Hombre en la tercera edad con taquiarritmia sostenida de QRS variable y estrecho

Elderly man with sustained tachyarrhythmia of variable Electrocardiographic Pattern and narrow QRS

68 yr old, white man, born in Paraíba, living in Fortaleza-CE, Brazil; married, painter.

The patient said to feel burning retrosternal pain, with no irradiation, accompanied by cold sweating, general discomfort and quick, regular and prolonged palpitations, starting on the afternoon of Nov 10, 2013. He denies experiencing dyspnea.

He mentioned a similar but less intense episode, 3 months ago.

BP=140/90

Medications in use: Valproic acid 500 mg 2 x day due to simple absence seizures, Salicylic Acid 100mg/d and Atenolol 50mg/d

He was medicated with IV amiodarone

Questions
1. What is the mechanism of the tachycardia?
2. What happened after amiodarone?
3. What to do now?

Warm regards,

Raimundo **Barbosa- Barros M.D.**

68 años , hombre, blanco, nacido en Paraíba, residente en Fortaleza-CE, Brasil, casado, pintor.

Paciente refiriendo dolor retroesternal en quemazón, sin irradiación, acompañado de sudoración fria, malestar general y palpitaciones rápidas regulares y prolongada comenzada en la tarde del día 10.11.13. Niega disnea.

Refiere un episodio similar de menor intensidad, hace 3 meses.

PA=140/90

Medicamentos en uso:

Acido valpróico 500 mg 2 x día por cuadro de quadros de ausência simples

AAS 100mg/d

Atenolol 50mg/d

Fue medicado con amiodarona EV

Preguntas

1. ¿Cuál es el mecanismo de la taquicardia?
2. ¿Qué pasó después de amiodarona?
3. ¿Qué hacer ahora?

Un abrazo

Raimundo Barbosa- Barros

Admisión -1   **Admission-1**
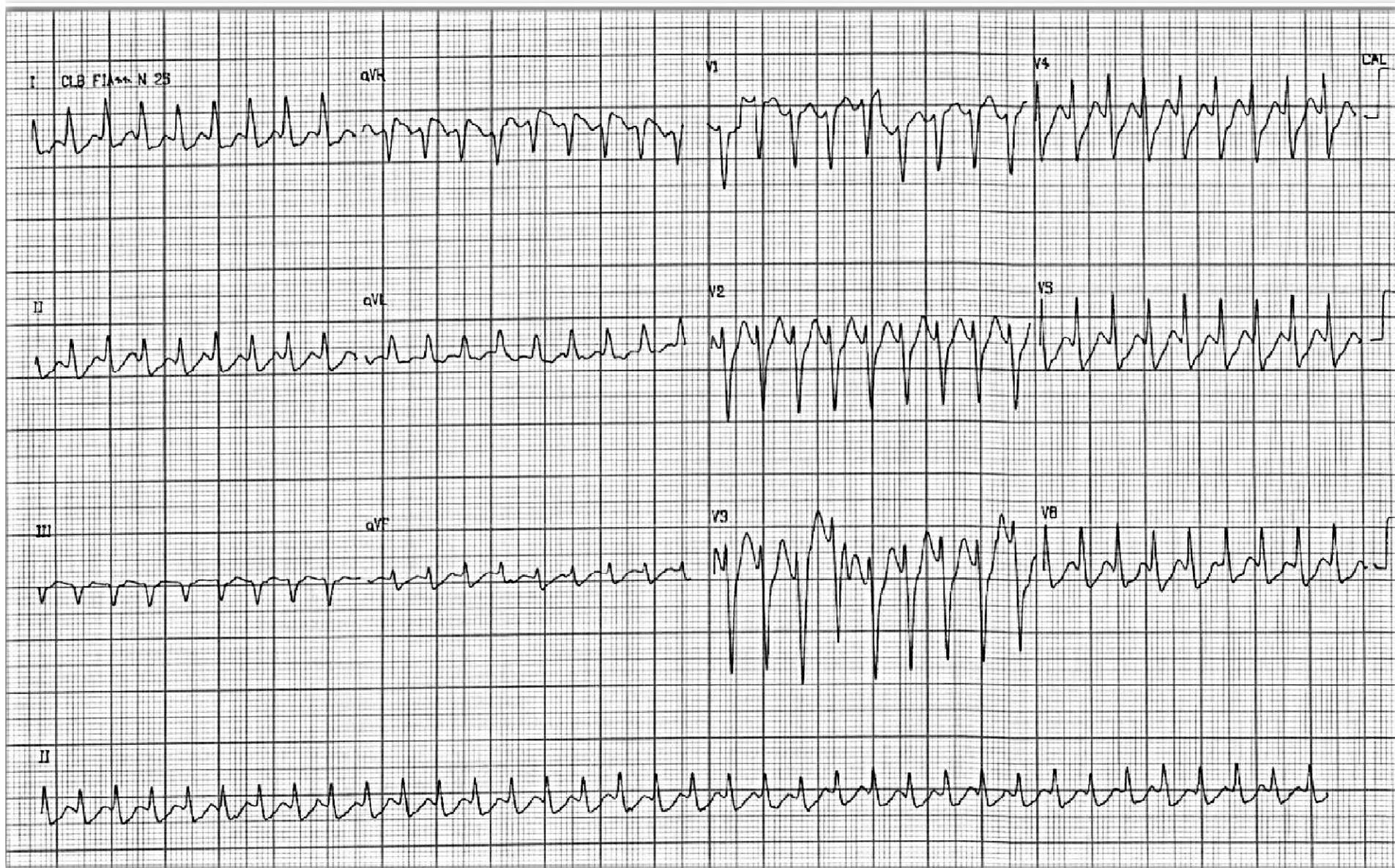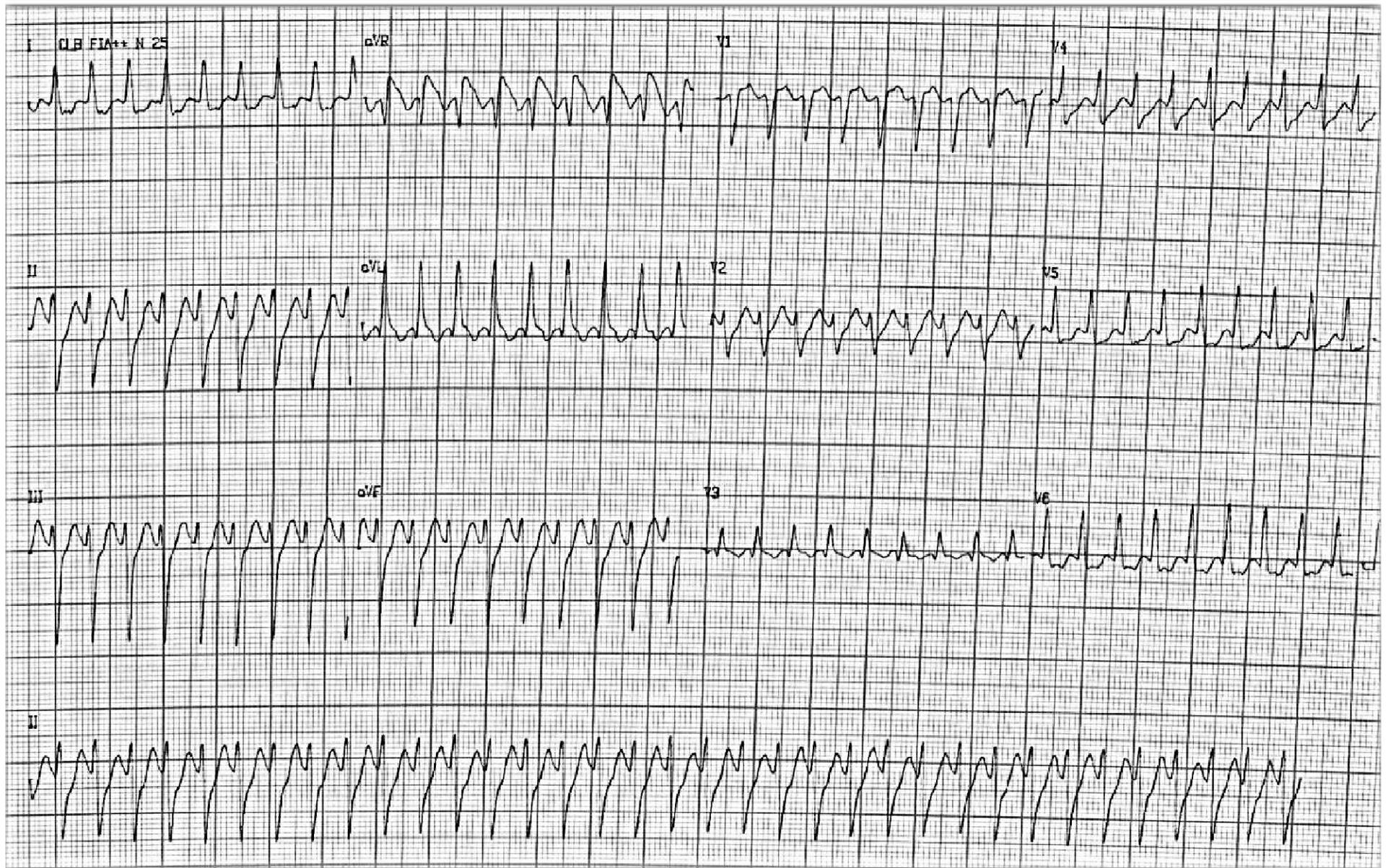
30 minutos después de la amiodarona/ 30 minutes after amiodarone
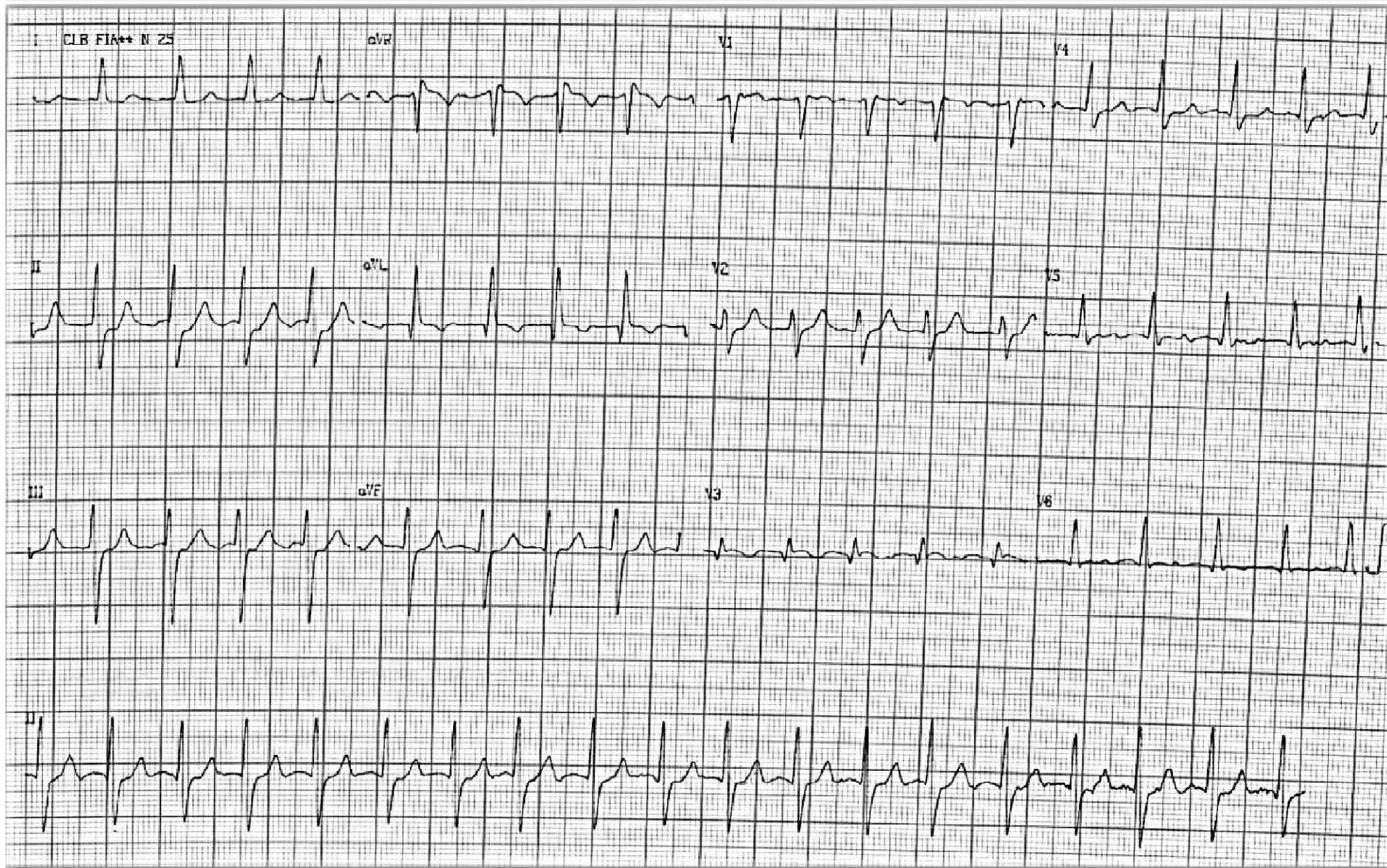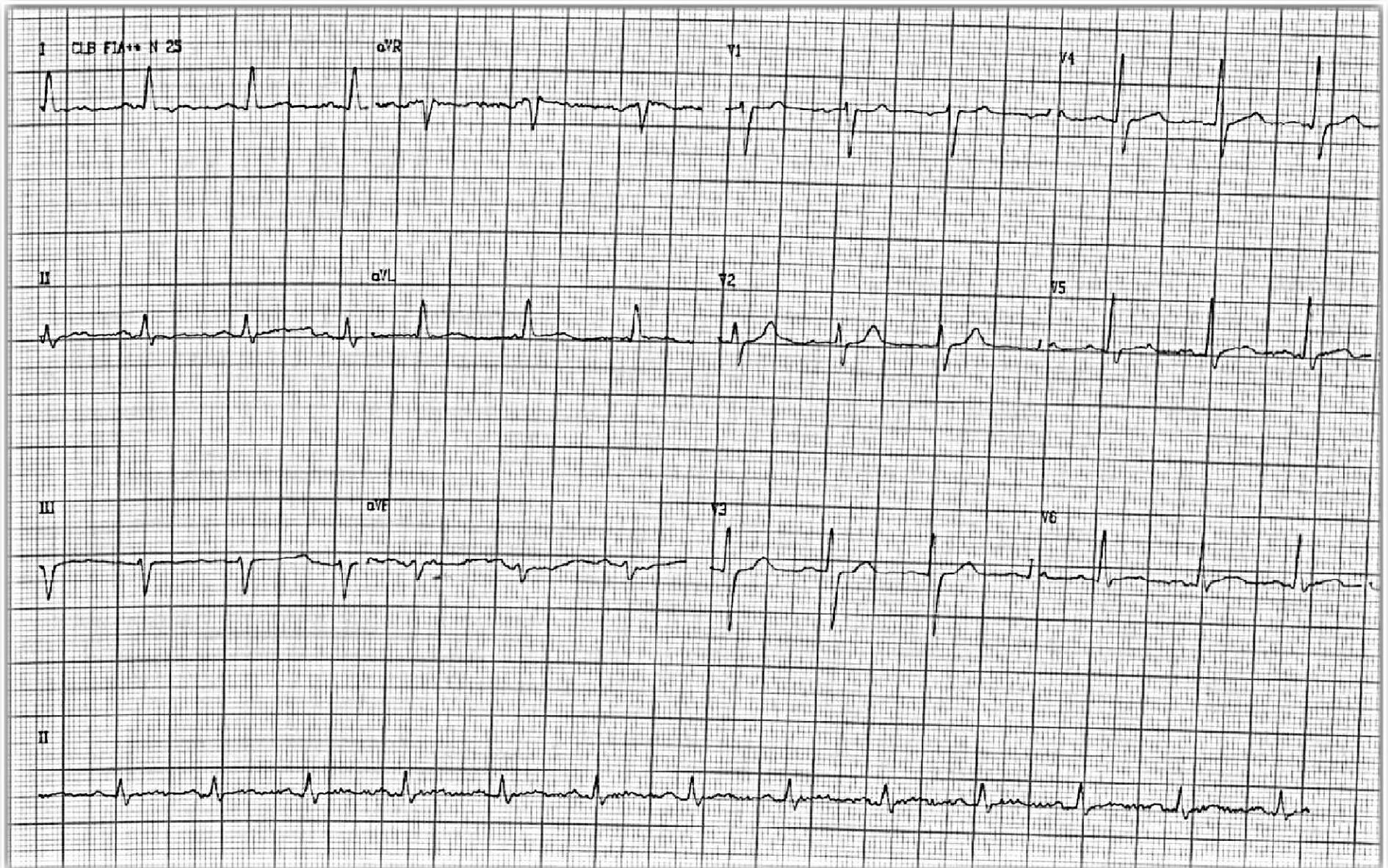
Colleagues opinions

Hi

Thanks for sharing the case.

ECG#1: Narrow complex and regular tachycardia. Very difficult to see P waves: Differential diagnosis include: Atrial tachycardia, Typical AVNRT and Orthodromic AVNRT (the 3 more frequent diagnosis).

ECG#2: It seems the same tachycardia with left anterior fascicular block aberrancy

ECG#3: After amiodarone, it seems atrial tachycardia with 2:1 conduction

One other possibility is a dual tachycardia with one of the 2 first ECGs being VT rather than atrial tachycardia (involving the posterior fascicle).

Management: full EP study with both atrial and ventricular stimulation. Ablation of the source if AT/

Best wishes

Dr Baranchuk, Adrian MD FACC

Assistant Professor of Medicine Cardiac Electrophysiology and Pacing

Director, EP Training Program Kingston General Hospital FAPC 3, 76 Stuart Street K7L 2V7, Kingston ON Queen's University Ph: 613 549 6666 ext 3801

Fax: 613 548 1387 barancha@kgh.kari.net

ECG #1 shows rapid narrow complex tachycardia with short RP configuration and negative P wave in aVL.

ECG # 2 shows LAFB? Amiodarone effect with NCT and P wave negative in Leads I and V6 at the same rate.

ECG #3 shows atrial fibrillation at a slow rate again probable amiodarone effect. Possibilities Orthodromic AVRT* over left sided AP or A.T. from the LA.

* impulses traveling in an anterograde manner through the AV node and in a retrograde manner through the accessory pathway; this is called orthodromic AVRT.

Melvin M. Scheinman, Professor of Medicine:
Department of Cardiac Electrophysiology, University of California San Francisco, San Francisco, California, USA.
Address:
UCSF
Electrophysiology Service
500 Parnassus Avenue
San Francisco, CA 94143-1354
Telephone/FAX/E-mail:
Phone: (415) 476-5706
Fax: (415) 476-6260
email: **scheinman@medicine.ucsf.edu**

Hi everybody
1. **ECG-1 first tracing** Looks like atrial tachycardia with 1/1 AV conduction at a rate of around 250 bpm and ascending ST depression.
2. **ECG-2 second tracing:** is the same except for a left anterior fascicular block.
3. **ECG-3 third tracing** after amiodarone: the AV conduction is now 2/1 with a ventricular rate of around 130 bpm

It does not looks like an typical atrial flutter.

Management

Best solution, after exclusion of thrombus in the left appendage, is to investigate the tachycardia and the circuit/focus location.

Other possibility: waiting some hours that amiodarone stopped the tachycardia or performed DC shock or esophageal overdrive.

Then ruling out underlying significant coronary stenosis.

Philippe Maury M.D.
Department of Cardiology, University Hospital Rangueil, 31059 Cedex 09 Toulouse, France.
Tel.: +33 (0) 5 61 32 30
mauryjphil@hotmail.com,

Spanish

Trazado 1: Flutter auricular con conducción AV 1:1
Trazado 2 Flutter auricular con conducción AV 1:1 + posiblemente de origen isquémico
Trazado 3 Reentrada nodal
Le daria adenosina IV

Marcelo Chambo

Trace 1: atrial flutter with 1:1 AV conduction
Trace 2 atrial flutter with 1:1 AV conduction + possibly ischemic in orirgin
Trace 3: Nodal Reentry
I would give adenosine IV
Marcelo Chambo.

Flutter con conducción 1:1
Flutter con conducción 2:1

Marigel

La suerte favorece sólo a la mente preparada. Isaac Asimov

Spanish

Estimado Raimundo y Potro:

1. TPSV por reentrada intranodal. Observo ondaP en DI, aVL y V1. El eje electrico ligeramente desviado a la izquierda. Luego del comienzo de la infusión de amiodarona, no afecta el ciclo de la taquicardia se desvía el eje eléctrico a la izquierda, con r inicial y cambio en la transición precordial en V3, bien podría deberse a fenómeno de fatiga de de la división anterosuperior. No me impresiona la taquicardia se expresión de isquemia miocardica.

2. En el tercer electrocardiograma aún continua con el ciclo de la taquicardia, pero solo han transcurrido 30 minutos de la infusión de amiodarona. Si se encuentra compensado aguardaría la amiodarona se distribuya y se corta la taquicardia. Si se encuentra descompensado o sintomático, o retorna taquicardia reentrante de alta frecuencia, infusión de adenosina y por ultimo cardioversión eléctrica.

Le colocaría un acceso central y tendría un marcapasos transitorio en la cabecera del paciente, recibe atenolol, infusión de amiodarona me impresiona que cuando revierta la taquicardia, padecerá de bradicardia extrema y siendo una complicación esperable por la terapéutica, preveer que pueda necesitar la colocación de urgencia de un marcapasos transitotio.

Un cordial saludo.

Martín Ibarrola

Dear Raimundo and Potro,

•PSVT by intranodal reentry. I see P wave in DI, aVL and V1. The electrical axis slightly shifted to the left. After the start of amiodarone infusion, it does not affect the tachycardia cycle, the electrical axis is shifted to the left, with initial r and change in precordial transition in V3, which could very well be due to fatigue phenomenon of the anterosuperior fascicle. It doesn't seem that the tachycardia is the expression of myocardial ischemia.

•In the third electrocardiogram, the tachycardia cycle still continues, but only 30 minutes elapsed since the amiodarone infusion. If the patient is compensated, I would wait for the amiodarone to spread and the tachycardia to end. If the patient is decompensated or symptomatic, or reentrant tachycardia of a high rate returns, adenosine infusion and finally, electrical cardioversion.

I would place a central access and I would place a transient pacemaker in the headboard of the patient. He receives atenolol, amiodarone infusion; it seems to me when the tachycardia reverts, he will suffer extreme bradycardia and with this being an expected complication by the therapy, you should foresee the need to place an emergency transient pacemaker.

Kind regards,

Martín Ibarrola

hola a todos los colegas del foro: en este caso opino que es una taquicardia por reentrada intranodal aunque no descartaria que fuera un fluter con conduccion 1:1. De todas formas me inclino por la primera.

Conducta de ser posible haria estudio electrofisiologico con intención de ablación. En caso de no ser posible como el atenolol no lo protegio me inclinaria por verapamil via oral aunque se pudiera valororar una droga 1c como la propafenona intuyendo (desde luego despues de complemantarios al respecto) que no tenga cardiopatia isquémica.

 Un saludo afectuoso y a esperar por los que de verdad saben.

Yamir from Cuba

English

Hello to all the colleagues in the forum,

In this case, it is my opinion that this is intranodal reentrant tachycardia, although I would not rule out a 1:1 conduction flutter. Anyway, I lean toward the first option.

If possible, the management should be electrophysiology study with ablation intention. If not possible, since atenolol did not protect the patient, I would lean toward oral verapamil, although a 1c drug could be considered, such as propafenone, with the intuition (of course, after supplementary studies about it) that there is no ischemic heart disease.

Warm regards and let's wait for those who really know.

Yamir - Cuba

Analisis del caso del Dr Raimundo Barbosa Barros y del Profesor Andrés Pérez -Riera M.D.Ph.D. Me parece que es una taquicardia por reentrada nodal "AV nodal reentrant tachycardia" (AVNRT). El tercer ECG muestra un bloqueo fascicular anterosuperior izquierdo dependiente de la taquicardia prolongada, o por la administración de amiadarona.El ECG de base sugiere una hipertrofia o isquemia basal del ventrículo izquierdo expresado por una onda T negativa en aVL y TIII.>TI en un corazón horizontal con una ligera desviación de eje frontal a la izquierda.Como tratamiento específico empezaria con verapamil 5 mm en 10cc glucosa 5% , 2 cc cada minuto y constante control del ritmo A los 3 minutos la arritmia habitualmente desapareceLa amiodarona lleva mas tiempo y no es especifica . Esta droga es mas especifica para las las taquicardias de reentrada extranodales "*AV reentrant tachycardia*"Por que yo recomiendo este tratamiento ? Porque un bolus de 5 mg de verapamil o adenosina, provocan bloqueos muy prologados de la parte superior de nodulo auriculo-ventricular acompaniadas con arritmias ventriculares que provocan palpitaciones y angustia al medico tratante ,y bastante peligroso cuando el paciente es un enfermo cardiaco o una jovencita con hypotension. Con el protocolo que yo recomiendo no hay ninguna complicacion y ningun transtorno arritmico despues de la reversion y tampoco descenso de la presion periferica ,y si ocurre se puede imediatamente suspender el tratamiento con poca droga inyectadaEsta es mi experiencia personal y no pase' mas angustias con este problema de taquicardias rapidas de este tipo

Que hacer despues del tratamiento / Si es la primera vez no hacer nada y se repite ablacion esta muy indicada un fraternal abrazo Samuel Sclarovsky - Israel

Englsih

Analysis of the case by Dr. Raimundo Barbosa Barros and Professor Andrés Pérez-Riera, M.D., Ph.D.: Dear friends of the forum, It seems to me this is AV nodal reentrant tachycardia (AVNRT).

The third ECG shows left anterior fascicular block dependent on prolonged tachycardia, or the administration of amiodarone.

Baseline ECG suggests basal hypertrophy or ischemia in the LV, expressed by negative T wave in aVL and TIII>TI in a horizontal heart with mild shift of the frontal axis to the left.

As specific treatment, I would start with verapamil 5 mm in glucose 10 cc 5%, 2cc per minute and constant rhythm control. After 3 minutes, the arrhythmia usually disappears.

Amiodarone requires more time and is not specific. This drug is more specific for AV reentrant tachycardias.

Why do I recommend this treatment? Because 5 mg of verapamil or adenosine in bolus cause very prolonged blocks in the upper part of the atrioventricular node, accompanied by ventricular arrhythmias that cause palpitations and distress to the attending physician, and quite dangerous when the patient is a cardiac patient or a young lady with hypotension. With the protocol I recommend, there is no complication and arrhythmic disorder after reversion and neither peripheral pressure decrease; if it happens the treatment can be interrupted immediately with a little drug injected.

This is my personal experience and I was no longer distressed with this rapid tachycardias problem.

What to do after the treatment? If it is the first time, do nothing, and repeat ablation is quite indicated.

Warm regards,

Samuel Sclarovsky

Final conclusions

ECG-1 Admission-1 : Narrow-complex QRS Paroxysmal Supraventricular Tachycardia(PSVT) regular rapid (heart rate 214 bpm), P' is separate of the QRS complex in aVL lead: Ortodromic Circus Movemet Tachycardia.
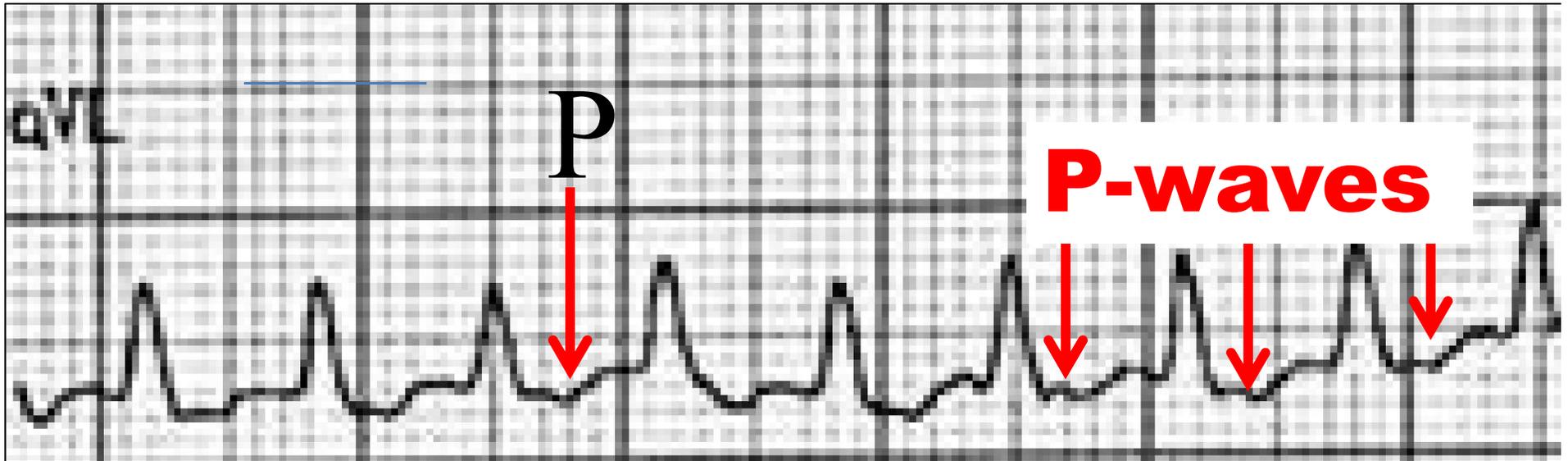
ECG-2 Admission-2: Narrow-complex QRS Paroxysmal Supraventricular Tachycardia(PSVT) regular rapid with aberrancy(LAFB-pattern) Aberrancy is common in CMT differently of AVNRT. Because presence of adverse signs (, chest pain, heart rate >200 bpm), we choose IV amiodarone.

ECG-3 : Tachycardic irregular rhythm: atrial fibrillation?

ECG-4 ECG preformed 2 hours after intravenous administration of amiodarone: Sinus rhythm with short PR interval and delta wave insinuation

What to do after the treatment? RFCA is indicated. Always before Transoesophageal echocardiography for prevention of thromboembolic accidents(61) Additionally, coronary computed tomography (CT) scan

Theoretical considerations of Narrow QRS-Tachycardias

Paroxysmal Supraventricular Tachycardia (PSVT) is an episodic condition with an abrupt onset and termination.

SVT in general is any tachyarrhythmia that requires atrial and/or atrioventricular (AV) nodal tissue for its initiation and maintenance. It is usually a narrow-complex tachycardia that has a regular, rapid rhythm; exceptions include atrial fibrillation (AF) and multifocal atrial tachycardia (MAT). Aberrant conduction during SVT results in a wide-complex tachycardia. (Second ECG.)

SVT is a common clinical condition that occurs in persons of all age groups, and treatment can be challenging.

Electrophysiologic studies are often needed to determine the source of the conduction abnormalities.

Manifestations of SVT are quite variable; patients may be asymptomatic or they may present with minor palpitations or more severe symptoms. Results from EPSs have helped to determine that the pathophysiology of SVT involves abnormalities in impulse formation and conduction pathways. The most common mechanism identified is reentry. (1-4)

Rare complications of paroxysmal SVT include myocardial infarction, congestive heart failure, syncope, and sudden death.

Classification

The development of intracardiac electrophysiologic studies has dramatically changed the classification of SVT, with intracardiac recordings having identified the various mechanisms involved in the condition. Depending on the site of origin of the dysrhythmia, SVT may be classified as an atrial or AV tachyarrhythmia. Another way to separate the arrhythmias is to classify them into conditions having either regular or irregular rhythms(5;6)

Atrial tachyarrhythmias include the following:
1. Sinus tachycardia
2. Inappropriate sinus tachycardia (IST)
3. Sinus nodal reentrant tachycardia (SNRT)
4. Atrial tachycardia
5. Multifocal atrial tachycardia
6. Atrial flutter
7. Atrial fibrillation

AV tachyarrhythmias include the following:
1. AV nodal reentrant tachycardia (AVNRT)
2. AV reentrant tachycardia (AVRT)
3. Junctional ectopic tachycardia (JET)
4. Nonparoxysmal junctional tachycardia (NPJT)

Etiology

SVT and paroxysmal SVT(PSVT) are triggered by a reentry mechanism. This may be induced by premature atrial or ventricular ectopic beats. Other triggers include hyperthyroidism and stimulants, including caffeine, drugs, and alcohol.

PSVT is observed not only in healthy individuals; it is also common in patients with previous myocardial infarction, mitral valve prolapse, rheumatic heart disease, pericarditis, pneumonia, chronic lung disease, and current alcohol intoxication. (7-10) Digoxin toxicity also may be associated with PSVT. (9-11)

Atrial tachyarrhythmias

Sinus tachycardia

Sinus tachycardia is the most common regular SVT. It has an accelerated sinus rate that is a physiologic response to a stressor. It is characterized by a heart rate faster than 100 beats per minute (bpm) and generally involves a regular rhythm. 100 bpm to 160 bpm (exceptionally 180 bpm). During the day, newborn babies: >180 bpm; between 1 week and 3 months: >220 bpm; between 3 months and 2 years: >170 bpm; from 2 years to 10 years: >110 bpm Underlying physiologic stresses such as hypoxia, hypovolemia, fever, anxiety, pain, hyperthyroidism, and exercise usually induce sinus tachycardia. (12;13) Certain drugs, such as stimulants (eg, nicotine, caffeine), medications (eg, atropine, salbutamol), recreational drugs (eg, cocaine, amphetamines, ecstasy), and hydralazine, can also induce the condition. Treatment involves addressing the basic underlying stressor.

Inappropriate Sinus Tachycardia(IST)

Concept: rare arrhythmia, hard to control and with sinus origin, characterized by persistently high, chronic tachycardic rhythm (HR over 100 bpm during the day), with more than a 3-month evolution without underlying secondary cause (Primary Sinus Tachycardia), enhanced and exaggerated adrenergic response in the face of minimal physical activities and physiological stress with inappropriate response to drugs with sinus "bradycardizing" action and sensitive to sinus node modification and close regions via catheter by radiofrequency energy. It is an accelerated baseline sinus rate in the absence of a physiologic stressor. In this situation, healthy adults may have an elevated resting heart rate and an exaggerated heart rate response to even minimal exercise. This tachyarrhythmia is observed most commonly in young women without structural heart disease. (14, 15, 10) The underlying mechanism of IST may be hypersensitivity of the sinus node to autonomic imput or an abnormality withing the sinus node and/or autonomic input Etiology: controversial. The following has been proposed: 1) beta-adrenergic hypersensitivity 2) depression of efferent reflects.

Clinical-electrocardiographic characteristics

1. Permanently high HR(>100 bpm) without justification. 2. P wave with sinus morphology, SÂP and normal polarity, which indicates sinus command or close. 3. Absence of vagal physiologic decrease of HR during sleep, with values always over 90 bpm in this period. 4. Exaggerated increase of heart rate faced with physiological stress. 5. Absence or poor response to bradycardizing drugs: β-blockers, amiodarone, verapamil or digitalis. 6. 1:1 A-V conduction. 7. Palpitations and/or pre-syncope clearly

related with rest or minimal physical efforts. 8. Clinical exclusion of secondary causes of sinus tachycardia: E.g. hyperthyroidism, pheochromocytoma and diabetes with autonomous dysfunction. 9. Holter study with medium HR over 90 bpm, with an almost normal HR during sleep and >100 bpm during the day. 10. Ergometer test: exaggerated response to HR at minimal effort. Within the first 90 seconds of the Bruce protocol, HR is observed over 130 bpm.

Management

1) Pharmacological: beta-blockers in high doses are the choice When they are contraindicate nondihydropyridine calcium antagonists or ivabradine. IST patients treated with ivabradine showed both HR normalization and quality-of-life improvement maintained in the long-term follow-up. Stopping ivabradine after 1 year unexpectedly showed that HR remained in the normal limits in 80% of the patients.(59)

If drugs fail:

2) Radiofrequency Cateter Ablation(RFCA). Regrettably, not in all the cases the return to normal rates implies abolishing the symptoms. Rate of post-radiofrequency relapses: 20% to 25% of the cases- Combined epicardial/endocardial sinus node ablation is a viable approach for patients with severely symptomatic IST after a failed endocardial attempt (60)

Sinus nodal reentrant tachycardia

SNRT is frequently confused with IST. SNRT is due to a reentry circuit, either in or near the sinus node. Therefore, it has an abrupt onset and offset. The heart rate is usually 100-150 bpm, and electrocardiographic tracings usually demonstrate a normal sinus P wave morphology. (14;15;10)

Atrial tachycardia

Atrial tachycardia is an arrhythmia originating in the atrial myocardium. Enhanced automaticity, triggered activity, or reentry may result in this rare tachycardia. (16;17;18;19;20).

The heart rate is regular and is usually 120-250 bpm.

The P-wave morphology is different from the sinus P waves and is dependent on the site of origin of the tachycardia. Because the arrhythmia does not involve the AV node, nodal blocking agents, such as adenosine and verapamil, are usually unsuccessful in terminating this arrhythmia.

Atrial tachycardia has also been associated with digoxin toxicity via the triggered mechanism

Multifocal Atrial Tachycardia (MAT) or caotic tachycardia

Multifocal atrial tachycardia is a tachyarrhythmia that arises within the atrial tissue; it is composed of 3 or more P-wave morphologies and heart rates. This arrhythmia is fairly uncommon; it is typically observed in elderly patients with chronic obstructive pulmonary disease(COPD: 80% of the cases). The heart rate is greater than 100 bpm, and electrocardiographic findings typically include an irregular rhythm,usually transitory and recurrent, which may be misinterpreted as atrial fibrillation. Treatment involves correcting the underlying disease process. (20-22) Magnesium and verapamil may sometimes be effective.

Prevalence: infrequent: 0.05% to 0.13% of hospital admittances.

1)COPD: 80%: triggered by intercurrent infection and/or excessive dose of xantines.

2)Diabetes mellitus is associated very frequently. 3) Hypertensive heart disease.

4) Coronary heart disease: both chronic coronary insufficiency and acute infarction. In the latter it is triggered by high levels of catecholamines. 5) Post-operative of general anesthesia: in general by hypoxia. 6) hypopotassemia. 7) hypomagnesemia. 8) Pulmonary embolism. 9) Pulmonary neoplasia.10) Valvular heart diseases.11) Digitalis intoxication: rare. 12) No heart disease: in patients dehydrated and with electrolytic imbalance.

MAT ELECTROCARDIOGRAHIC CHARACTERIZATION
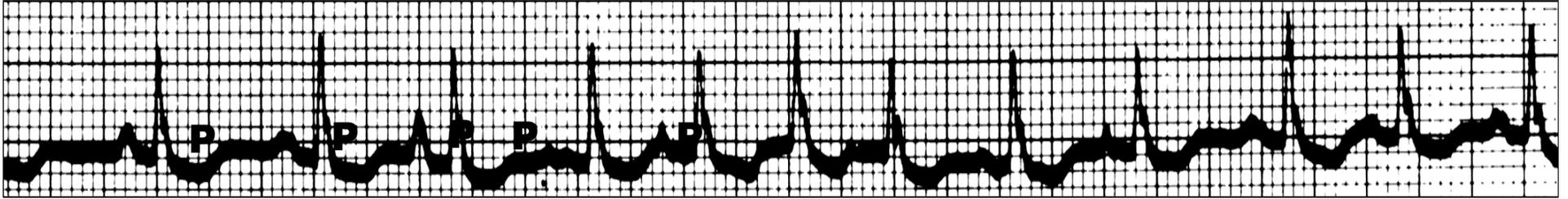
1) P waves with at least three different morphologies in the same lead.

2) Irregular P-P.

3) Presence of isoelectric line.

4) Rate of P between 100 and 130 bpm.

5) Usual AV conduction is 1:1.

6) PP, PR and RR intervals variable in duration.

7) Preceded or followed by atrial fibrillation and flutter, ectopic atrial tachycardia, sinus tachycardia.

8) It usually progresses into atrial fibrillation, and it may be confused with the latter by RR irregularity.

9) Possible presence of intermittent periods of concomitant AF or atrial flutter.

MALE PATIENT, 70 YEARS OLD, SEVERE EMPHYSEMA, COPD,
RIGHT CORONARY INSUFFICIENCY:
CHRONIC COR PULMONALE AND USE OF DIGOXIN

Comments: peaked P waves: "P pulmonale", P-P cycles with marked variation, different P waves not conduced to the ventricles with Wenckebach-type AV block, indicating digitalis intoxication, variable P morphology, slow atrial rate between 100 and 140 bpm.
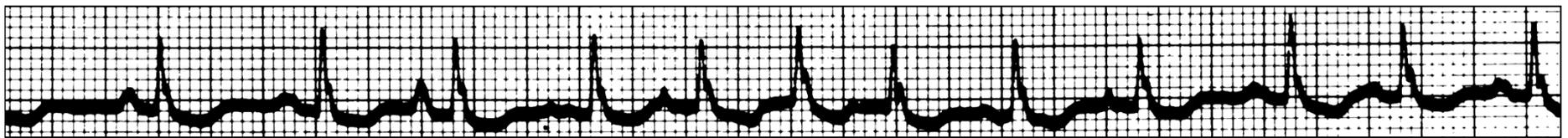
Electrocardiographic characterization.

DIFFERENTIAL DIAGNOSIS OF MAT

	MULTIFOCAL ATRIAL TACHYCARDIA	ATRIAL FIBRILLATION
PP, PR and RR intervals:	Variable.	Variable.
P wave:	Present and with three or more morphologies in the same lead and rate between 100 and 130 bpm.	Absent. Possible presence of "f" waves that present a rate between 400 and 700 bpm.
Atrial event voltage:	The P wave may be increased, indicating chamber enlargement.	It may not exist, be of small voltage ("fine") or of voltage greater than 1 mm ("coarse").
AV conduction:	Usually 1:1	There is "concealed conduction" phenomenon in the junction.

Differential diagnosis with AF.

Atrial fibrillation(AF) CONCEPT: atrial arrhythmia characterized by continuing, fragmented, chaotic, very fast and disorganized electrical activity, which causes at one and the same moment, the appearance of "islands" of depolarized cell groups, in depolarization phase and repolarized, which spread in different directions without effective atrial contraction of the biatrial chamber (loss of atrial mechanical activity) that is translated into surface ECG by absence of P waves and substituting them, small high rate waves, which are irregular and with variable voltages, called fibrillatory or "f" waves, or in certain cases only baseline. AF is an extremely common arrhythmia arising from chaotic atrial depolarization. The atrial rate is usually 300-600 bpm, while the ventricular rate may be 170 bpm or more. Electrocardiographic findings characteristically include an irregular rhythm with fibrillatory atrial activity. This arrhythmia is associated with the following condition(12;23;11): Rheumatic heart disease, hypertension, ischemic heart disease, pericarditis, thyrotoxicosis, alcohol intoxication, mitral valve prolapse and other disorders of the mitral valve and digitalis toxicity When AF occurs in young or middle-aged patients in the absence of structural heart disease or any other apparent cause, it is called lone or idiopathic, cryptogenic, lone, or isolated AF. : it is defined as the AF in absence of any etiologic factor, associated to normal ventricular function by echocardiogram. True lone AF does not possess positive familial background. AF of familial/genetic origin: it may be confused with the previous variety, because a cardiac or extra-cardiac cause is not found. Just the identification of the gene permits us to reckon it. Thus far, mutations have been identified in several chromosomes such us 10q22-q24, 11p15.5, KCNQ1, KCNE1 and

others.

Congenital short QT syndrome: it has a high tendency to atrial fibrillation, because there is an increase in the function of the potassium rectifier channels. Three mutations were described: in potassium channels: HERG, KCNQ and KCNJ2

Relevant factors in atrial fibrillation

I)   Type of underlying disease when it is present

II) Medium ventricular heart rate

III) Prevention of recurence\Potential of thromboembolism

Atrial flutter

CONCEPT: atrial tachyarrhythmia not very frequent and 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. *Atrial flutter*

Atrial flutter may be a transitional rhythm and can progress to atrial fibrillation. Electrocardiographic findings of typical atrial flutter include negative sawtooth flutter waves in leads II, III, and aVF. AV conduction is most commonly 2:1, which yields a ventricular rate of approximately 150 bpm. (11;12;23)

The ECG is characterized by the typical atrial "F" waves with sawtooth or picket-fence appearance, frequently observed better in DII, DIII, aVF and V1, with median atrial HR of 250-350 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.

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.

Causes of atrial flutter

1. Coronary heart disease: Chronic; AMI: 03% (5.3% of the cases).
2. COPD
3. Rheumatic mitral and/or tricuspid valve disease.
4. Severe aortic stenosis
5. Bronchopneumonia
6. Acute pulmonary embolism: transitory character
7. Bronchogenic carcinoma
8. Thyrotoxicosis: transitory. Frequent rate of ventricular response 1:1. Ventricular HR is 300 bpm
9. Congenital heart diseases: E.g.: ASD not operated > 40 years old. Ebstein anomaly
10. Post-operative of heart surgery. E.g.: Mustard surgery for TGA correction
11. Digitalis intoxication (exceptional)
12. As part of the "brady-tachy" syndrome
13. Myocarditis
14. Pericarditis
15. As part of WPW syndrome (frequent AV 1:1 conduction with syncope and possible sudden death)
16. Mitral valve prolapse syndrome
17. Hypertensive heart disease
18. Alcoholism.

ELECTROPHYSIOLOGICAL MECHANISMS OF ATRIAL FLUTTER

A)   DROMOTROPIC MECHANISMS

1)   Macro-reentry around fixed or functional, anatomical or surgical barriers.

2)   Micro-reentry.

B)   AUTOMATIC MECHANISMS

1)   Unifocal atrial focus.

2)   Multifocal atrial focus with very high shock.

Mechanisms.

DROMOTROPIC MECHANISMS BY MACRO-REENTRY IN ATRIAL FLUTTER

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".

SVC

"MOTHER CIRCUS WAVE"

DAUGHTER WAVES

INTERCAVAL MACROREENTRY

COUNTER-CLOCKWISE

F

F

INTERATRIAL SEPTUM

FREE WALL

F

CRANEOCAUDAL

CAUDOCRANEAL

IVC
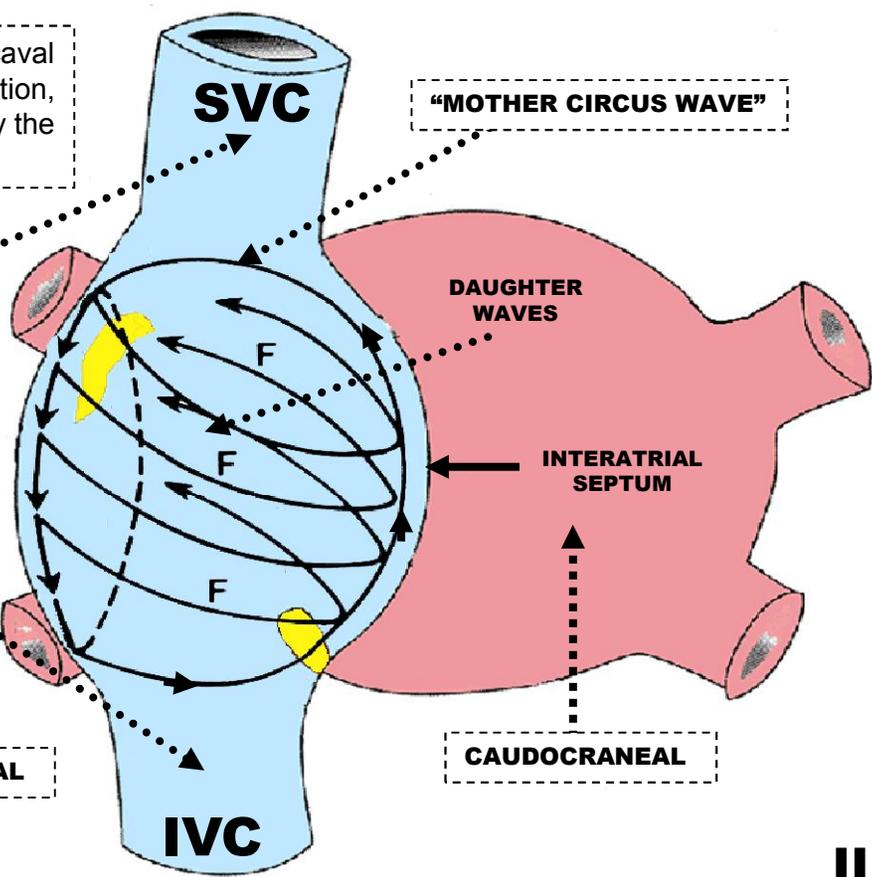
III

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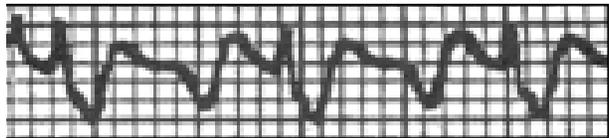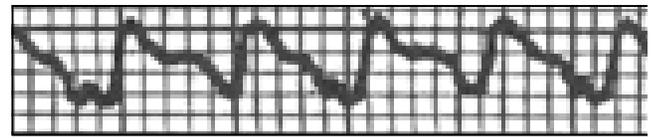
F waves of negative polarity in II, III and aVF without baseline

Outline of macro-reentry in atypical flutter.

DROMOTROPIC MECHANISMS BY MACRO-REENTRY IN ATRIAL FLUTTER

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.

CW ROTATION

F

F

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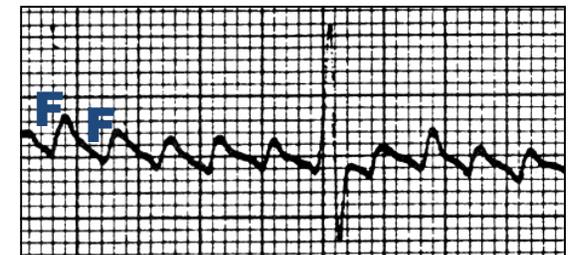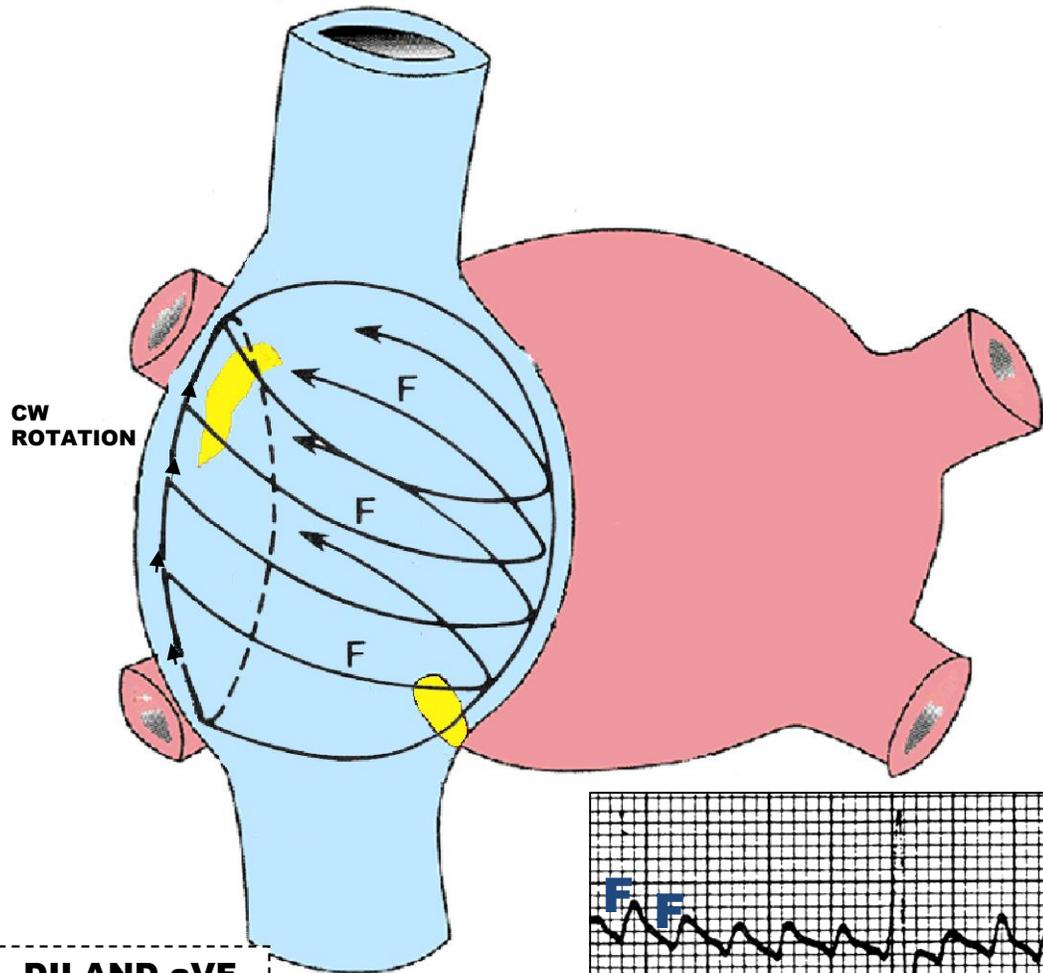
F WAVES OF POSITIVE POLARITY IN II, DII AND aVF AND WITH A MUCH GREATER RATE WITHOUT BASELINE

F F

Atrial rate of 345 bpm

Differences between flutter type I and type II.

Dromotropic Mechanism by Macro-reentry in atral flutter

|	TYPE I COMMON OR CLASSICAL	TYPE II, ATYPICAL OR RARE
Rate of F waves:	240 to 339 bpm	340 to 433 bpm.Over 300 bpm
MOST FREQUENT MECHANISM:	Intercaval macro-reentry, counterclockwise circular motion, 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.
Polarity of F waves	Negative polarity in II, III and aVF without baseline.	Positive polarity in II, III and aVF without baseline.
Drugs that may be used	Antagonists of Na^{2+} channels, except lidocaine, mexiletine and tocainide. Class IA drugs: Quinidine, (600 to 800 mg/day); Procainamide (200 to 400 mg 4/6h) and Disopyramide (300 to 400 mg/day) and IC: Propafenone (400 to 900 mg/day) and Flecainide: single dose of 300 mg. Ibutilide.	Blockers of K^+ channels: class III drugs, particularly Amiodarone (200 to 400 mg/day) and Sotalol (80 to 160 mg/day).
Response to artificial transesophageal pacing	Good	Null

CLINICS OF ATRIAL FLUTTER

History: palpitations, fatigue, poor tolerance to exercise, dyspnea, angina, dizziness and syncope.

Physical examination: heart rate approximately 150 bpm in most cases, regular or slightly irregular pulse, frequent hypotension, left CHF, syncope.

History and physical examination.

ELECTROCARDIOGRAPHIC CHARACTERIZATION

1) Characteristics of atrial activation.

2) Absence of isoelectrical "plateau" between F waves.

3) AV conduction: ventricular rate depends on AV nodal conduction; more frequently between 150 bpm and 220 bpm.

4) Intraventricular conduction that determines QRS complex duration. When it is conducted with aberrance, QRS is prolonged.

Electrocardiographic characterization.

CHARACTERISTICS OF ATRIAL ACTIVATION IN ATRIAL FLUTTER
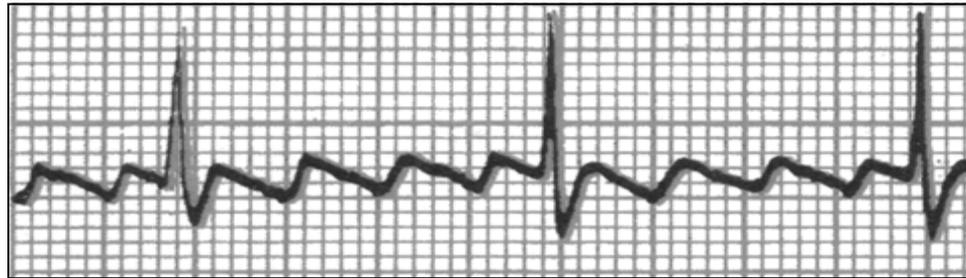
SAWTOOTH APPEARANCE

ATRIAL DEPOLARIZATION

ATRIAL REPOLARIZATION

ABSENCE OF ISOELECTRICAL
"PLATEAU"
BETWEEN F WAVES
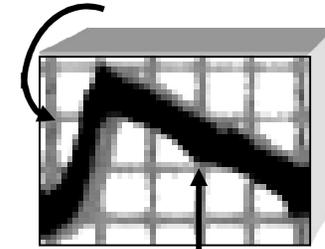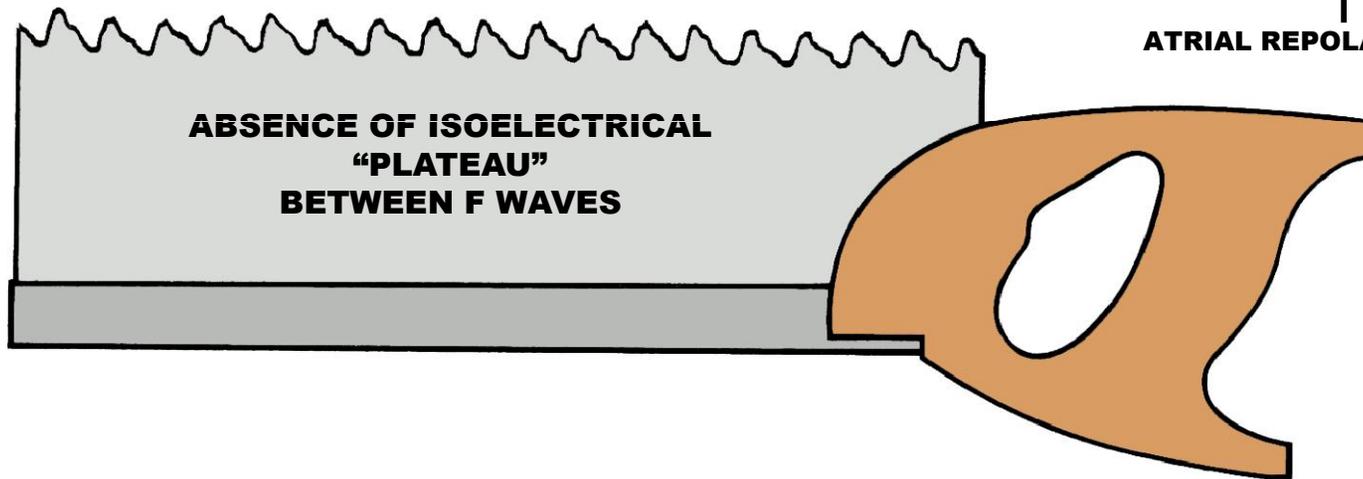
Waves with sawtooth or picket fence appearance called F waves, with a heart rate between 250 and 350 bpm, observed better in the inferior wall and V_1 with slowly descending and rapidly ascending ramp. These waves resemble an inverted P wave, followed by an ascending ramp: "Tp" waves.

Electrocardiographic characterization.

CHARACTERISTICS OF ATRIAL ACTIVATION IN ATRIAL FLUTTER

In the common form, the polarity of F waves is negative in inferior leads II, III and aVF: caudocephalic atrial activation: type I, common or classical flutter.

The DII or V_1 leads should be recorded separately, in prolonged tracings (from 15s to 20s) to establish the relationship between F waves and QRS complexes.

During atrial flutter with 2:1 conduction, the ''F'' waves may be masked in the DII leads and be very visible in V1.

Electrocardiographic characterization.

CHARACTERISTICS OF ATRIAL ACTIVATION IN ATRIAL FLUTTER

CAUSES OF F WAVES OF LOW RATE FLUTTER

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

Electrocardiographic characterization.

CHARACTERISTICS OF ATRIAL ACTIVATION IN ATRIAL FLUTTER

CAUSES OF F WAVES OF FLUTTER OF HIGH RATE

1) Flutter in children: median HR of 300 bpm with 1:1 response.

2) Flutter of ventricular pre-excitation.

3) Type II flutter of Wells: rate of F between 340 and 433 bpm: it cannot be interrupted with pacing.

Electrocardiographic characterization.

TYPES OF ATRIOVENTRICULAR CONDUCTION IN ATRIAL FLUTTER

A) REGULAR: 2:1 – $\frac{1}{2}$

1:1 – suggests ventricular pre-excitation

3:1 – 1/3

4:1 – 1/4

B) IRREGULAR

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

AV conduction in flutter.

REGULAR ATRIAL FLUTTER WITH 1:1 AV CONDUCTION

Examples of 1:1 flutter.

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:

A) Pre-excitation of WPW type, because the stimulus is conduced in anterograde fashion by the anomalous pathway;
B) Atrial flutter secondary to hyperthyroidism;
C) Flutter of the pediatric group;
D) 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.

REGULAR ATRIAL FLUTTER WITH 2:1 AV CONDUCTION

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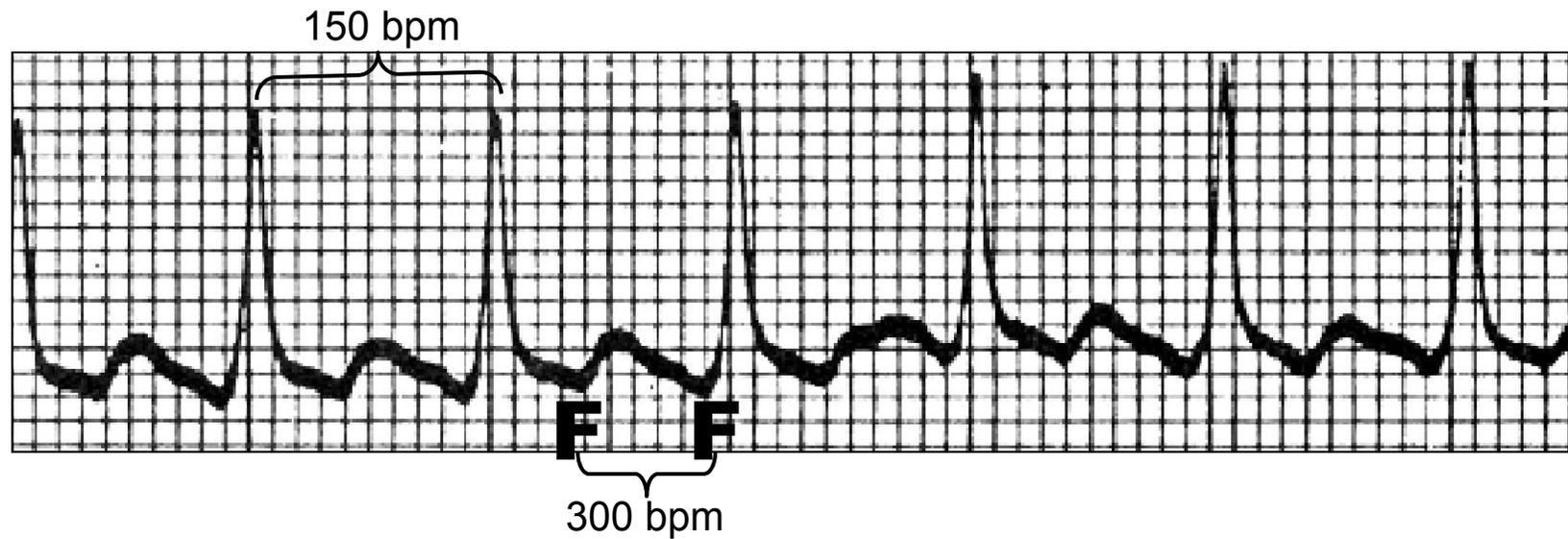
The most frequent ratio in non-treated patients is 2:1 with atrial and ventricular rate of 300/150 bpm respectively; this ratio is due to the physiological interference in the junction. If the ventricular response rate is regular and constant (e.g.: always 2:1) the F-R interval will be too, varying between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

AV tachyarrhythmias

AV nodal reentrant tachycardia

The most common cause of paroxysmal SVT is AVNRT. AVNRT is diagnosed in 50-60% of patients who present with regular narrow QRS tachyarrhythmia. (3;23;24).

The heart rate is 120-250 bpm and is typically quite regular.

AVNRT may occur in healthy, young individuals, and it occurs most commonly in women.(25) Most patients do not have structural heart disease. However, occasionally these individuals may have an underlying heart condition such as rheumatic heart disease, pericarditis, myocardial infarction, mitral valve prolapse, or preexcitation syndrome. (3;24;25)

An understanding of the electrophysiology of AV nodal tissue is very important in comprehending the mechanism of AVNRT. In most people, the AV node has a single conducting pathway that conducts impulses in an anterograde manner to depolarize the bundle of His. In certain cases, AV nodal tissue may have 2 conducting pathways with different electrophysiologic properties. One pathway (alpha) is a relatively slow conducting pathway with a short refractory period, while the second pathway (beta) is a rapid conducting pathway with a long refractory period. The coexistence of these functionally different pathways serves as the substrate for reentrant tachycardia. (3;23;24;9)Electrophysiologic studies have demonstrated dual AV nodal pathways in 40% of patients.

The onset of AVNRT is triggered by a premature atrial impulse. A premature atrial impulse may reach the AV node when the fast pathway (beta) is still refractory from the previous impulse but the slow pathway (alpha) may be able to conduct. The premature impulse then conducts through the slow pathway (alpha) in an anterograde manner; the fast pathway (beta) continues to recover

because of its longer refractory period.

After the impulse conducts in an anterograde manner through the slow pathway (alpha), it may find the fast pathway (beta) recovered. The impulse then conducts in a retrograde manner via the fast (beta) pathway. If the slow pathway (alpha) has repolarized by the time the impulse completes the retrograde conduction, the impulse can reenter the slow pathway (alpha) and initiate AVNRT.
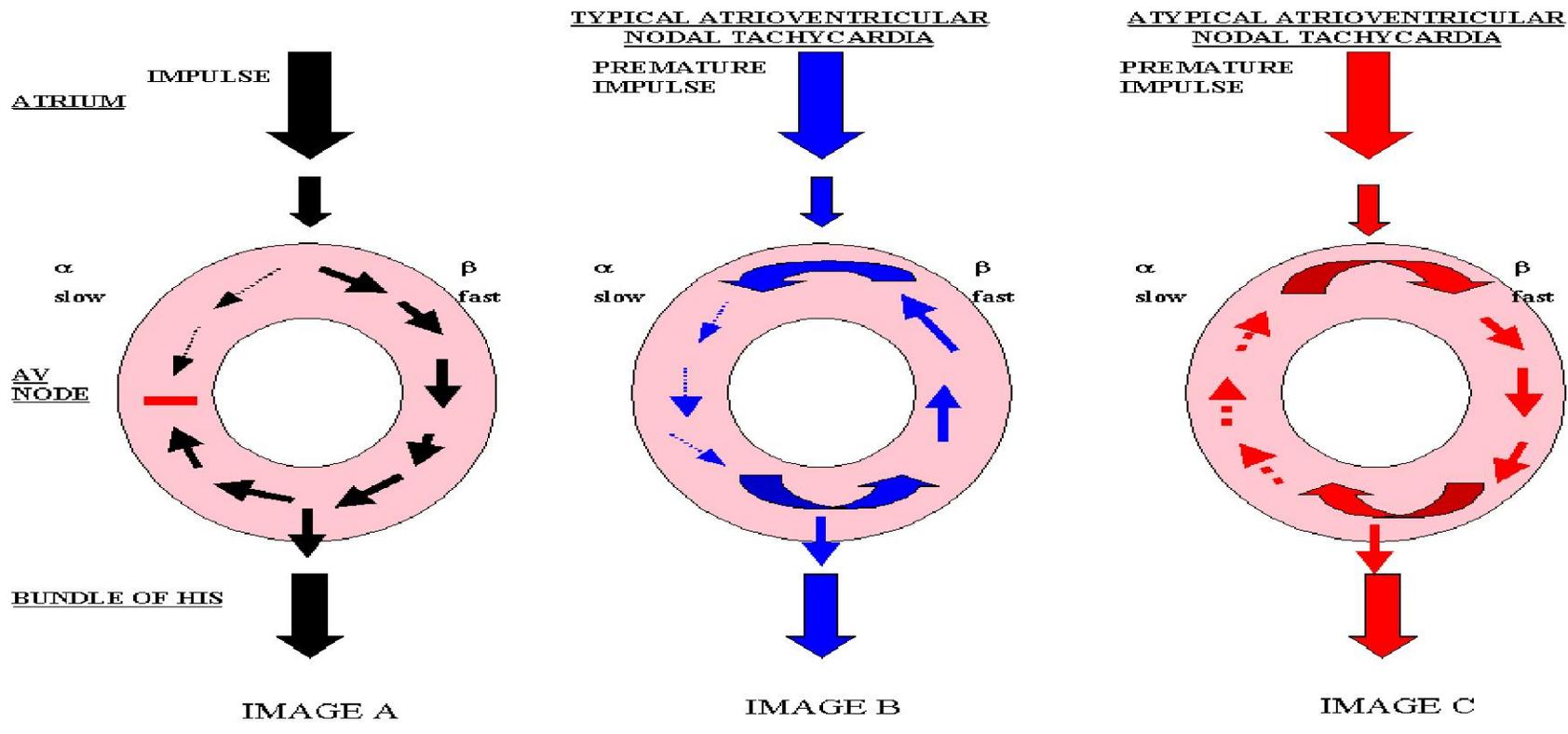
Image A displays the slow pathway and the fast pathway, with a regular impulse being conducted through the atrioventricular node. Image B displays a premature impulse that is conducted in an anterograde manner through the slow pathway and in a retrograde manner through the fast pathway, as is seen in typical atrioventricular nodal tachycardia. Image C displays the premature impulse conducting in a retrograde manner through the pathway and the impulse reentering the pathway with anterograde conduction, which is seen commonly in patients with atypical atrioventricular nodal tachycardia.

Importantly, note that AVNRT does not involve the ventricles as part of the reentry circuit; the necessity of perinodal atrial tissue to the circuit is controversial. Because the impulse typically conducts in an anterograde manner through the slow pathway and in a retrograde manner through the fast pathway, the PR interval is longer than the RP interval. Thus, in patients with typical AVNRT, the P wave is usually located at the terminal portion of the QRS complex. (3;23;11;24;9)

In patients with atypical AVNRT, anterograde conduction is via the fast pathway, while retrograde conduction is via the slow pathway. For these atypical patients, the RP interval is longer than the PR interval. (24;26;23;25;3;9;27;11)

AV Reentrant Tachycardia (AVRT)

AVRT is the second most common form of paroxysmal SVT. The incidence rate of AVRT in the general population is 0.1-0.3%. AVRT is more common in males than in females (male-to-female ratio of 2:1), and patients with AVRT commonly present at a younger age than do patients with AVRT. AVRT is associated with the Ebstein anomaly, although most patients with AVRT do not have evidence of structural heart disease. AVRT occurs in the presence of accessory pathways, or bypass tracts. Accessory pathways are errant strands of myocardium that bridge the mitral or tricuspid valves. (24;28;9;10) AVRT results from the presence of 2 or more conducting pathways; specifically, the AV node and 1 or more bypass tracts. In a normal heart, only a single route of conduction is present. Conduction begins at the sinus node, progresses to the AV node, and then to the bundle of His and the bundle branches. However, in AVRT, 1 or more accessory pathways connect the atria and the ventricles. The accessory pathways may conduct impulses in an anterograde manner, a retrograde manner, or both. (29;30;24;31;28;32;9;10) When impulses travel down the accessory pathway in an anterograde manner, ventricular preexcitation results. This produces a short PR interval and a delta wave, as is observed in persons with WPW syndrome. Importantly, note that not all accessory pathways conduct in an anterograde manner. Concealed accessory pathways are not evident during sinus rhythm, and they are only capable of retrograde conduction. A reentry circuit is most commonly established by impulses traveling in an anterograde manner through the AV node and in a retrograde manner through the accessory pathway; this is called orthodromic AVRT.

A reentry circuit may also be established by a premature impulse traveling in an anterograde manner through a manifest accessory pathway and in a retrograde manner through the AV node; this is called antidromic AVRT. While the orthodromic AVRT is typically a narrow-complex tachycardia, antidromic AVRT inscribes a bizarre, wide-complex tachycardia.(33;34;35)

Patients with WPW syndrome can develop AF and atrial flutter . The rapid non decremental conduction via the accessory pathways can result in extremely rapid rates, which can degenerate to VF and cause SCD. Patients with preexcitation syndromes with AF must not be administered an AV nodal blocking agent; these agents can further increase conduction via the accessory pathway, which increases the risk of ventricular fibrillation and death. (36;37;7;38;8;39).

ECG Recognition

Usually initiated by atrial premature beat, heart rate 170 to 250 bpm, regular rhythm or slightly irregular in casses of changing conduction velocities through the AV node, P wave buried within the QRS complex; either distorts the end of the QRS complex or is not seen at all, QRS complex narrow but often disotrted by the P'wave, causing a pseudo S wave in the inferior leads and V6 and/or pseudo r' in lead V1.

Usually benign and self-limiting. Easily terminated by a vagal maneuver(patient shuld be taught several valgal maneuvers. Occasionally refractory to vagal maneuvers and referred for RFCA.

Treatment: RFCA of slow atrionodal pathway

Junctional ectopic tachycardia and nonparoxysmal junctional tachycardia

JET and NPJT are rare; they presumably arise because of increased automaticity, triggered activity, or both. They are usually observed following valvular surgery, after myocardial infarction, during active rheumatic carditis, or with digoxin toxicity. These tachycardias are also observed in children following congenital heart surgery. Electrocardiographic findings include a regular narrow QRS complex, although P waves may not be visible. Patients with AV dissociation have also been described(9; 40;41).

Epidemiology

Occurrence in the United States

The incidence of paroxysmal SVT is approximately 1-3 cases per 1000 persons. The incidence rate of the WPW pattern on electrocardiographic tracings is 0.1-0.3% in the general population, although not all patients develop SVT.(7-10)

In a population-based study, the prevalence of paroxysmal SVT was 2.25 cases per 1000 persons, with an incidence of 35 cases per 100,000 person-years. (43) AVNRT is more common in patients who are middle aged or older, while adolescents are more likely to have SVT mediated by an accessory pathway.

Paroxysmal SVT is observed not only in healthy individuals; it is also common in patients with previous myocardial infarction, mitral valve prolapse, rheumatic heart disease, pericarditis, pneumonia, chronic lung disease, and current alcohol intoxication. (7-10) Digoxin toxicity also may be associated with paroxysmal SVT. (9;10;11)

Sex-related demographics

Most series of RFCA reflect a higher proportion of female patients with AVNRT than male patients. This may reflect a true higher incidence in women, or it may reflect the sample of patients who are referred (or choose) to undergo extensive evaluation and/or RFCA.

In a population-based study, the risk of developing paroxysmal SVT was twice as high in women as it was in men. (43)

Age-related demographics The prevalence of paroxysmal SVT increases with age. AVNRT is seen more commonly in persons who are middle aged or older, while adolescents usually have SVT from an accessory pathway.(43) The relative frequency of tachycardia mediated by an accessory pathway decreases with age. **Prognosis:** Patients with symptomatic WPW syndrome have a small risk of SD. Otherwise, prognosis in paroxysmal SVT is dependent on any underlying structural heart disease; patients with a structurally normal heart have an excellent prognosis. **Morbidity and mortality** Paroxysmal SVT may start suddenly and last anywhere from seconds to days. Patients may or may not be symptomatic, depending on their hemodynamic reserve and heart rate, the duration of the paroxysmal SVT, and coexisting diseases. Paroxysmal SVT can result in HF, pulmonary edema, myocardial ischemia, and/or myocardial AMI secondary to an increased HR in patients with poor Lvfunction. (9;10;11) In fact, one study found that one third of patients with SVT experienced syncope or required cardioversion.(44) Incessant SVT can cause tachycardia-induced cardiomyopathy. Patients with WPW syndrome may be at risk for cardiac arrest if they develop AF or atrial flutter in the presence of a rapidly conducting accessory pathway (ie, a pathway with a short anterograde refractory period). Extremely rapid ventricular rates during AF or atrial flutter can cause deterioration to VF. This complication is unusual and occurs primarily in patients who have had prior symptoms due to WPW syndrome. SCD may be the initial presentation of WPW syndrome, but how often this occurs is unclear. In the absence of manifest preexcitation (ie, WPW syndrome), the risk of sudden death with paroxysmal SVT is extremely small.

History: Because symptom severity depends on the presence of structural heart disease and on the hemodynamic reserve of the patient, individuals with paroxysmal SVT may present with mild symptoms or severe cardiopulmonary complaints. Their frequency rates are as follows(42;44): Palpitations (>96%), dizziness(75%), shortness of breath (47%), syncope (20%), chest pain (35%), fatigue (23%), diaphoresis (17%), and nausea (13%). Palpitations and dizziness are the most common symptoms reported by patients with SVT. Chest discomfort may be secondary to a rapid heart rate, and it frequently subsides with the termination of the tachycardia. Persistent SVT may lead to tachycardia-induced cardiomyopathy. History should include time of onset, any triggers, any previous episodes or arrhythmia, and previous treatment. A detailed past medical and cardiac history and a complete list of all medications should be obtained. Patients who are hemodynamically unstable should be resuscitated immediately with cardioversion. An ECG should be performed as soon as possible. Many patients with frequent episodes of paroxysmal SVT tend to avoid activities such as exercising and driving due to past episodes of syncope or near-syncope.

Physical examination: Pertinent findings are generally limited to the patient's cardiovascular and respiratory systems. Patients often appear quite distressed. Tachycardia may be the only finding in persons who are otherwise healthy and have significant hemodynamic reserve. Patients who have limited hemodynamic reserve may be tachypneic and hypotensive. Crackles may be auscultated secondary to HF. An S_3 may be present, and large jugular venous pulsations may also be visualized. (9;44;10).

Paroxysmal Supraventricular Tachycardia Differential Diagnoses

1. Atrial Fibrillation
2. Atrial Flutter
3. Atrial Tachycardia
4. Atrioventricular Nodal Reentry Tachycardia (AVNRT)
5. Sinus Node Dysfunction
6. Ventricular Fibrillation
7. Ventricular Tachycardia.

Approach Considerations

Laboratory studies: A cardiac enzyme evaluation should be ordered for patients with chest pain, patients with risk factors for myocardial infarction, and patients who are otherwise unstable and present with heart failure, hypotension, or pulmonary edema. Young patients with no structural heart defects have a very low risk of myocardial infarction.

Other laboratory tests include the following: Electrolyte levels - Should be checked because electrolyte abnormalities can contribute to paroxysmal SVT

Complete blood count (CBC) - Helps to assess whether anemia is contributing to the tachycardia or ischemia Thyroid studies - The results are rarely diagnostic of hyperthyroidism Digoxin level - Obtain for patients on digoxin, because paroxysmal SVT is one of the many dysrhythmias that can be caused by supratherapeutic levels of this drug.

Electrophysiology Electrophysiologic studies have dramatically changed the diagnosis of SVT. Intracardiac recordings have helped to map accessory pathways and reentry circuits in patients, and they have also assisted cardiologists and electrophysiologists in understanding the mechanisms behind these tachyarrhythmias.

At present, electrophysiologic studies are generally performed in combination with radiofrequency catheter ablation.

Chest radiography

Obtain a chest radiograph to assess for the presence of pulmonary edema and cardiomegaly. Infections such as pneumonia, which in certain cases are associated with paroxysmal SVT, can also be confirmed with findings from this imaging method. (9;40;10;41;11).Congenital heart defects such as Ebstein anomaly of the tricuspid valve can be suspected.

Transthoracic echocardiography

A transthoracic echocardiogram may be helpful if structural or congenital heart disease is suggested.

Magnetic resonance imaging

Cardiac magnetic resonance imaging (MRI) can be useful, especially if a congenital heart disease is being considered.

Electrocardiography

Electrocardiographic findings permit classification of the tachyarrhythmia, and they may allow a precise diagnosis. P waves may not be visible; when present, they may be normal or abnormal, depending on the mechanism of atrial depolarization. (9;10;35) Electrocardiographic characteristics of the various SVTs are as follows:

Sinus tachycardia – HR > 100 bpm; P waves similar to sinus rhythm

Inappropriate sinus tachycardia (IST) - Findings similar to sinus tachycardia; P waves similar to sinus rhythm

Sinus nodal reentrant tachycardia (SNRT) - P waves similar to sinus rhythm; abrupt onset and offset

Atrial tachycardia - HR 120-250 bpm; P-wave morphology different from sinus rhythm; long RP interval (in general); AV block does not terminate tachycardia

Multifocal atrial tachycardia - Heart rate 100-200 bpm; 3 or more different P-wave morphologies

Atrial flutter- Atrial rate of 200-300 bpm; flutter waves; AV conduction of 2:1 or 4:1

Atrial fibrillation - Irregularly irregular rhythm; lack of discernible P waves

AV nodal reentrant tachycardia (AVNRT) - HR of 150-200 bpm; P wave located either within the QRS complex or shortly after the QRS complex; short RP interval in typical AVNRT and long RP interval in atypical AVNRT

AV reentrant tachycardia (AVRT) - HR of 150-250 bpm; narrow QRS complex in orthodromic conduction and wide QRS in antidromic conduction; diagnosis excluded by AV block during SVT; P wave after QRS complex

Following the termination of the tachycardia, an ECG should be performed during the sinus rhythm to screen for WPW syndrome. Echocardiography and/or Holter monitoring also may be useful. These tests can help to assess the frequency and duration of SVT episodes, although they have a low yield. Echocardiography may be helpful in screening for structural or congenital heart disease.

Characterizing a patient's SVT by comparing the RP interval to the PR interval is helpful. Long RP tachycardias result when atrial activity precedes the QRS complex. In short RP tachycardias, atrial activity occurs with or shortly after ventricle excitation, and the P wave is found within the QRS complex or shortly after the QRS complex(9;10; 40;41). The classifications of SVTs based on the RP interval are as follows:

Short RP tachycardias – Typical AVNRT, AVRT, junctional ectopic tachycardia (JET), and nonparoxysmal junctional tachycardia (NPJT)

Long RP tachycardias – Sinus tachycardia, SNRT, atrial tachycardia, atrial flutter, atypical AVNRT, and a permanent form of junctional reciprocating tachycardia

Two consecutive P waves without an intervening QRS complex may be due to atrial tachycardias, in some cases, AVNRT, but they are unlikely to be due to AVRT.

Vagal maneuvers and nodal blocking agents like adenosine work in AVNRT in some cases, but not in atrial tachycardias. Blocking the tachycardia with adenosine or vagal maneuvers may assist in diagnosing the rhythm as well as treating it.

Approach Considerations

Patients with symptomatic WPW syndrome should be told of the potential for cardiac arrest. In general, these patients should not be treated longitudinally with calcium channel blockers or digoxin unless the pathway is known to be of low risk (long anterograde refractory period). This is because of the potential for more rapid ventricular rates should AF or atrial flutter occur.

Patients with preexcited AF should not be treated with intravenous AV nodal blocking agents, such as adenosine, beta-blockers, calcium channel blockers, and digoxin. Rather, if the patient is hemodynamically stable, intravenous procainamide should be administered. If the patient is unstable, direct current cardioversion should be performed.

Most of the patients who present with paroxysmal supraventricular tachycardia (paroxysmal SVT) have AVNRT or AVRT. These arrhythmias depend on AV nodal conduction and therefore can be terminated by transiently blocking this conduction.

Electrical cardioversion

Electrical cardioversion is the most effective method for restoring sinus rhythm. Synchronized cardioversion starting at 50J can be used immediately in patients who are hypotensive, have pulmonary edema, have chest pain with ischemia, or are otherwise unstable.If AF has been present for longer than 24-48 hours, defer cardioversion until the patient has been adequately anticoagulated to prevent thromboembolic complications. (40; 36; 45;46;47;48;49;41)

Inpatient care: Patients who require cardioversion, are unstable, and have comorbid illnesses should be admitted to the hospital. Patients who are young, healthy, and asymptomatic may be discharged and advised to have a follow-up examination with their primary physician or cardiologist. If the patient is having more frequent episodes of paroxysmal SVT and medical therapy is not successful or desired, then RFCA should be proposed.

Diet and activity: Dietary changes depend on underlying medical problems. Changes in physical activity depend on underlying cardiac problems and other comorbidities.

Consultations: A cardiologist should be consulted for patients with frequent episodes of paroxysmal SVT, syncope, and/or preexcitation syndromes. Consultation with a cardiologist should also be obtained for patients in whom medical management has failed. An electrophysiologist should be consulted for patients considered for RFCA. Pediatric patients should be referred to a pediatric electrophysiologist.

Transfer: Patient transfer to a center with RFCA is reasonable if this therapy is planned. Alternatively, patients can be discharged home and scheduled for outpatient procedures. Exceptions include patients with syncope, profound symptoms, or preexcited atrial fibrillation or atrial flutter.

Monitoring: Patients treated medically should be monitored regularly. Patients cured with RFVC are typically seen once in a follow-up examination following the procedure, then as needed for recurrent symptoms.

The first-line treatment in hemodynamically stable patients, vagal maneuvers, such as breath-holding and the Valsalva maneuver (ie, having the patient bear down as though having a bowel movement), slow conduction in the AV node and can potentially interrupt the reentrant circuit.

Carotid massage is another vagal maneuver that can slow AV nodal conduction. Massage the carotid sinus for several seconds on the nondominant cerebral hemisphere side. This maneuver is usually reserved for young patients. Due to the risk of stroke from emboli, auscultate for bruits before attempting this maneuver. Do not perform carotid massage on both sides. A Valsalva maneuver, if performed properly by the patient, can frequently avert an attack.

Short-Term Pharmacologic Management

When SVT is not terminated by vagal maneuvers, short-term management involves intravenous adenosine or calcium channel blockers. Adenosine is a short-acting drug that blocks AV node conduction; it terminates 90% of tachycardias due to AVNRT or AVRT. Adenosine does not usually terminate atrial tachycardia, although it is effective for terminating SNRT.[40, 36, 45, 50, 48, 41]

Typical adverse effects of adenosine include flushing, chest pain, and dizziness. These effects are temporary because adenosine has a very short half-life of 10-20 seconds. (49)

Other alternatives for the acute treatment of SVT include calcium channel blockers, such as verapamil and diltiazem, as well as beta-blockers, such as metoprolol or esmolol. Verapamil is a calcium channel blocker that also has AV blocking properties. It has a longer half-life than adenosine and may help to maintain sinus rhythm following the termination of SVT. It is also advantageous for controlling the ventricular rate in patients with atrial tachyarrhythmia. (19;51;54;46; 10;48;49; 11)

Atrial fibrillation and atrial flutter

The treatment of atrial fibrillation and atrial flutter involves controlling the ventricular rate, restoring the sinus rhythm, and preventing embolic complications. The ventricular rate is controlled with calcium channel blockers, digoxin, amiodarone, and beta-blockers. The sinus rhythm may be restored with either pharmacologic agents or electrical cardioversion. Medications such as ibutilide convert AF and atrial flutter of short duration to sinus rhythm in approximately 30% and 60% of patients, respectively.

Long-Term Pharmacologic Management

The choice of long-term therapy for patients with SVT depends on the type of tachyarrhythmia that is occurring and the frequency and duration of episodes, as well as the symptoms and the risks associated with the arrhythmia (eg, heart failure, sudden death). Evaluate patients on an individual basis, and tailor treatment to the best therapy for the specific tachyarrhythmia.

Patients with paroxysmal SVT may initially be treated with calcium channel blockers, digoxin, and/or beta-blockers. Class IA, IC, or III antiarrhythmic agents are used less frequently because of the success of radiofrequency catheter ablation. (40;36;45;46;47;48;49;41;53)

Percutaneous Radiofrequency Catheter Ablation

Prior to the advent of percutaneous RFCA, open cardiac surgical procedures were the only means of curing paroxysmal SVT. Currently, however, open surgical procedures are rarely performed, and RFCA is considered the first-line treatment of many SVTs. It is generally performed using conscious sedation in an outpatient setting or with an overnight hospital stay for observation. RFCA involves focally ablating the crucial component of the arrhythmic mechanism. For example, in AVNRT, the slow pathway is ablated, which prevents the reentry cycle. The accessory pathway is targeted in patients with AVRT. Focal atrial tachycardia, atrial flutter, and, in some cases, AF can also be cured with RFCA. Consider RFCA for any patient with symptomatic paroxysmal SVT in whom long-term medical treatment is not effectively tolerated or desired. In addition, because of the risk of SCD, perform RFCA on patients with symptomatic WPW syndrome.(9; 40;10;41;13) The optimal management strategy for patients with asymptomatic preexcitation syndromes remains uncertain. (54;55;19;9;15) The efficacy of RFCA often exceeds that of medical therapy for symptoms, recurrences requiring medical intervention, and the prevention of consequences, such as defibrillator discharges in patients with an implanted defibrillator and SVT. (A study by Mainigi et al found that SVT causes a significant number of inappropriate implantable cardioverter-defibrillator therapies and that RFCA is an effective strategy to avoid these inappropriate therapies. (56) RFCA is more than 90% effective in curing paroxysmal SVT.

Complications Potential complications of RFCA include: Hematoma, bleeding, infection, pseudoaneurysm, myocardial infarction, cardiac preformation, heart block that requires a pacemaker, thromboembolic complications (deep venous thrombosis and systemic embolism, cardiac tamponade, stroke, need for emergency surgery) , radiation burn, increased risk of malignancy - Lifetime risk of fatal malignancy as a result of radiation exposure is low. Death - Risk is approximately 0.1% Bohnen et al performed a prospective study to assess the incidence and predictors of major complications from contemporary RFCA procedures. Major complication rates ranged between 0.8% (SVT) and 6% (VT associated with structural heart disease), depending on the ablation procedure performed. The investigators reported that renal insufficiency was the only independent predictor of a major complication.(57)

Medication Summary: s previously stated, short-term management of SVT involves intravenous adenosine or calcium channel blockers. In cases of wide-complex tachycardia, hemodynamically stable patients can be treated with intravenous procainamide, propafenone, or flecainide. Amiodarone is preferred in patients with impaired LV function or in patients with HF or structural heart disease.(52)

Treatment for AF and atrial flutter includes medications that control the ventricular rate (calcium channel blockers, digoxin, amiodarone, beta-blockers), restore the sinus rhythm (such as ibutilide), and prevent embolic complications. Long-term pharmacologic therapy for patients with SVT depends on the type of tachyarrhythmia that is occurring and the frequency and duration of episodes, as well as the symptoms and the risks associated with the arrhythmia (eg, heart failure, sudden death).

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