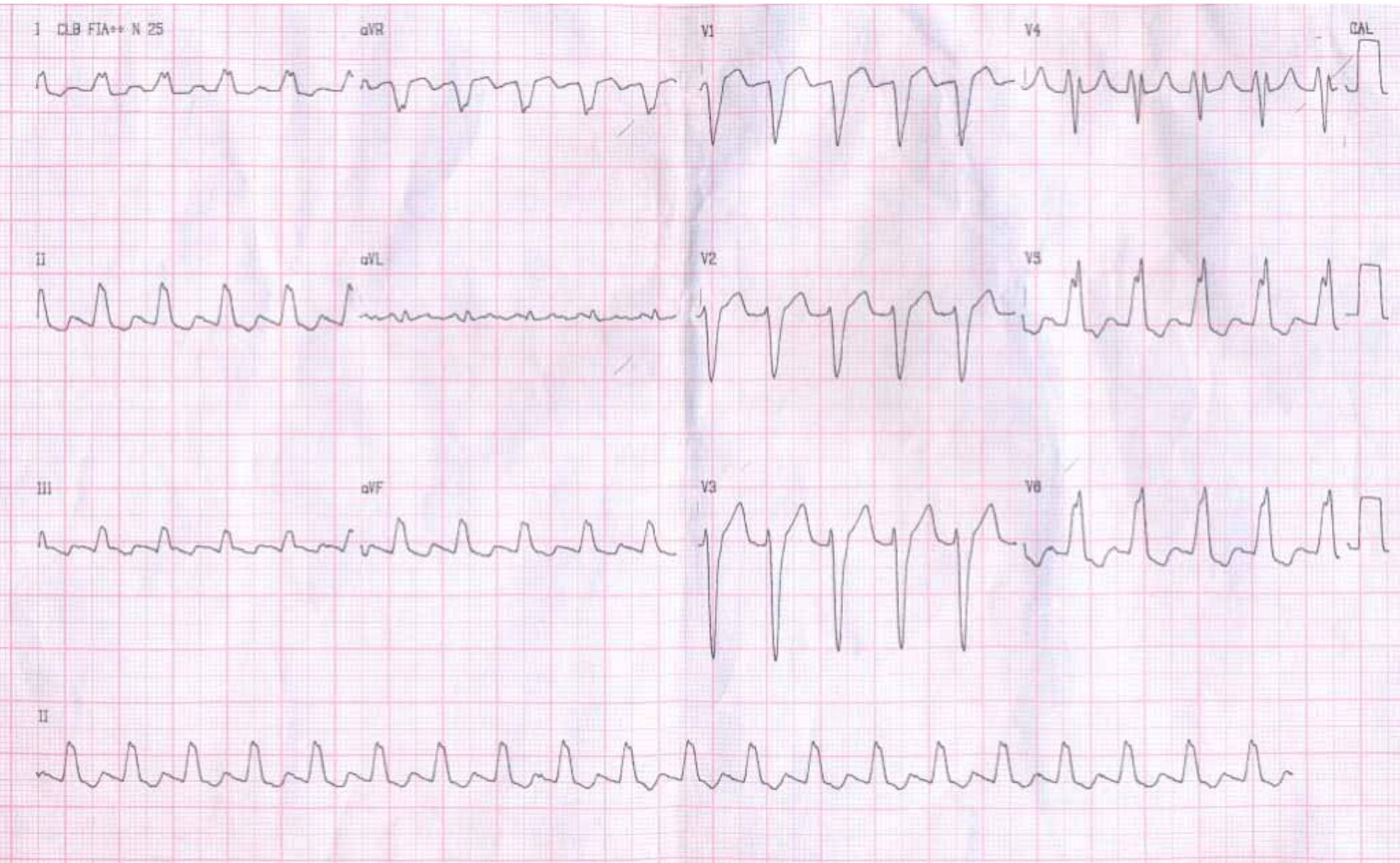
Additional clinical data I will send later. ECHO shows severe mitral insuficience, difusse hypocinesia, low EF and dilated LV and LA.
Normal coronariography.

Question:
Ventricular tachycardia TV?
or Supraventricular tachycardia with aberrancy TSV?

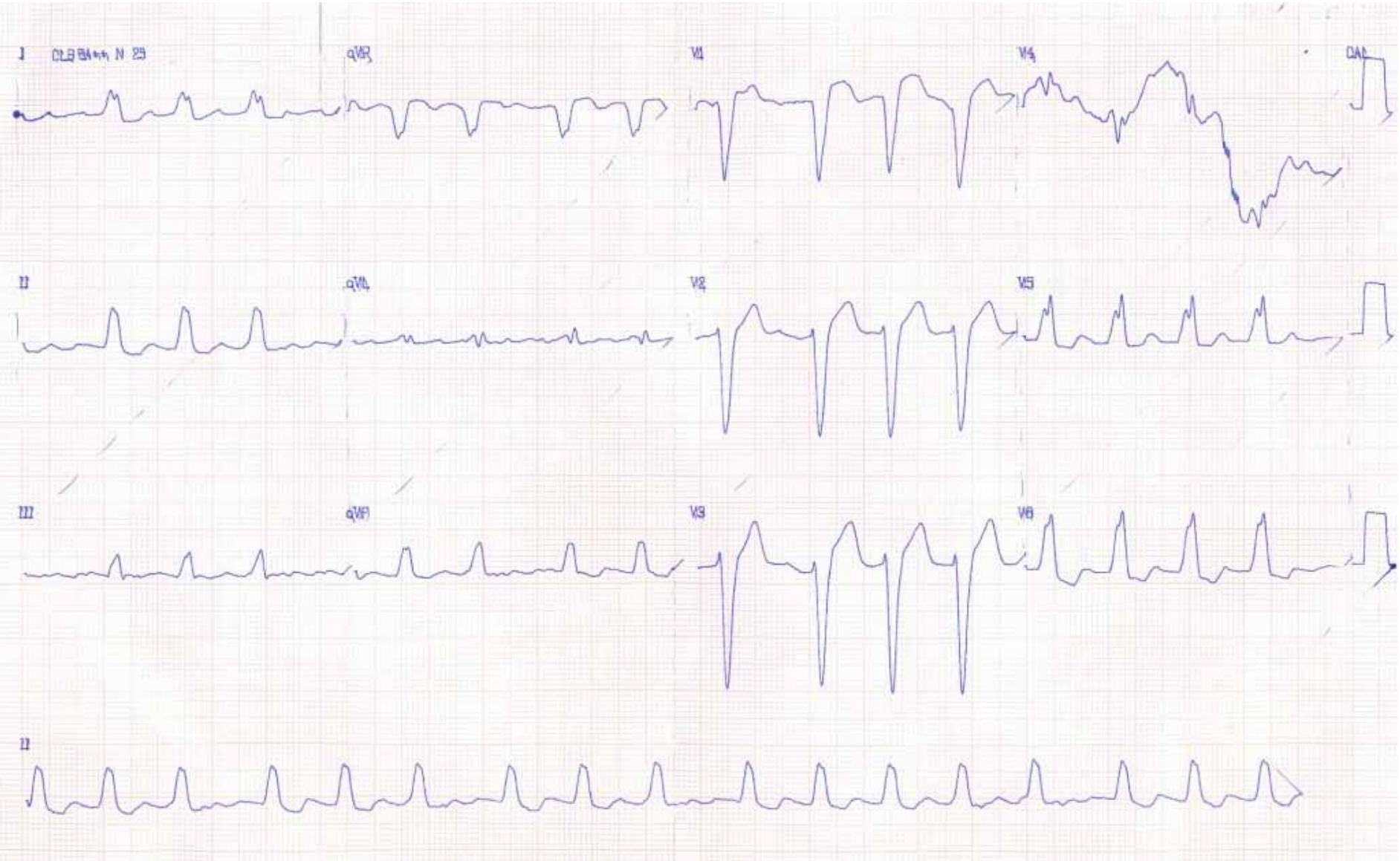
First ECG



Second ECG after IV amiodarone



Third ECG



Colleagues opinions

Estimados amigos:

Algunos datos pienso que sugieren el origen ventricular de la taquicardia a saber: Ausencia de complejos RS en las precordiales, complejo de tipo QR en V5 y en la tira de ritmo parece apreciarse disociación VA (latido 4, 11…).

Por la edad y los antecedentes podría ser de etiología isquémica con foco de origen en el septo alto (perimitral), aunque no se pueden ser descartartadas la displasia de ventrículo derecho y la taquicardia ventricular idiopática del tracto de salida del ventrículo derecho.

Menos probablemente puede ser una taquicardia prexcitada (vía accesoría nodoventricular).

Sería interesante ver un ECG en ritmo sinusal.

Dr. Jerónimo Rubio Sanz Area de Electrofisiología y Estimulación Hospital Clínico Universitario Valladolid- España

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Dear Friend

I think that some data suggest ventricular origin: Ventricular tachycardia: Lack of RS complex in precordial leads, QRS complexes QR type on V5 and the pace seems to have been pulled VA dissociation (beat 4, 11 ...).

By age and background could be of ischemic origin with focus on the high septum (perimitral), but can not discard right ventricular dysplasia or right ventricular outflow tract idiopathic ventricular tachycardia of right ventricle. Less likely to be a prexcited tachycardia (nodoventricular accessory pathway). It would be interesting to see an ECG in sinus rhythm.

Queridos El Potro e Mr. Fox

Me parece TVMS com padrão de BCRE portanto de VSVD

Dear Andres and Raimundo: I think that it is a VT from RVOT because LBBB pattern with inferior axis.

Adail - Bahia – Brasil

ECG2: Aleteo auricular tipico, istmo dependiente, activacion antihoraria, conducción 2:1. Bloqueo de rama izquierda
ECG3: FA, bloqueo de rama izquierda Me estoy perdiendo de algo mas?
Saludos
AB

ECG2: typical atrial flutter(AFL), isthmus dependent counterclockwise activation, 2:1 conduction. LBBB
ECG3: Atrial fibrillation (AF), left bundle branch block(LBBB)
Am I missing something else?
AB

Dr Barancha
Eu penso que o ECG da admissão sugere Flutter atrial com condução AV 1:1 associado com bloqueio de ramo esquerdo. O que voce acha?

I think that the admission ECG suggest AFL with 1:1 AV conduction associate with left bundle branch block. What do you think. Raimundo Barbosa Barros.

Querido Raimundo: El profesor Edgardo me mando el primer ECG offline, y me recordó que fue rotulado como TV. Puede ser, pero el eje es semejante al nuevo ECG mostrado que tiene flutter auricular, por lo tanto flutter con conduccion 1:1 debiera permanecer como diagnostico diferencial. Sin embargo parece tener disociación, lo que habla en favor de TV. No seria el primer caso en tener TV, AA y FA. Sin embargo, insisto en el ECG 2, impresiona Aleteo Auricular tipico.
Saludos
AB

Dear Raimundo: Professor Edgar sent me the first ECG offline, and reminded me that was labeled as a VT. Perhaps, but the shaft is similar to the new ECG showed AFL has therefore should flutter with 1:1 conduction remain differential diagnosis. However it seems to have dissociation, which speaks in favor of VT. It would not be the first case to have VT, AA and AF. However, I insist on the ECG 2, impresses typical AFL.
GreetingsAB

Caro Raimundinho Você pensa que o primeiro ECG é flutter atrial com condução AV 1:1. No entanto flutter atrial pode conduzir ao ventrículo em uma forma de 1:1, produzindo uma taxa ventricular de 300 batimentos por minuto, ou perto disto apenas em crianças, em pacientes com a síndrome pré-excitação, naqueles cujos nós AV conduzem rapidamente e, ocasionalmente, em pacientes com hipertireoidismo.O presente caso teria alguma destas condições? Andres.

Dear Raymon "the fox": You think that the first ECG has AFL with 1:1 AV conduction. However AFL can conduct to the ventricle in a 1:1 fashion, producing a ventricular rate of 300 beats or near per minute in children, in patients with the pre-excitation syndrome, in those whose AV nodes conduct rapidly, and occasionally in patients with hyperthyroidism. The present case has someone of these conditions? Andrés

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O paciente tem 69 anos. Não há sinais de pré-excitação. Estamos aguardando provas de função tiroidiana e coronariografia.O ECO é compatiível com insuficiencia mitral importante e disfunção ventricular.Eu pensei em flutter atrial 1:1 devido a morfologia de BRE semelhnate aos outros ECGs com redução da FC. The patient is 69yo, he has not preexcitation, we are waiting the coronariography and thyroid tests. I think in flutter 1:1 because LBBB in other ECGs with < HR
Raimundo

Hola amigos, feliz año para todos
Con respecto al caso de nuestros amigos brasileños, 69 años, con factores de riesgo cardiovascular, taquicardia con QRS ancho, es TV hasta que se demuestre lo contrario. Si utilizamos el algoritmo de aVR propuesto por Vereckei et al, presenta onda q en aVR inicial mayor a 40 ms, al mismo tiempo el complejo es QS, con una rampa inicial muy rapida (hasta el nadir)y luego un ascenso lento, con una relación menor que 1, ademas en la tira de DII impresiona disociación AV, confirmandose eventualmente el diagnostico de Taquicardia Ventricular. Igual no se porque no mandan mas datos clinicos. Pero por otro lado me llama la atención primero que esta taquiarritmia no sea al menos presincopal, ya que es rapidisima o no refieran tampoco fallo de bomba agudo.
Otra cosa es que hicieron amidoarona cuando lo indicado seria cardiovertirlo, aunque podemos discutir como lo hemos hecho antes, que en teoria el paciente "no presentaba compromiso hemodinamico" y por lo tanto me da tiempo para hacer un diagnostico diferencial con distintas maniobras como MSC, electrograma intraesofagico, etc. Los siguientes ECG si impresionan en primer lugar un aleteo auricular tipico 2:1 y posteriormente una FA. Ahora bien me gustaria saber si este paciente en forma ambulatoria ademas de seguro presentar alguna cardiopatia, y tal vez lo mas probable de origen isquemico, presentaba episodios de FA paroxisticos o TA o flutter y en algun momento recibio propafenona o flecainida, porque en ese caso no seria descabellado pensar en un aleteo auricular 1:1 por efecto proarritmico, y otra opcion es que como dijo Adrian, convivan las tres entidades.
Saludos y un gusto volver a leer como se han venido masacrando estos ultimos dias,

Dr. Francisco Jose Femenia Unidad de Arritmias, Departamento de Cardiología, Hospital Español de Mendoza, Argentina.

Hello friends, happy new year for everyone.

About the case from our Brazilian friends: 69-year-old patient, with cardiovascular risk factors, tachycardia with wide QRS. This is VT until the contrary is proven.
If we use the aVR algorithm proposed by Vereckei et al, he presents q wave in initial aVR greater than 40 ms, while at the same time the complex is QS, with a very fast initial slope (up to the nadir) and then a slow take off with a ratio lower than 1; besides in the strip of DII, there is apparent AV dissociation, with the diagnosis of ventricular tachycardia finally being confirmed. Anyway I don't know why they are not sending more clinical data.
But on the other hand, it strikes me first that this tachyarrhythmia is not at least pre-syncopal, as it is very, very fast, or that there is no mention of acute pump failure.
Another issue is that they administered amiodarone when the indication is cardioversion, although it could be argued as we did before, that in theory the patient "did not present hemodynamic compromise" and therefore there is time to make a differential diagnosis with different maneuvers like carotid sinus massage, intraesophageal electrogram, and so on.
The following ECGs do seem in the first place, to be typical 2:1 AFL and subsequently AF. Now, I would like to know if this ambulatory patient, besides presenting some heart disease for certain, most likely of ischemic origin, presented paroxysmal AF or AT or flutter episodes, and at some time received propafenone or flecainide, because in this case it would not be crazy to think of 1:1 AFL by proarrhythmic effect, and another option is like Adrian said, for the three entities to coexist.

Regards, and it is nice to read again how you have been decimating each other these last few days,
Francisco Jose Femenia MD(Nickname: **hot-dog.)** ***Arrhythmia Unit, Department of Cardiology, Spanish Hospital of Mendoza, Argentina.***

ECG 1: AFL with 1:1 + LBBB. Bundle branch reentry VT should be discussed in ECG 1. But this probability is 1/10000000
ECG 2: AFL with 2:1
ECG 3: AF + LBBB

ECG1: Aleteo atrial 1:1+ BCRI. TV por reentrada rama a rama podria ser discutida en el ECG1 pero esta posibilidad es remota(1/1000000000)
ECG2: Aleteo auricular 2:1
ECG3: FA+ BCRI.

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Estimados amigos del foro
Con respecto a la taquicardia con QRS ancho pienso que se trata de una TV por reentrada entre ramas. Tiene elevada frecuencia, BCRI y hay descenso rapido de la "s" en precordiales derechas, sugiriendo activacion del miocardio a traves del sistema His-Purkinje. Tal vez tenga una miocardiopatia dilatada -con tantos mails enviados no recuerdo si en alguno vi que tenia deterioro de la función ventricular.- Los otros dos trazados, post amiodarona, muestran el primero(ECG2) = aleteo auricular el segundo(ECG 3) = fibrilacion auricular.Los QRS siguen mostrando BCRI signo que hace pensar en daño miocardico (30% de las miocardiopatias dilatadas cursan con BCRI).

Abrazo a todos

Dr. Carlos Lavergne Neuquén Patagonia Argentina.

Dear Forum´s Friends
With regard to the wide QRS tachycardia I think that it is a Bundle Branch Renetrant VT because has high frequency, LBBB pattern, rapid descendent “S" in right precordial leads, suggesting myocardial activation through the His-Purkinje system. Maybe he has a dilated cardiomyopathy- mails sent so many do not remember if I saw some deterioration has ventricular function .- In the other two ECGs, post amiodarone, we observe:
ECG2 = AFL
ECG 3 = atrial fibrillation(FA). The QRS continued showing LBBB pattern that is a signal that suggests myocardial damage (In ≈ 30% of cases of dilated cardiomyopathies show LBBB ECG pattern)

Embrace all
Carlos Lavergne MD Neuquén Patagonia Argentina

FINALS COMMENTS

Andrés Ricardo Pérez-Riera MD

Chief of Electrovectorcardiogram sector - ABC Faculty – ABC Foundation- Santo André- São Paulo Brazil

1. ECG 1: **Broad sustained LBBB-pattern regular tachycardia, HR 231 beat/min, QS pattern in right precordial leads and the typical pattern of LBBB is also seen in left lead V6(Pure broad R wave no Q and no S), the down stroke of the QS wave in V_1 is clean and swift (no slurs or notches), the distance from the beginning of the QRS complex to the nadir of the QS wave is < 60ms and QRS duration <160ms in V_1 negative patterns. Finally, the ECGs 2 and 3 with similar QRS axis and LBBB-pattern to that of the ECG1. Conclusion: AFL 1:1 with LBBB pattern.**

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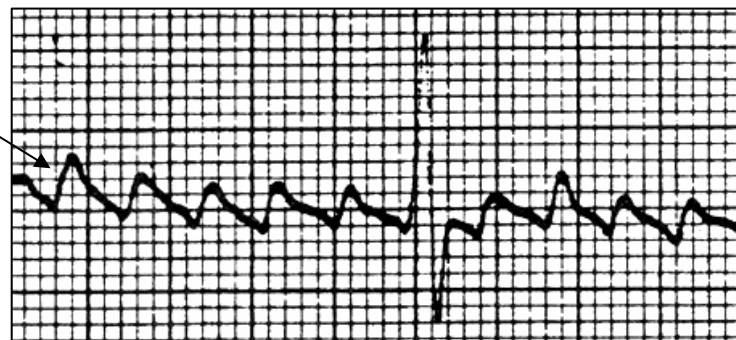
1. **ECG1: Taquicardia sustentada de QRS largo, com padrão de BCRE, regular, FC 231bpm, QS nas precordiais direitas e padrão típico de BRE também na precordial esquerda V_6 (R pura em torre não Q e não S), a rampa descendente limpa e rápida da onda QS em V_1 (sem borrões ou entalhes), a distância a partir do início do complexo QRS ao nadir da onda S < 60ms e a duração do QRS <160 ms com V_1 mostrando complexo QRS negativo. Finalmente, os ECGs subseqüentes 2 e 3 que possuem um eixo elétrico similar e de BCRE ao do ECG1. Conclusão: Flutter atrial 1:1 com padrão de BCRE.**

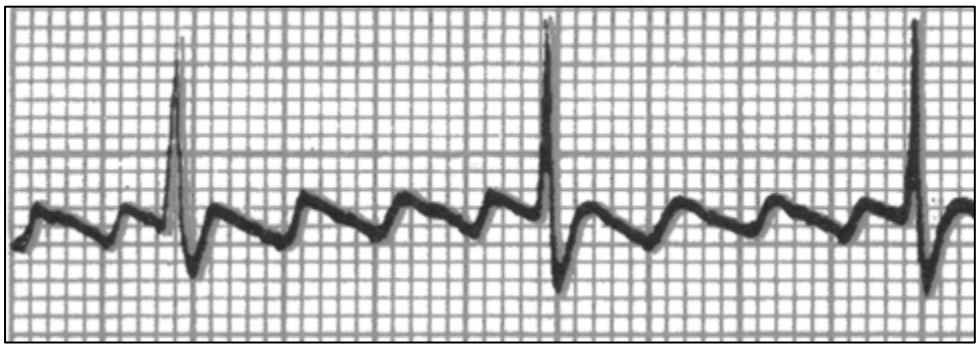
AFL with 1:1 AV conduction (rare) is a medical emergency. The ventricular rate near 300 bpm must be treated immediately. 1:1 AV conduction may be found in the following circumstances: Preexcitation of WPW type, because the stimulus is conduced in anterograde fashion by the anomalous pathway, AFL secondary to hyperthyroidism, flutter of the pediatric group, consequence of initial use of IA class drugs (quinidine, procainamide or disopyramide) by atrial slowing and by vagolytic anti-cholinergic action in the AV junction that this group of drugs causes, especially if the drugs were used without administering dixogin, calcium antagonists or β-blockers previously in order to control the rate of ventricular response. Finally AFL with AV conduction 1: 1 could be indicative of enhanced AV conduction.

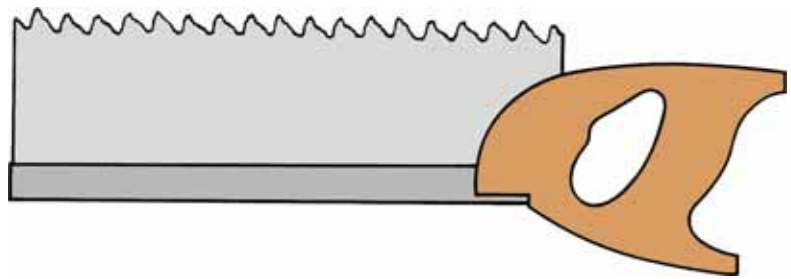
O flutter atrial com condução AV 1:1 (raro) é uma emergência médica. A freqüência ventricular está próxima de 300 bpm e deve ser tratado imediatamente. Condução AV 1:1 podem ser encontrados nas seguintes circunstâncias: pré-excitação tipo WPW, onde o estímulo é conduzido de forma anterógrada pela via anômala, flutter atrial secundário ao hipertireoidismo, flutter atrial do grupo pediátrico, e como conseqüência do uso prévio de antiarrítmicos da classe IA (procainamida, quinidina ou disopiramida), os quais, pela sua ação anti-colinérgica vagolítica excercida na junção AV, especialmente se as mencionadas drogas foram usadas sem administrar previamente dixogina, antagonistas do cálcio ou beta-bloqueadores a fim de controlar a taxa de resposta ventricular.
Finalmente flutter atrial com condução AV 1: 1 poderia ser conseqüência de condução AV acelerada.

AFL is the second most common tachyarrhythmia, after AF. Much has been learned about the mechanism of AFL in the past two decades and therefore how this arrhythmia is treated has changed.
Radiofrequency catheter ablation is now the long-term treatment of choice in patients with symptomatic AFL.
AFL has traditionally been characterized as a macro re-entrant arrhythmia with atrial rates between 240-450 beats per minute.
The ECG usually demonstrates a regular rhythm, with "F' waves that can appear sawtoothed, also called flutter waves or F waves.



The ECG is characterized by the typical atrial "F" waves with sawtooth or picket-fence appearance, frequently observed better in II, III, aVF and V_1, with median atrial HR of 240-350 bpm (the HR of atypical or type II flutter is 350 to 450 bpm), characteristic absence of isoelectric line between F waves, variable degrees of AV block or rarely, 1:1 conduction.





Absence of isoelectrical "Plateau" between F waves

Since the atrioventricular (AV) node cannot conduct at the same rate as the atrial activity, one commonly sees some form of conduction block, typically 2:1 or 4:1. Ventricular HR is usually half of the atrial HR (i.e., 150 beats/min). A ventricular rate significantly slower in absence of drugs, suggests normal AV conduction.
This block may also be variable and cause AFL to appear as an irregular rhythm.
AFL nearly always present, accompanied by organic substrate, which has as electrophysiological mechanism in most of the cases, a macro-reentry circuit in circle that covers the whole RA; more rarely by a unifocal or multifocal atrial focus with very high shock, or exceptionally by focal micro-reentry in the RA. There are always intra-atrial or inter-atrial dromotropic alterations, with a minimal extension being necessary in the movement of the circle, refractoriness dispersion and autonomous tone variations.

Pathophysiology: AFL is caused by a reentrant circuit that is confined to the right atrium (RA). The impulses travel through the atrial septum, then across the right atrium, then inferiorly through the RA free wall, and then back across through an isthmus bounded by the coronary sinus os and the tricuspid valve annulus. This isthmus is also called the atrial flutter isthmus and is the target for radiofrequency catheter ablation of atrial flutter. When the electric activity in this circuit moves in a counterclockwise direction, the atrial flutter is called typical AFL.. The ECG will demonstrate the classic negative sawtooth pattern in leads II, III, and aVF.

When the electric activity moves in a clockwise direction, the ECG will show positive flutter waves in leads II, III, and aVF, and may appear somewhat sinusoidal. This type of AFL is called reverse typical AFL.

Atrial flutter related to an incision made in the atria, as in congenital heart surgery, can also give rise to a re-entrant circuit. This causes incisional atrial re-entry.

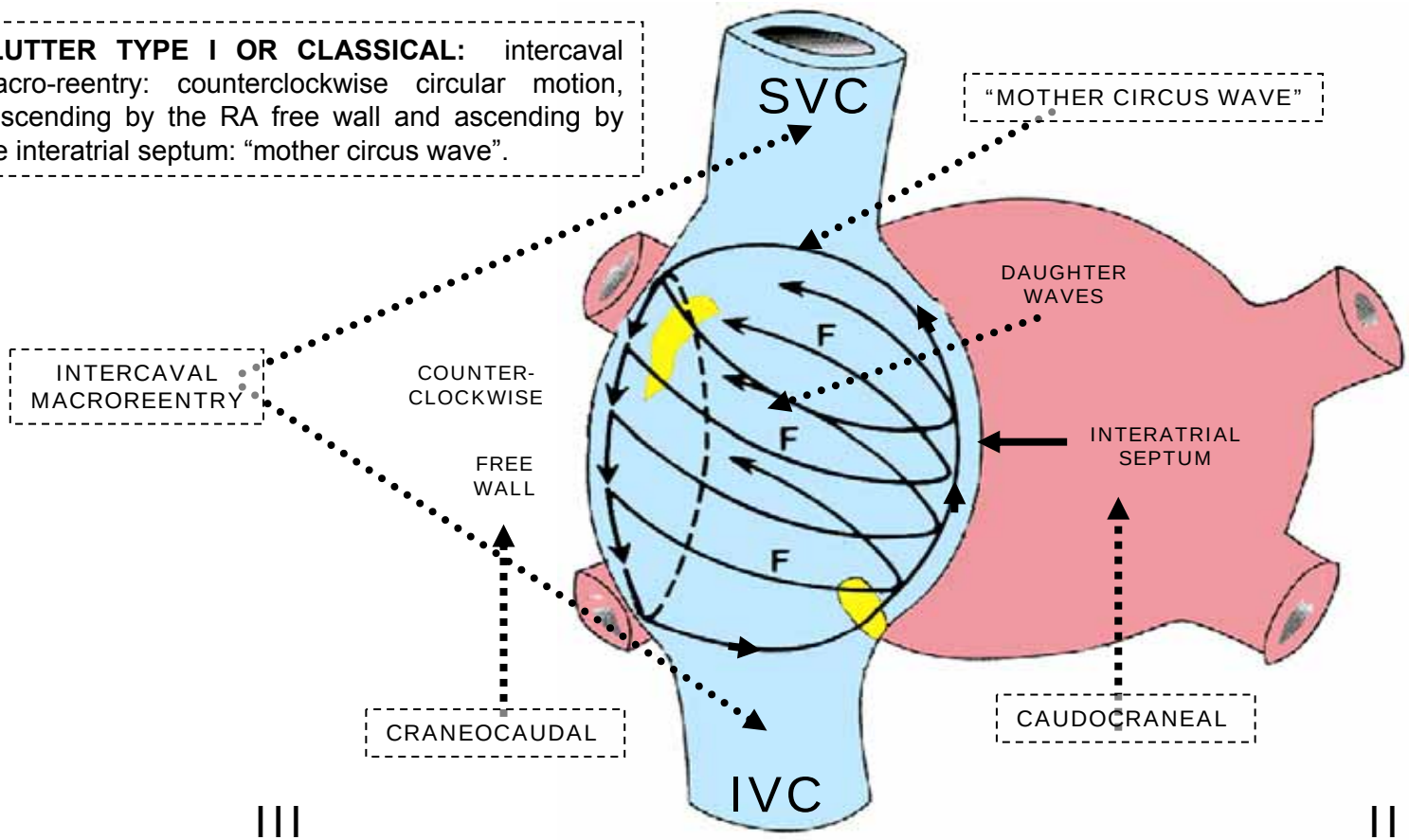
Atrial flutter has been reported in rare cases to arise from the left atrium.The above forms of atrial flutter are also called type I flutter. These are the most common forms.

Type II AFL, also known as atypical flutter, is still poorly characterized, but may result from an intra-atrial re-entrant circuit operating at a faster rate.

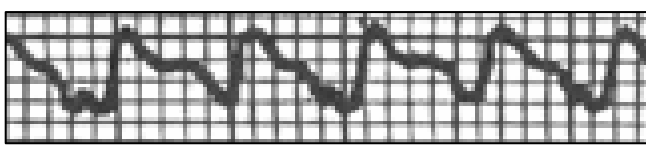
AFL is associated in patients with heart failure, valvular disease, chronic obstructive pulmonary disease, hyperthyroidism, pericarditis, pulmonary embolism, and a history of open heart surgery.

FLUTTER TYPE I, CLASSICAL OR COMMON

FLUTTER TYPE I OR CLASSICAL: intercaval macro-reentry: counterclockwise circular motion, descending by the RA free wall and ascending by the interatrial septum: “mother circus wave”.

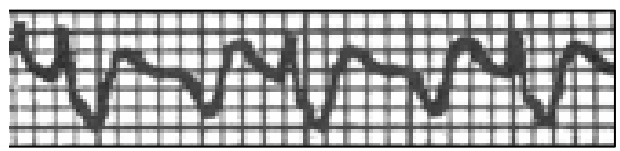


III



F waves of negative polarity in II, III and aVF without baseline

II

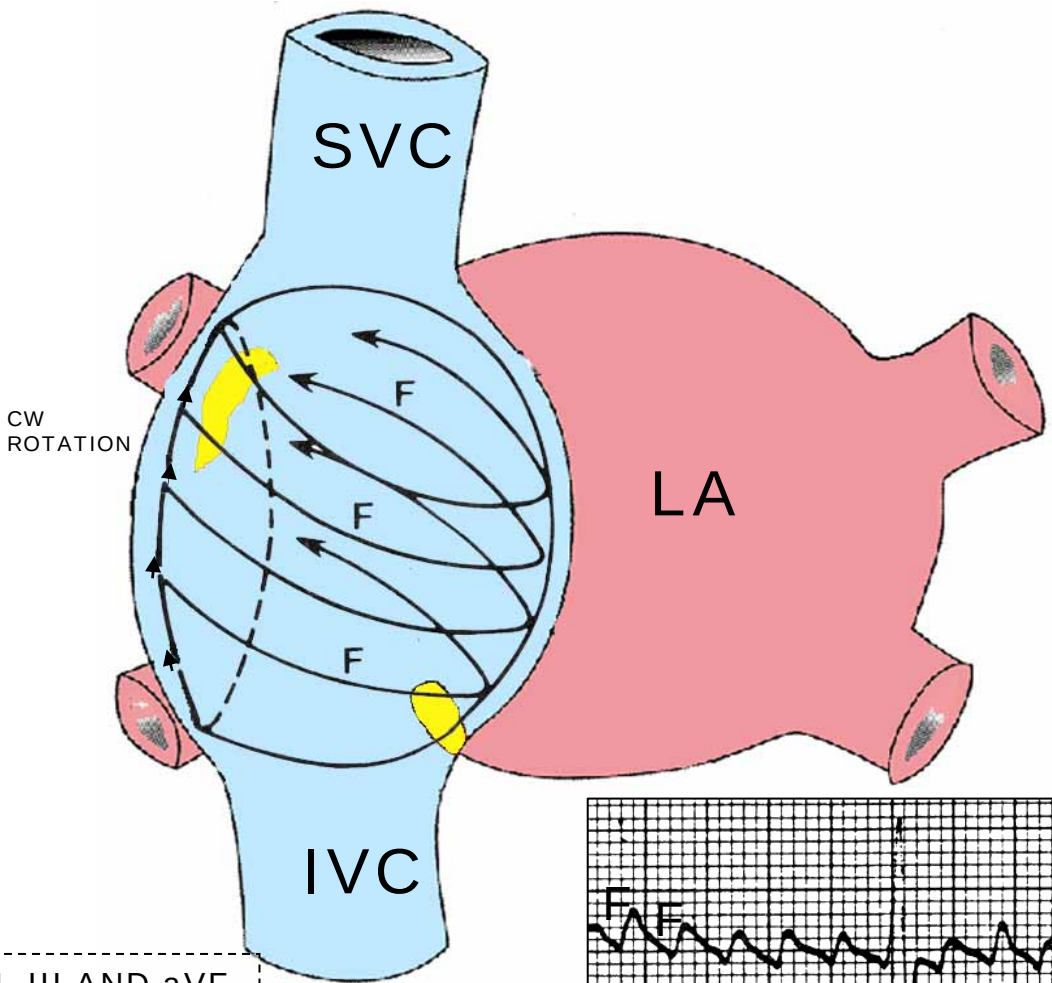


FLUTTER TYPE II OR ATYPICAL

TYPE II FLUTTER OTHER NAMES:

Atypical, rare, antidromic, type B, rapid, left atrial. Macro-reentry in the junction of the right appendage with the RA near the atrioventricular sulcus.

Clockwise circular motion descending by the septum and ascending by the RA free wall.



Atrial rate of 345 bpm

F WAVES OF POSITIVE POLARITY IN II, III AND aVF AND WITH A MUCH GREATER RATE WITHOUT BASELINE

Frequency

AFL affects approximately 88 out of 100,000 new patients each year. In the United States, this represents approximately 200,000 patients presenting with AFL annually. Not very frequent and less prevalent than AF: 10 to 1 in favor of the latter. It is estimated in 0.0006% to 0.004% between 50,000 patients in a General Hospital.

Mortality/Morbidity: For the most part, morbidity and mortality are due to complications of rate (ie, syncope, congestive heart failure (CHF). In patients who suffer from AFL, the risk of embolic occurrences approaches that of AF.

Gender: 2:1 male-to-female ratio.

Age: The prevalence of AF increases with age and varies from 1 case out of 200 persons for people younger than 60 years, to almost 9 cases out of 100 persons for people older than 80 years. Aged 25-35 years: 2-3 cases per 1000 people; Aged 55-64 years: 30-90 cases per 1000 people.; Aged 65-90 years: 50-90 cases per 1000 people.

Causes

Patients at highest risk for AFL include those with long-standing hypertension, valvular heart disease (rheumatic), left ventricular hypertrophy, coronary artery disease with or without depressed left ventricular function, pericarditis, pulmonary embolism, hyperthyroidism, and diabetes. Additionally, CHF for any reason is a noted contributor to this disorder.

Additional causes include the following: Postoperative revascularization, digitalis toxicity

Rare causes: Myotonic dystrophy in childhood[1], brugada syndrome[2]

1. **Suda K, Matsumura M, Hayashi Y. Myotonic dystrophy presenting as atrial flutter in childhood. *Cardiol Young*. Feb 2004;14(1):89-92.**
2. **Schimpf R, Giustetto C, Eckardt L, et al. Prevalence of supraventricular tachyarrhythmias in a cohort of 115 patients with Brugada syndrome. Ann Noninvasive Electrocardiol. 2008 Jul;13:266-169.**

1. Hypertensive heart disease
2. Coronary artery disease

Chronic

AMI: 03%

5 .3% of the cases.

3 COPD.
4 Valve Heart disease.
5 Severe aortic stenosis.
6 Postoperative revascularization
7 Bronchopneumonia.
8 Acute pulmonary embolism: transitory character.
9 Bronchogenic carcinoma.
10 Thyrotoxicosis: transitory. Frequent rate of ventricular response 1:1. Ventricular HR is 300 bpm.
11 Congenital heart diseases: E.g.: ASD not operated > 40 years old. Ebstein anomaly
12 Post-operative of heart surgery. E.g.: Mustard surgery for TGA correction.
13 Digitalis intoxication (exceptional)
14 As part of the “brady-tachy” syndrome.
15 Myocarditis.
16 Pericarditis.
17 As part of WPW syndrome (frequent AV 1:1 conduction with syncope and possible sudden death)
18 Mitral valve prolapse syndrome.
19 Alcoholism.
20 digitalis toxicity
21 Rare causes: Myotonic dystrophy in childhood1, brugada syndrome2

History

Symptomatic AFL is typically a manifestation of the rapid ventricular rate that decreases cardiac output.Palpitations, fatigue or poor exercise tolerance, mild dyspnea, presyncope. Less common symptoms include angina, profound dyspnea, or syncope. Thromboembolic events are possible with this arrhythmia.

Beware of the patient with a history of pre-excitation syndrome (Wolff-Parkinson-White), if they have a very short PR interval (<.115s) and no delta wave. Patients may experience 1:1 conduction of the flutter waves, and this can degenerate into ventricular fibrillation (VF).

AFL rhythm itself is unstable and usually reverts either to AF or sinus rhythm. It would be unusual for a patient to remain in stable chronic AFL.

Physical Examination

Pertinent physical findings are limited to cardiovascular system. If embolization has occurred from intermittent AFL, findings are related to brain and/or peripheral vascular involvement. Tachycardia may or may not be present, depending on the degree of AV block associated with the AFL activity. Cardiac rate, often approximately 150 beats per minute because of a 2:1 AV block (This is dependent on the atrial firing rate, which may be influenced by medications as well as intrinsic cardiac factors.)

Regular or slightly irregular heartbeat, as the AV block may be variable

Hypotension is possible, but normal blood pressure is observed more commonly.

Peripheral embolization may occur

CHF may be found, usually caused by left ventricle dysfunction.

Laboratory Studies

Obtain thyroid function studies.

Obtain serum electrolyte and digoxin levels if appropriate.

Obtain CBC if anemia is suspected or a history of recent or current blood loss is associated with presenting symptoms.

Consider obtaining blood gas measurements in patients with hypoxia or carbon monoxide intoxication. Also, seek a history of stimulant drug usage (eg, ginseng, cocaine, ephedra, methamphetamine).

Imaging Studies

Chest radiographic findings are usually normal in those with AFL. Look for radiographic evidence of pulmonary edema in subacute cases.

Other Tests

Electrocardiography (ECG)

Atrial rate during typical (ie, type I) AFL is usually 250-350 beats per minute, although class IA and IC antiarrhythmic drugs and amiodarone can reduce the rate to approximately 200 beats per minute. If this occurs, the ventricles can respond in a 1:1 fashion to the slower atrial rate.

Atrial rate ordinarily is about 300 beats per minute. In untreated patients, the ventricular rate is half the atrial rate (ie, 150 beats per minute). A significantly slower ventricular rate in the absence of drugs suggests abnormal AV conduction.

AFL can conduct to the ventricle in a 1:1 fashion, producing a ventricular rate of 300 beats per minute in children, in patients with the pre-excitation syndrome, in those whose AV nodes conduct rapidly, and occasionally in patients with hyperthyroidism.

The rate in atypical (ie, type II) flutter is 340-433 beats per minute. Re-entry within the right atrial (tricuspid annulus) is responsible for most cases of AFL.

In cases of typical AFL, ECG reveals identically recurring regular sawtooth flutter waves (see the image below) and evidence of continual electrical activity.

Flutter waves are often visualized best in leads II, III, aVF, or V1. The flutter waves for typical (type I) AFL are inverted (negative) in these leads because of a counterclockwise re-entrant pathway. Sometimes, they are upright (positive) when the re-entrant loop is clockwise. Flutter waves (particularly 2:1) can deform the ST complex in such a manner as to mimic an ischemic injury pattern on the 12-lead ECG.

	TYPE I	TYPE II
HR	240 to 339 bpm	Faster 340 to 433 bpm.
MECHANISM	Intercaval macro-reentry, CCWR, descending by the RA free wall and ascending by the interatrial septum: "mother circus wave".	Macro-reentry in the junction of the right appendage with the RA near the atrioventricular sulcus. Clockwise circular motion descending by the septum and ascending by the RA free wall.
F WAVES POLARITY	Inverted in inferior leads	Upright in II, III, and aVF
RESPONSE TO TRANSESOPHAGEAL ATRIAL PACING	Good	Null

Flutter and AF eventually coexist with alternating patterns (ie, fib-flutter, flitter) in the same tracing. Waves with sawtooth or picket fence appearance called F waves, with a heart rate between 250 and 350 bpm, observed better in the inferior wall and V1 with slowly descending and rapidly ascending ramp. These waves resemble an inverted P wave, followed by an ascending ramp: "Tp" waves.

Characteristics of atrial activation.
Absence of isoelectrical “plateau” between F waves.
AV conduction: ventricular rate depends on AV nodal conduction; more frequently between 150 bpm and 220 bpm.
Intraventricular conduction that determines QRS complex duration. When it is conducted with aberrance, QRS is prolonged.
In the common form, the polarity of F waves is negative in inferior leads II, III and aVF:caudo cephalic atrial activation: type I, common or classical flutter.
The II or V1 leads should be recorded separately, in prolonged tracings (from 15s to 20s) to establish the relationship between F waves and QRS complexes.
During AFL with 2:1 conduction, the ‘’F’’ waves may be masked in the II leads and be very visible in V_1.

CAUSES OF F WAVES OF LOW RATE FLUTTER

Great mega-atria show atrial rate sometimes lower than 200 bpm.
Class IA drugs: drugs of intermediate kinetics (Quinidine, procainamide, disopyramide and ajmaline) reduce Vmax. and extend action potential.
Cass IC: they are drugs of slow kinetics (Propafenone, Flecainide, Encainide, Moricizine and Lorcainide).
Class III drugs: K+ channel block (prolongation of action potential in phase 3) (Amiodarone).

CAUSES OF F WAVES OF FLUTTER OF HIGH RATE

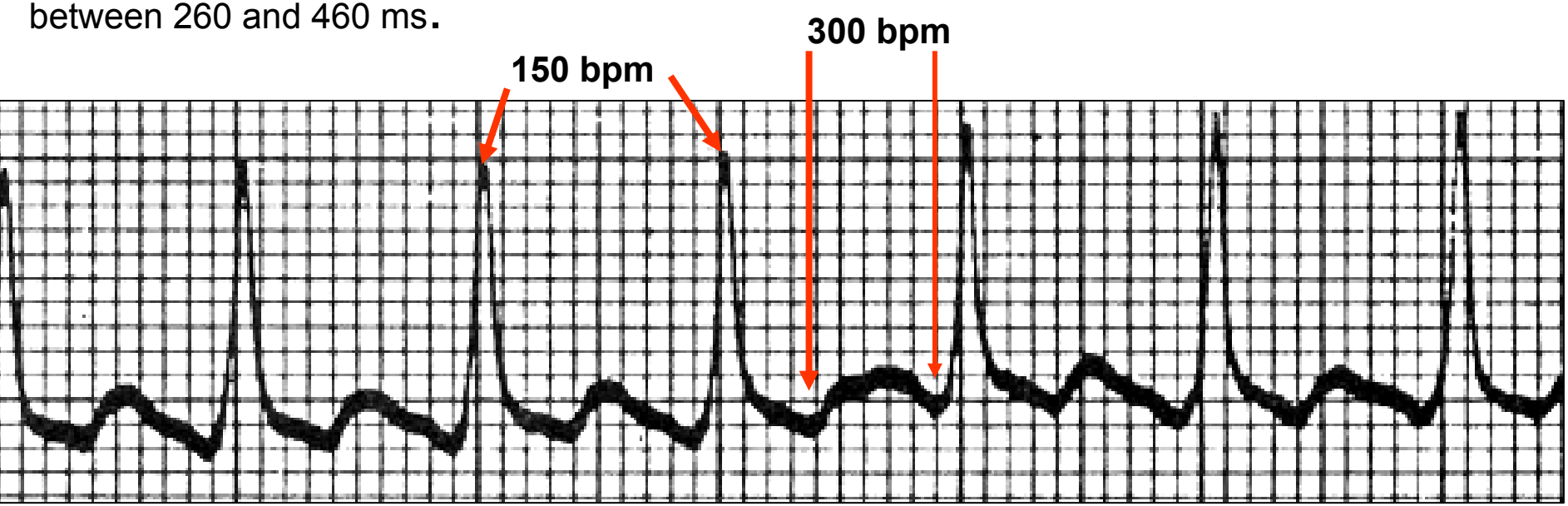
Flutter in children: median HR of 300 bpm with 1:1 response.
Flutter of ventricular pre-excitation.
Type II flutter of Wells: rate of F between 340 and 433 bpm: it cannot be interrupted with pacing.

TYPES OF AV CONDUCTION

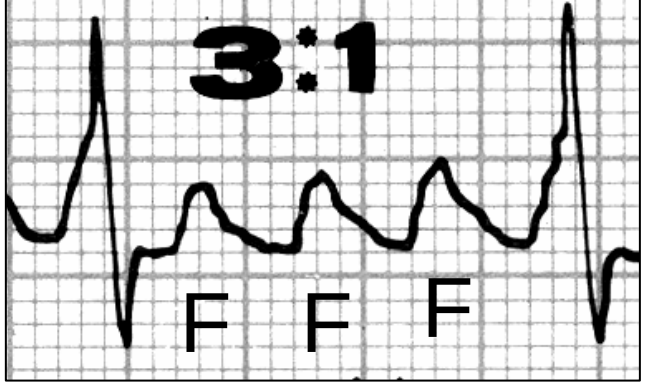
REGULAR:

1:1 – suggests ventricular preexcitation, pediatric, hyperthyroidism, enhanced AV

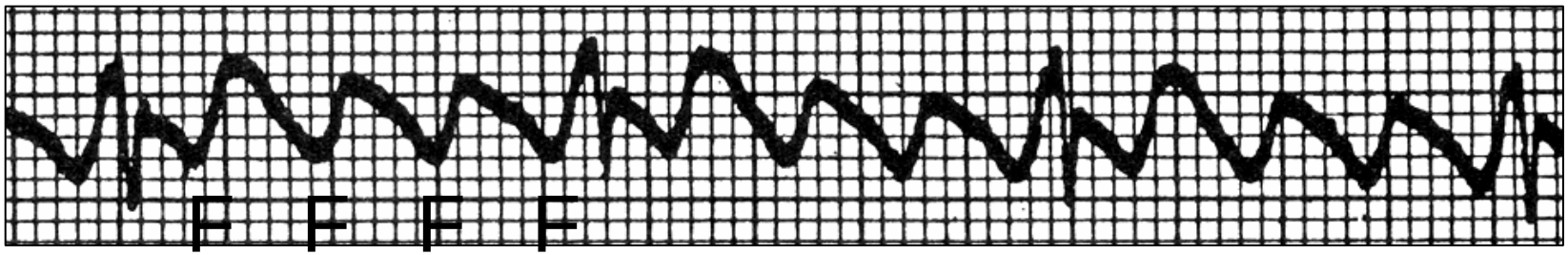
2:1 The most frequent ratio in non-treated patients is 2:1 with atrial and ventricular rate of 300/150 bpm respectively; this ratio is due to the physiological interference in the junction. If the ventricular response rate is regular and constant (e.g.: always 2:1) the F-R interval will be too, varying between 260 and 460 ms.



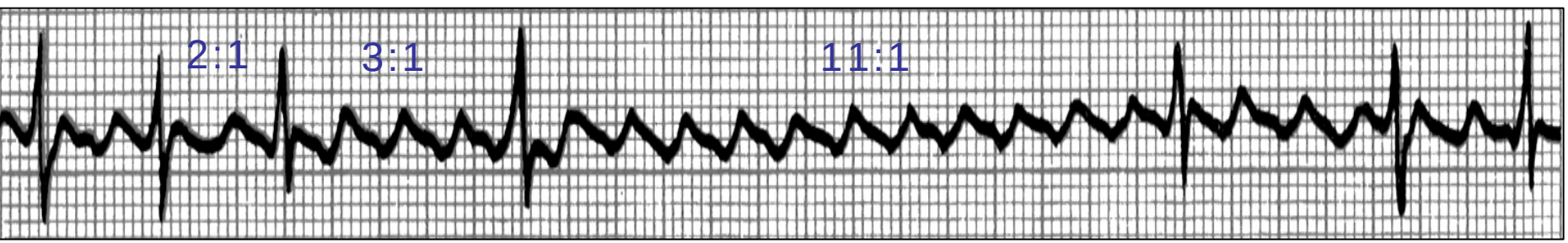
3:1



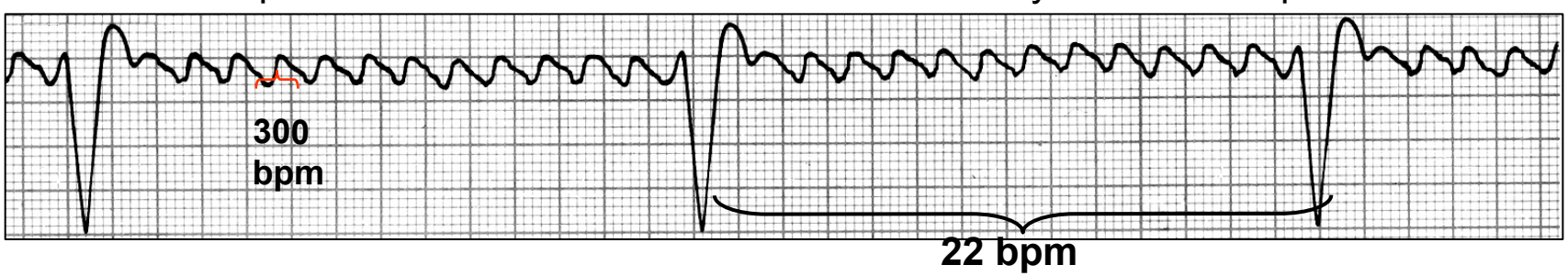
4:1



IRREGULAR



ABSENT: with complete AV block – the ventricular rate is usually low and independent from atrial rate.



Transthoracic echocardiography
A transthoracic echocardiogram is the preferred modality for evaluating AFL. It can evaluate right and left atrial size, as well as the size and function of the right and left ventricles, which assists in diagnosing valvular heart disease, left ventricular hypertrophy (LVH), and pericardial disease. Transthoracic echocardiogram has low sensitivity for intra-atrial thrombi.

Exercise testing
Exercise testing can be utilized to identify exercise-induced AF and to evaluate ischemic heart disease.

Holter monitoring
A Holter monitor can be used to help identify arrhythmias in patients with nonspecific symptoms, identify triggers, and detect associated atrial arrhythmias.
Management

Prehospital Care
In general, avoiding class I and III agents (eg, procainamide) in the prehospital setting is safest because of possible induction of 1:1 conduction. Generally, the rate can be slowed safely with calcium channel blockers or beta-adrenergic blockers.

Emergency Department Care Assess airway, breathing, and circulation. Hemodynamic concerns will dictate initial treatment.

Treatment options for AFL include the following:

Antiarrhythmic drugs/nodal agents

Direct-current (DC) cardioversion: AFL is considerably more sensitive to electrical CD cardioversion than AF, and usually requires a lower energy shock. 20-50J is commonly enough to revert to sinus rhythm. Conversely, it is relatively resistant to chemical cardioversion, and often deteriorates into AF prior to spontaneous return to sinus rhythm**.** Cardioversion for unstable patients If the patient is unstable (eg, hypotension, poor perfusion), synchronous direct-current DC is commonly the initial treatment of choice. CV may be successful with energies as low as 25 Joules, but since 100 Joules is virtually always successful, this may be a reasonable initial shock strength. If the electrical shock results in AF, a second shock at a higher energy level is used to restore normal sinus rhythm.

Rapid atrial pacing to terminate AFL

Blood pressure can be supported and rate controlled with medication.

Look for underlying causes. At times, treatment of the underlying disorder (eg, thyroid disease, valvular heart disease) is necessary to effect conversion to sinus rhythm.

In a study by Stiell et al[1], the Ottawa Aggressive Protocol, which includes chemical cardioversion, electrical cardioversion, if needed, and discharge home from the ED, was found safe, rapid, and effective, and may reduce hospital admission; further study of this protocol is needed.

Consultations: Most cases will require internal medicine or cardiology consultation. A cardiologist may become involved, primarily if the patient presents with complicating factors or an obvious ongoing cardiac ischemia (or infarction) not treatable with rate reduction measures and standard chest pain protocols.

1. **Stiell IG, Clement CM, Perry JJ, Vaillancourt C, Symington C, Dickinson G, et al. Association of the Ottawa Aggressive Protocol with rapid discharge of emergency department patients with recent-onset atrial fibrillation or flutter. *CJEM*. May 2010;12(3):181-91.**

Medication Rate control with nodal agents: Rate control is the goal of medication in AFL or AFTo slow the ventricular response, verapamil or diltiazem may be the appropriate initial treatment. Adenosine produces transient AV block and can be used to reveal flutter waves. These drugs generally do not convert AFL to normal sinus rhythm. If the flutter cannot be cardioverted, terminated by pacing, or slowed by the drugs mentioned above, digoxin can be administered alone or with either a calcium antagonist or a beta-blocker. Intravenous amiodarone has been shown to slow the ventricular rate and is considered as effective as digoxin. Digoxin toxicity is very rarely a cause of flutter; however, ascertaining that flutter is not caused by digoxin toxicity is important. Another caveat is to beware of the vagolytic action of quinidine, procainamide, and disopyramide if used to slow the flutter rate. These drugs can effect AV conduction, resulting in a 1:1 ventricular response to the AFL. Before administration of these drugs, be sure to slow the conduction rate with digoxin or calcium channel blockers.β-adrenergic blockers are especially effective in the presence of thyrotoxicosis and increased sympathetic tone. Other antiarrhythmic drugs that can terminate AFL /AF include procainamide, disopyramide, propafenone, sotalol, flecainide, amiodarone, and ibutilide.

Dronedarone was approved by the US Food and Drug Administration on July 2, 2009. It is a deiodinated derivative of amiodarone that has no organ toxicity. Its use will likely extend to both atrial and ventricular arrhythmias. Dronedarone has multiple actions (all 4 Von Williams class effects). Unlike amiodarone, it does not have the iodine moiety. The lack of iodination may offer a better side-effect profile. Dronedarone has been shown to (1) have antiadrenergic effects, (2) prolong atrial and ventricular refractory periods, and (3) prolong AV node conduction as well as the paced QRS complex. In animal models, dronedarone has been shown to decrease ischemia-induced ventricular arrhythmias. Antiarrhythmic drugs alone control AFL in only 50-60% of patients.

Radiofrequency Catheter Ablation (RFCA)

Because of the reentrant nature of AFL, it is often possible to ablate the circuit that causes AFL. This is done in the electrophysiology lab by causing a ridge of scar tissue that crosses the path of the circuit that causes AFL.

RFCA of the isthmus, as discussed above, is a common treatment for typical AFL. RFCA has become the first line of therapy for AFL. Advances in catheter and mapping technologies have led to better understanding and different approaches for treating this arrhythmia. Since the early 1990s, RFCA has been used to interrupt the re-entrant circuit in the right atrium and prevent recurrences of AFL.

RFCA is immediately successful in more than 90% of cases and avoids the long-term toxicity observed with antiarrhythmic drugs. Cavotricuspid isthmus ablation using a nonfluoroscopic thre-dimensional (3D) navigation system is effective and safe[1].

Procedure and fluoroscopic times were shorter with the use of 10mm-tip as compared with the others techniques. The long-term risk of recurrence was lower when we used the 10mm-tip catheter and the survival free of a second procedure was higher among patients treated with this catheter[2]. On long-term follow-up, after RFCAof isolated typical AFL, more than three-quarters of patients remained free from AF. Conversely, in patients with preablation AF, typical AFL ablation reduced the number of AF episodes as well as the number of hospitalizations and arrhythmia-related symptoms3.

1. Alvarez M, Tercedor L, Herrera N, et al Cavotricuspid Isthmus Catheter Ablation Without the Use of Fluoroscopy as a First-Line Treatment. J Cardiovasc Electrophysiol. 2010 Nov 29. doi: 10.1111/j.1540-8167.2010.01962.x
2. Leiria TL, Becker G, Kus T, et al. Improved Flutter Ablation Outcomes Using a 10mm-tip Ablation Catheter. Indian Pacing Electrophysiol J. 2010 Dec 26;10:496-502.
3. Bandini A, Golia P, Caroli E, et al. Atrial fibrillation after typical atrial flutter ablation: a long-term follow-up. J Cardiovasc Med (Hagerstown). 2010 Oct 30. [Epub ahead of print]

Calcium channel blockers

These agents reduce the rate of AV nodal conduction and control ventricular response. Formulations administered IV are discussed only as they relate to the control of severe symptoms (eg, rapid ventricular rate in emergent situations).

Diltiazem (Cardizem): DOC during depolarization. Inhibits calcium ion from entering slow channels or voltage-sensitive areas of vascular smooth muscle and myocardium.

AdultInitial dose: 0.25 mg/kg IV over 2 min as bolus; repeat at 0.35 mg/kg if inadequate rate reduction after 15 minMaintenance dose: 5-10 mg/h (up to 15 mg/h) IV can be infused for up to 24 h.***Pediatric***Not established

Verapamil Second DOC. Can diminish PVCs associated with perfusion therapy and decrease risk of ventricular fibrillation and ventricular tachycardia. By interrupting re-entry at AV node, can restore normal sinus rhythm (NSR) in patients with paroxysmal supraventricular tachycardias (PSVT).During depolarization, inhibits calcium ion from entering slow channels or voltage-sensitive areas of vascular smooth muscle and myocardium.

Adult: 2.5-5 mg IV bolus initially reduces ventricular rate within 5 min; can be repeated to total of 15 mg IV, follow by maintenance infusion of 0.05-0.2 mg/min

Pediatric

<1 years: 0.1-0.2 mg/kg IV bolus over at least 2 min under continuous ECG monitoring; usual single-dose range 0.75-2 mg

>1 years: 0.1-0.3 mg/kg IV bolus over at least 2 min; usual single-dose range 2-5 mg; not to exceed 5 mg

β-blockers: These agents slow the sinus rate and decrease AV nodal conduction. β-blockers now have more of a secondary role in rate control in AFL/AF. Be sure to monitor blood pressure carefully. **Metoprolol:** Selective β1-adrenergic receptor blocker that decreases automaticity of contractions. ***Adult*** 5-15 mg IV over 5-15 min in 5-mg increments

Pediatric: Not established