

UNUSUAL TACHYARRHYTHMIA

Bidirectional Ventricular Tachycardia: BVT

Case of Dr. Raimundo Barbosa de Barros Fortaleza - Ceará Brazil
Commentaries Andrés Ricardo Pérez Riera MD Sao Paulo Brazil

Prezado Dr. Andrés

Paciente do sexo feminino, 47 anos, portadora de cardiomiopatia dilatada, em uso de digoxina, carvedilol, captopril e furosemida.

Deu entrada na sala de emergência com queixas de palpitações, náuseas, vômitos e piora do quadro dispnéico.

Qual o diagnóstico e a terapia adequada?

Um abraço

Raimundo Barbosa Barros

Appreciated Dr. Andrés

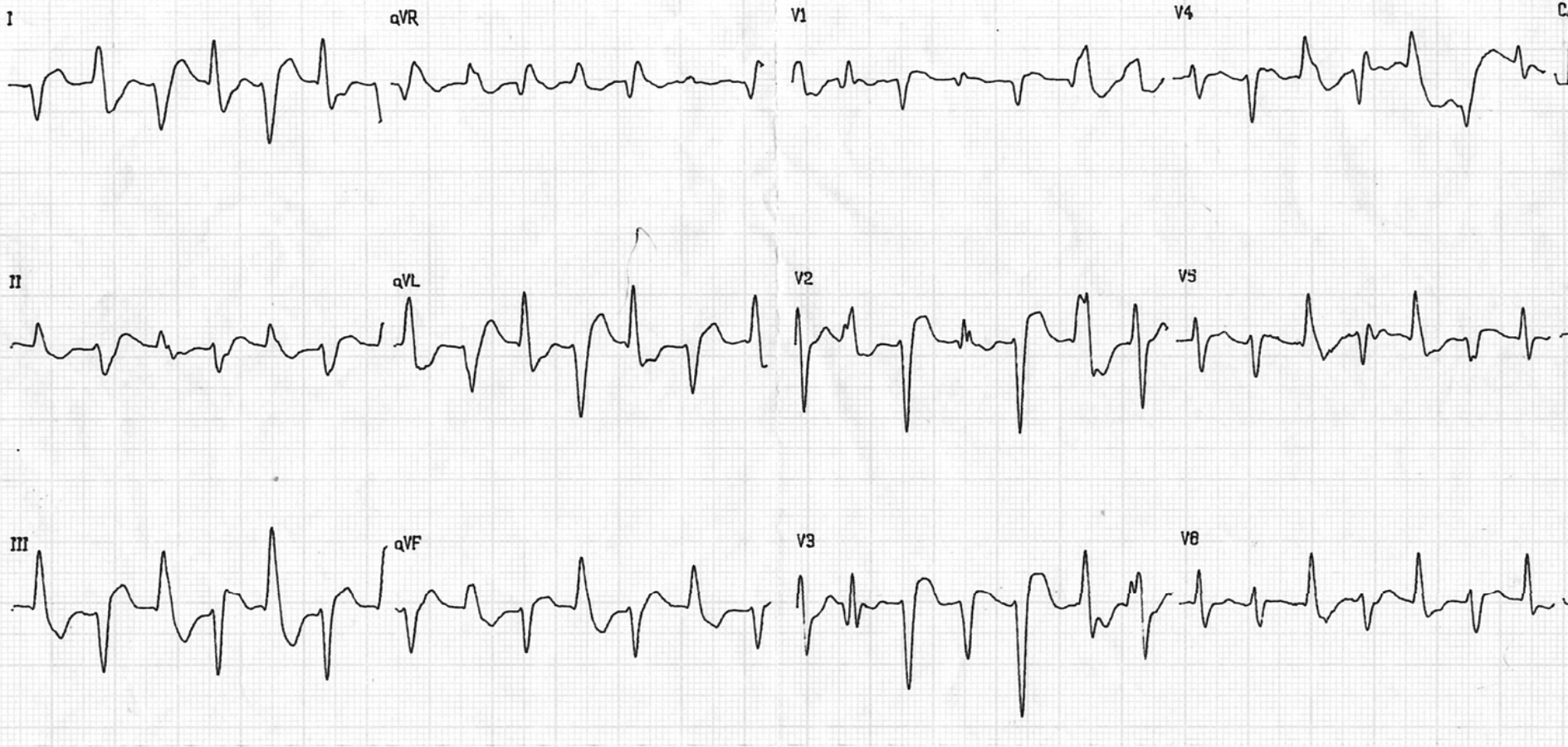
Feminine 47 years old, carrier idiopathic dilated cardiomyopathy. In regular use of digoxin, carvedilol and furosemide in therapeutic dose.

Gave entry in our emergency room with complain of palpitations, nauseas vomiting and worsening dispneic picture.

Which the diagnosis and the appropriate therapy?

A hug

Raimundo Barbosa Barros



Note the alternating QRS axes on frontal plane from right axis deviation followed by left axis deviation(alternating aberrant conduction by left anterior fascicle and left posterior fascicle¹. Additionally, irregular alternating right and left bundle branch block-type on precordial leads is observed². This pattern could occur in the setting of a supratherapeutic serum level of digoxin as a consequence of acute renal failure.

Conclusion: Type II bidirectional tachycardia: BVT Type II the intervals among the QRS complexes are shorts or longs. The intervals among equals QRS complexes are constants, and the intervals among different morphological QRS complexes are not equals.

1. Morris SN, Zipes DP His bundle electrocardiography during bidirectional tachycardia *Circulation* 1973;38: 32-36.
2. Khan IA. Exercise-induced bidirectional ventricular tachycardia with alternating right and left bundle branch block-type patterns--a case report. *Angiology*. 2002 Sep-Oct;53:593-598.
3. Rosenbaum MB, Elizari MV, Lazzari JO. The mechanism of bidirectional tachycardia. *Am Heart J*. 1969 Jul;78:4-12.

BVT: HISTORICAL CHRONOLOGY DISCOVERIES

1911: Levy and Lewis¹ suggested from experiments in cats that two independently firing ventricular pacemakers were operating.

1922: First description² Schwensen concluded that this rhythm was ventricular in origin.

1923: Felderbaum³ proposed that the focus might be in the atrioventricular node or main His.

1925: Luten D⁴ and Sir Thomas Lewis⁵ are favorable to single ventricular focus origin conducted with alternant aberration by bundle branch block.

1926: First Gallavardin⁶ suggest a single ventricular focus origin and a double circle movement with eight morphology to explain the alternating QRS complexes
.....and two years later

1928: Palmer and White⁷

1. Levy AG, Lewis T. Heart irregularities, resulting from the inhalation of low percentage of chloroform vapor, and their relationship to ventricular fibrillation. *Heart* 1911; 3:99.
2. Schwensen E: Ventricular tachycardia as a result o the administration of digitalis *Heart* 1922; 9: 199.
3. Felderbaum D. Paroxysmal ventricular tachycardia; Report of a case of unusual type *Am J Med Sci.* 1922; 166: 199-211.
4. Luten D: Clinical studies of digitalis. III. Advanced toxic rhythm. *Arch Intern Med* 1925; 35:87.
5. Lewis T: The mechanism and graphic registration of the heart beat. Ed 3, London, Shawand Sons, 1925.
6. Gallavardin L Tachycardie ventriculaire á paroxysmes tachycardiques *Arch Mal. Coeur* 1926; 19: 153.
7. Palmer RS, White PD: Paroxysmal ventricular tachycardia with rhythmic alternation in the direction of the ventricular complexes of the electrocardiogram *Am Hear J* 1928; 3: 454.

1939: Sherf and Kisch¹ postulated a classification in tree types: I, II and III.

•**Type I** – rare – has equal intervals among the QRS complexes.

•**Type II** the intervals among the QRS complexes are shorts or longs. The intervals among equals QRS complexes are constants, and the intervals among different morphological QRS complexes are not equals.

•**Type III:** The morphology of QRS complexes are different and the intervals are irregularly variables.

1947: Zimdahl and Kramer² postulated the origin in two focuses: supraventricular and ventricular because at least one QRS pattern complex could be abolished with the carotid massage.

1961: Agustin Castellanos Jr³, and later Sherf and Bonemann⁴ (**1967**) also suggest the single ventricular origin conducted with alternant aberration by bundle branch block.

1969: Mauricio Rosembaum Marcelo Elizari and Lazzari⁵ studied 13 patients and concluded that bidirectional tachycardia is supraventricular with a functional triphasic block: right bundle branch and alternant aberrant conduction in the two division of the left bundle branch. Additionally, these authors observed that the was perfectly rhythm regular and vagal stimulus responsive.

1. **Sherf D, Kisch D Ventricular tachycardia with variform ventricular complexes. Bull. New York M. Coll. 1939;3:73.**

2. Zimdahl WT, Kramer LI. On the mechanism of paroxysmal tachycardia with rhythmic alteration in the direction of the ventricular complexes. Am Heart J. 1947;33:218.

3. **Castellanos A Jr. The genesis of bidirectional tachycardias. Am Heart J. 1961 Jun; 61:733-739.**

4. Scherf D, Bornemann C. **Tachycardias with alternation of the ventricular complexes.** Am Heart J. 1967 Nov;74:667-674.

5. **Rosenbaum MB, Elizari MV, Lazzari JO. The mechanism of bidirectional tachycardia. Am Heart J. 1969 Jul;78:4-12.**

1973(April): Michael Rosen¹ demonstrated experimentally in dogs with microelectrodes techniques the effects of ouabain in toxic concentration on phase 4 of canine Purkinje fibers transmembrane potentials: Triggered Activity in phase 4 (delayed after depolarizations or DAD)¹.

1973 (June) Stafford I Cohen et al². demonstrated in human heart using ECG and His bundle potential the left ventricular origin and enhanced phase 4 depolarization on the divisions of the left bundle branch.

1975: First description associated with Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) and isoproterenol infusion³.

1976: first effective publication related purified digoxin-specific Fab fragments are capable of rapid reversal of advanced digoxin toxicity⁴.

1992(May): first description of BVT induced by herbal aconite poisoning⁵.

1. Rosen MR, Gelband H, Merker C, Hoffman BF. Mechanisms of digitalis toxicity. Effects of ouabain on phase four of canine Purkinje fiber transmembrane potentials. *Circulation*. 1973 Apr;47:681-689.
2. Cohen SI, Deisseroth A, Hecht HS. Infra-His bundle origin of bidirectional tachycardia. *Circulation*. 1973 Jun;47:1260-1266.
3. Reid DS, Tynan M, Braidwood L, Fitzgerald GR. Bidirectional tachycardia in a child: a study using His bundle electrography. *Br Heart J*. 1975;37:339-344
4. Smith TW, Haber E, Yeatman L, Butler VP Jr. Reversal of advanced digoxin intoxication with Fab fragments of digoxin-specific antibodies. *N Engl J Med*. 1976 Apr 8;294:797-800.
5. Tai YT, Lau CP, But PP, Fong PC, Li JP. Bidirectional tachycardia induced by herbal aconite poisoning. *Pacing Clin Electrophysiol*. 1992 May;15:831-839.

2004: first description of BVT associated with Andersen -Tawil – Syndrome¹.

2006: first description of BVT associated with fatty infiltration of the anterior right ventricular free wall².

2007: first description of BVT treated successfully with flecainide in Andersen-Tawil-Syndrome³.

2009: first description of BVT associated with acute myocardial infarction⁴.

1. **Pouget J, Philip N, Faugere G, Pellissier JF. Andersen syndrome: a particular form of paralysis with cardiac dysrhythmia Rev Neurol (Paris). 2004;38-42.**
2. **Haghjoo M, Arya A, Alizadeh A, Sadr-Ameli MA. Bidirectional ventricular tachycardia and fatty infiltration of the anterior right ventricular free wall. Cardiol Rev. 2006 Jul-Aug;14:e5-7.**
3. **Bökenkamp R, Wilde AA, Schaliij MJ, Blom NA. Flecainide for recurrent malignant ventricular arrhythmias in two siblings with Andersen-Tawil syndrome. Heart Rhythm. 2007 Apr;4:508-511.**
4. **Sonmez O, Gul EE, Duman C, Düzenli MA, Tokaç M, Cooper J. Type II bidirectional ventricular tachycardia in a patient with myocardial infarction. J Electrocardiol. 2009 Nov-Dec;42:631-632.**

BVT POSSIBLES CAUSES

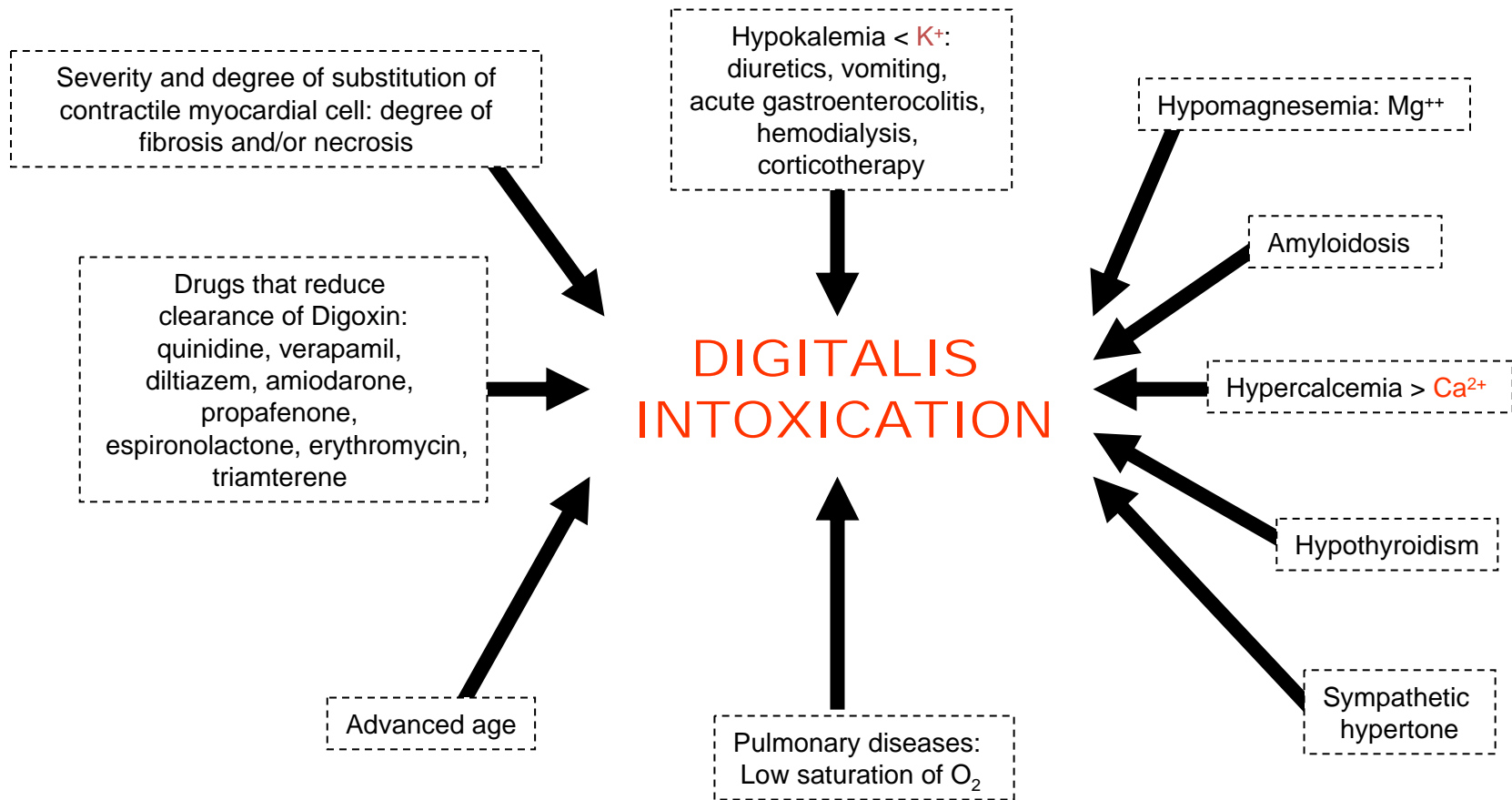
I) Acquired

1. Digitalis toxicity consequence of supra-therapeutic serum level of digoxin or digitalis poisoning
2. Digoxin-cyclosporine interaction in transplantation heart
3. Hypokalemia
4. Herbal aconitine poisoning
5. Isuprel infusion
6. Arrhythmogenic Right Ventricular Dysplasia (ARVD)
7. Hypertrophic Cardiomyopathy (HCM)
8. Dilated Cardiomyopathy (DCM)
9. Congenital heart disease
10. Myocarditis
11. Myocardial infarction/myocardial ischemia
12. Fatty infiltration of right ventricular free wall

II) Congenital familial-hereditary with structurally normal heart or channelopathies

1. Andersen-Tawil Syndrome hyperkalemic periodic paralysis
2. Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)
 - Autosomal dominant RYR2
 - Autosomal recessive CASQ2 gene mutation

FACTORS THAT PREDISPOSE DIGITALIS INTOXICATION AND INCREASE SENSITIVITY TO DIGITALIS



MANIFESTATIONS THAT SUGGEST DIGITALIS INTOXICATION

➤ MAIN CARDIAC ARRHYTHMIAS

- Sinus bradycardia.
- Sinus arrest.
- Blocks: of SA outflow, Ex.: Wenckebach.
- AV block of all degrees with predominance of first degree (conduction slowing and prolongation of refractory period in AV node), of outflow.
- AV dissociation: dominant suppression of pacemaker with passive escape of a low junctional focus or inappropriate acceleration of subsidiary pacemaker or more rarely, dissociation within the AV node proper.
- Atrial tachycardia with variable AV conduction: the most common one and almost pathognomonic.

MANIFESTATIONS THAT SUGGEST DIGITALIS INTOXICATION

➤ MAIN CARDIAC ARRHYTHMIAS

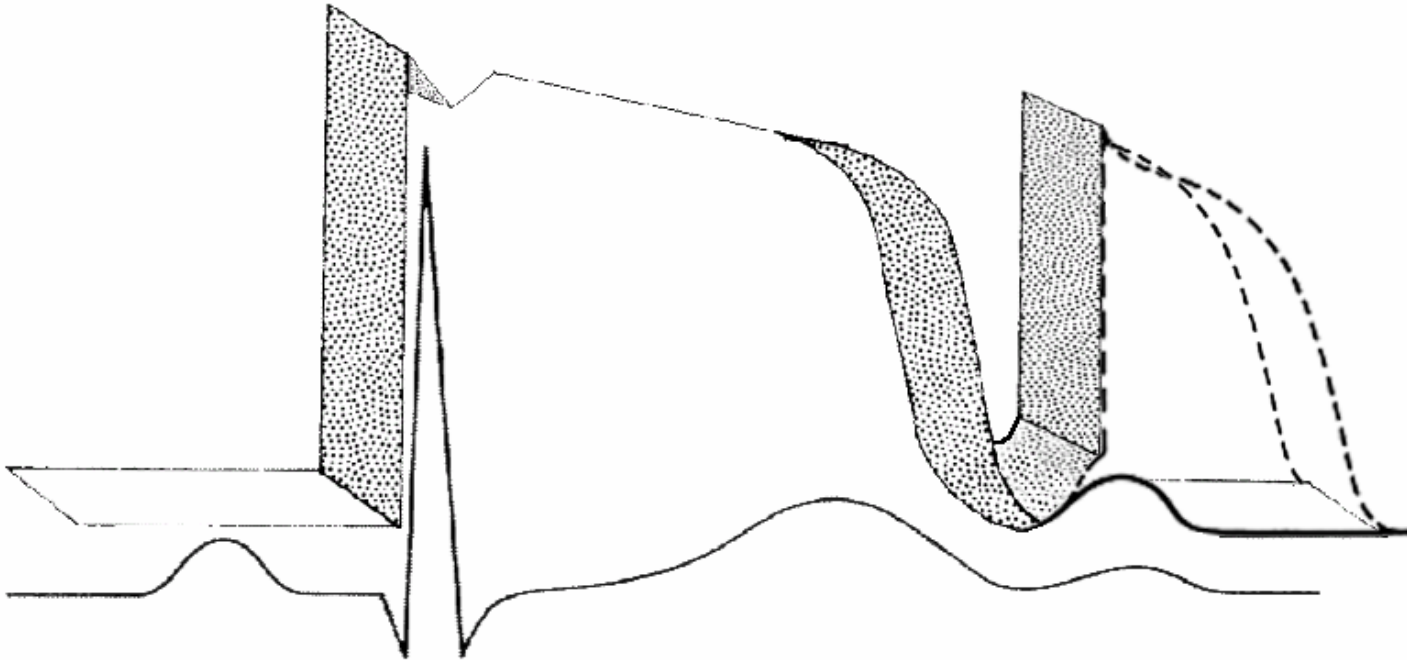
- Sudden appearance of atrial tachycardia during digitalis therapy in patient with AF.
- Accelerated junctional rhythm or nonparoxysmal junctional tachycardia with frequent isorhythmic dissociation.
- Isolated or bigeminal extrasystoles.
- Fascicular VT.
- AIVR or accelerated idioventricular rhythm.
- **Bidirectional (BVT) or bifascicular VT.**
- Ventricular flutter.
- Slow VF.

MANIFESTATIONS THAT SUGGEST DIGITALIS INTOXICATION

➤ EXTRACARDIAC

- **GASTROENTERIC:** anorexia, nausea, vomiting, diarrhea and abdominal pain.
- **NEUROPSYCHIATRIC:** headache, confusion, fatigue, dizziness, restlessness, delirium, lability of humor, sleepiness, psychosis, mental confusion, memory lapses, pseudo dementia.
- **VISUAL:** dyschromatopsia more common for red, green, and yellow.

BVT: ELECTROPHYSIOLOGICAL MECHANISM IN DIGITALIS INTOXICATION



Delayed Afterdepolarizations (DADs) triggered activity: they are oscillations of the membrane potential that occur after having completed phase 3 of AP or in phase 4. When they reach the limit, trigger a new AP. They are observed in high rates (tachycardia-dependent). Their mechanism is caused by the opening of the INa channel, sensitive to increased intracellular Ca²⁺ concentration. It is a stimulus for repetitive release of even more Ca²⁺ from sarcoplasmic reticulum. The causes of DADs include digitalis, catecholamine, hypercalcemia and rapid pacing.

Arrhythmias caused by DADs are PVCs, tachycardia related to digitalis excess, some catecholamine-dependent atrial and ventricular tachycardias, .

ECG CHARACTERIZATION O BVT

1. Rapid rhythm in which QRS morphology alternated so that the direction of the complexes is predominantly upward in one beat and downward in the next
2. Heart rate: between 140 to 180bpm
3. QRS duration: may be equal to, less than or greater than 120ms
4. Right Bundle Branch Block pattern is seen in V_1 lead.
5. In frontal plane is observed a QRS complex axis between -60° to -80° and $+120^\circ$.

ECG CHARACTERISTIC OF BVT SECONDARY TO DIGITALIS INTOXICATION

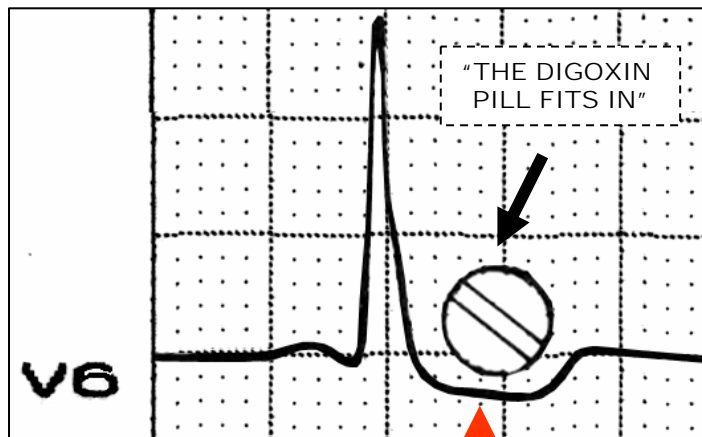
- 1) Heart rate between 140 and 180bpm
- 2) QRS duration <140 ms
- 3) RBBB pattern
- 4) QRS axis in frontal plane from right ($+120^\circ$) to left (-60°) alternatively

EPS: The absence of His bundle activity preceding the earliest onset of ventricular activation strongly favored a ventricular origin.

DIGITALIS EFFECT CONCEPT

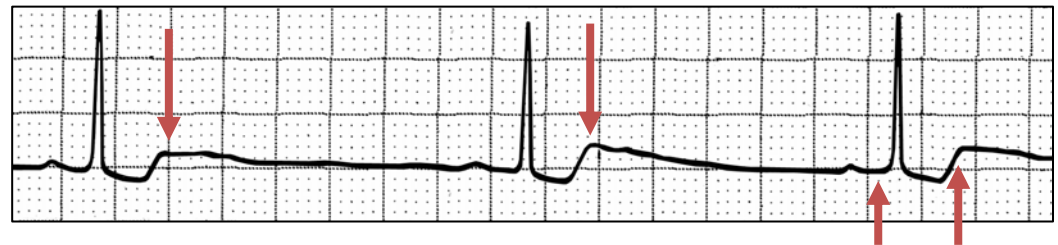
The earliest modification of digitalis effect on ECG or “digitalis action” are:

- 1) Prolonged PR interval.
- 2) ST segment: shortening and superior convexity (“in spoon”) by shortening of phases 2 and 3 of action potential (AP);
- 3) QT and QTc intervals shortening: main cause of acquired short QT;
- 4) T wave flattening with apiculate form of terminal portion in 10% of cases. Possible symmetrical inversion of T wave (pseudo-ischemic T wave);
- 5) Prominent U wave.

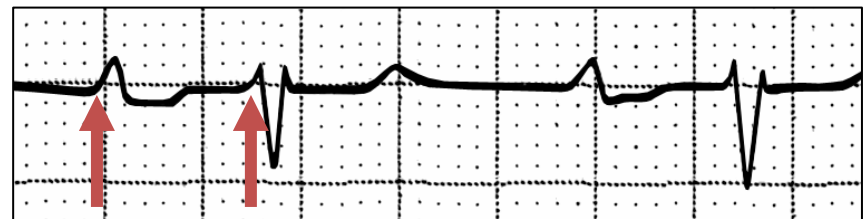


SHORT ST SEGMENT "IN SPOON"

APICULATE FORM OF TERMINAL PORTION OF WAVE



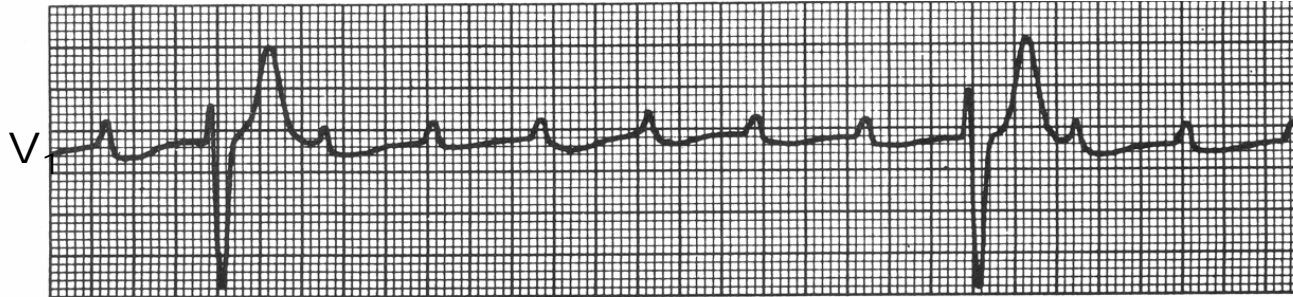
QTc INTERVAL SHORTENING



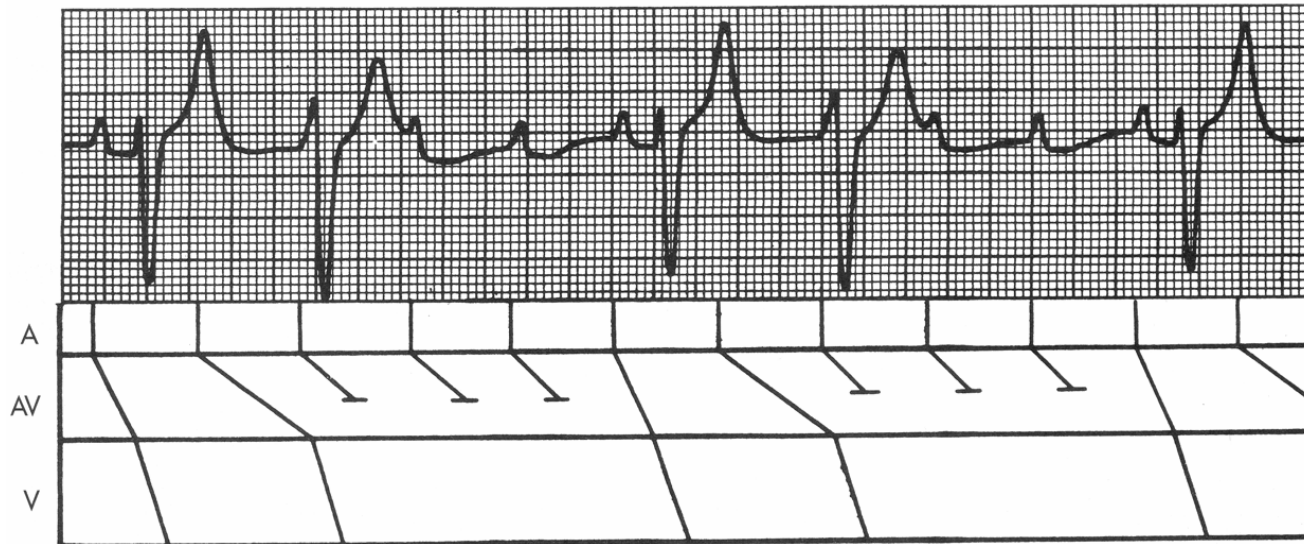
PROLONGED PR INTERVAL

VARIABLE HIGH-DEGREE AV BLOCK WITH 7:1 AND 5:1 CONDUCTION RATE BY ACUTE INTOXICATION BY DIGOXIN IN SUICIDE ATTEMPT

7:1 AV CONDUCTION RATE



5:1 AV CONDUCTION RATE

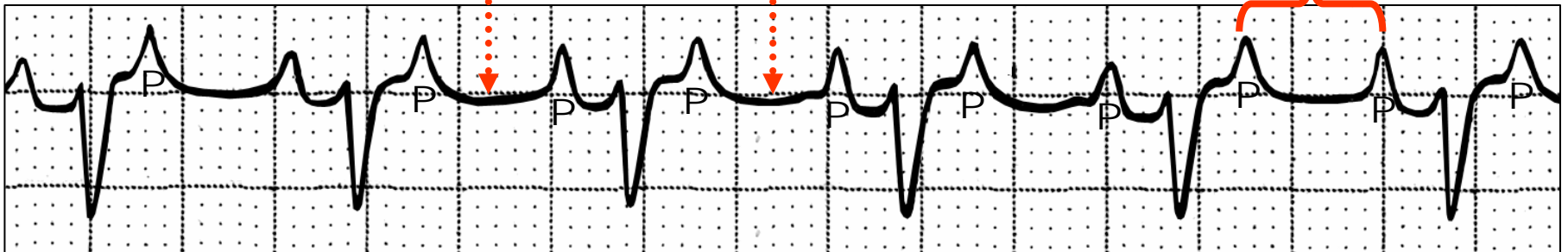


Example of high degree AV block, variable with conduction rate 7:1 and 5:1 by acute intoxication by digoxin in a patient who ingested 1 box of digoxin as a suicidal attempt.

ATRIAL TACHYCARDIA WITH 2:1 BLOCK

P WAVES SEPARATED BY
ISOELECTRIC LINE

P-P INTERVAL
214 bpm

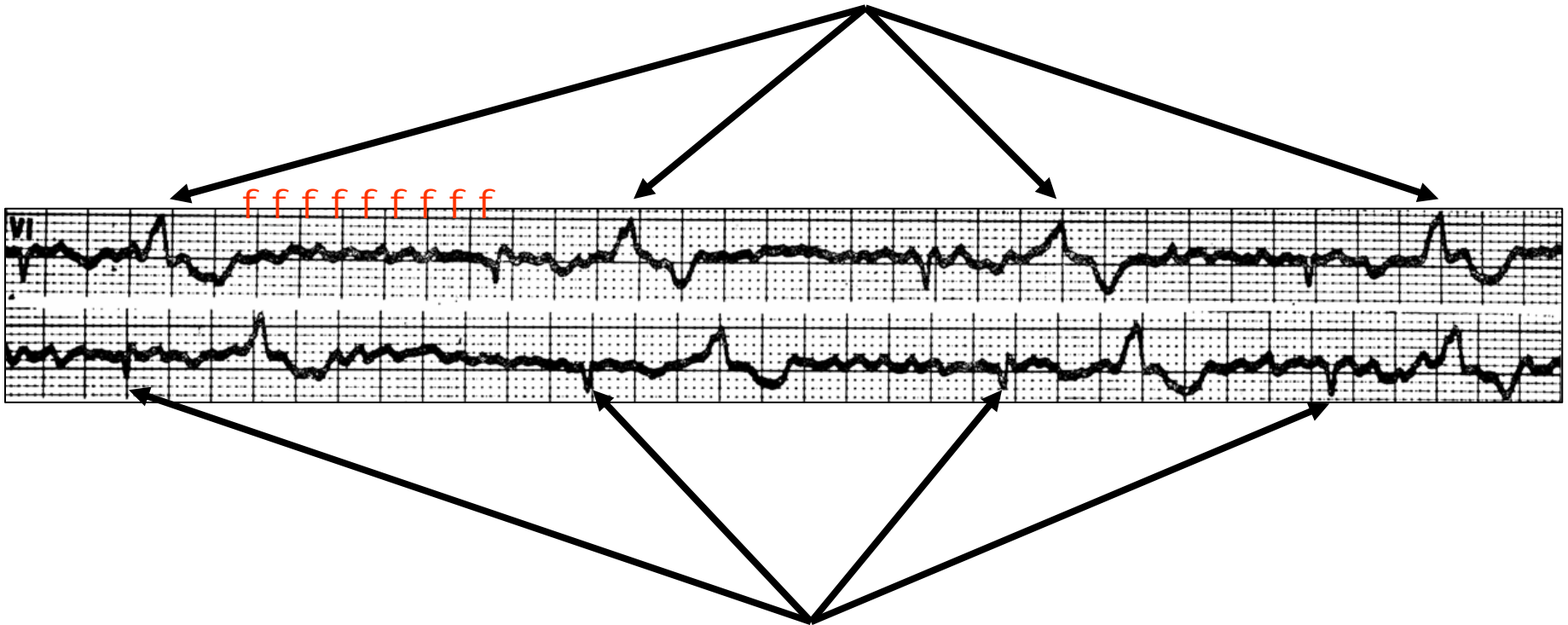


P-P interval rate: usually between 100 and 180 bpm.

- 1) Automatic form: 150 bpm to 250 bpm
- 2) By microreentry: between 130 and 150 bpm.

AF AND BIGEMINAL VENTRICULAR EXTRASYSTOLES

MONOFOCAL BIGEMINAL VENTRICULAR EXTRASYSTOLES

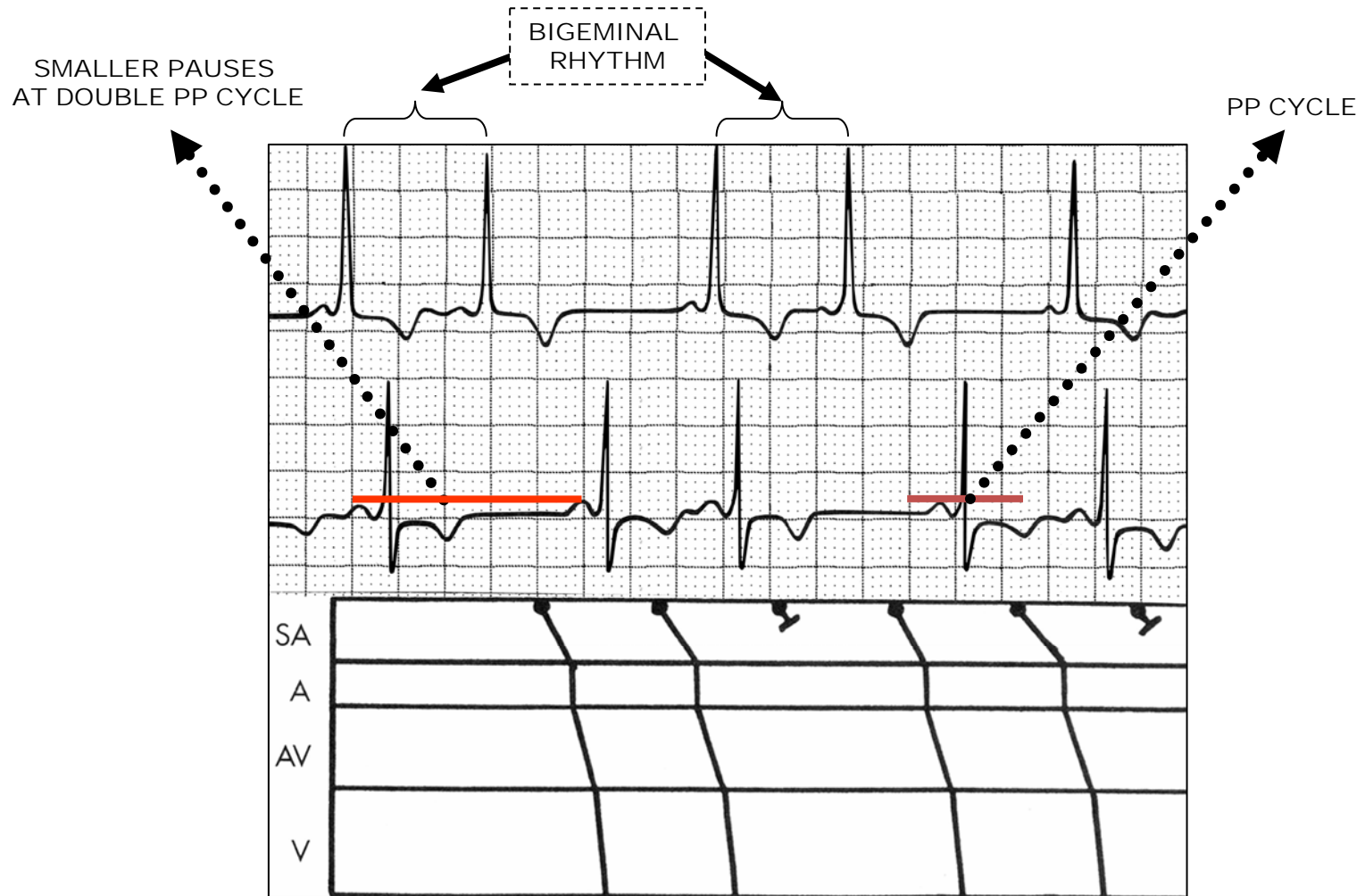


SMALL AND RELATIVELY NORMAL CONDUCTED BEATS

AF with slow and irregular ventricular response rate. Advanced 2nd degree AV block: typical of digitalis intoxication.

Example of atrial tachycardia with 2:1 block, considered atypical arrhythmia of digitalis intoxication.

SINUS RHYTHM WITH SINOATRIAL BLOCK 3:2 SINUS WENCKEBACH TYPE



Toxicity from the digitalis family of cardiac glycoside medications remains common. Successful treatment depends on early recognition; however, the diagnosis of potentially life-threatening toxicity remains difficult because the clinical presentation is often nonspecific and subtle.

The hallmark of cardiac toxicity is increased automaticity coupled with concomitant conduction delay. Though no single dysrhythmia is always present, certain aberrations such as frequent premature ventricular beats, bradydysrhythmias, paroxysmal atrial tachycardia with block, junctional tachycardia, and bidirectional ventricular tachycardia are common.

Treatment depends on the clinical condition rather than serum drug level. Management varies from temporary withdrawal of the medication to administration of digoxin-specific-antibody Fab fragments (Fab) purified from immunoglobulin G produced in sheep for life-threatening cardiovascular compromise. The dose of Fab fragments is equal to the amount of digoxin or digitoxin in the patient's body as estimated from medical histories or determinations of serum digoxin or digitoxin concentrations. First-line therapy with Fab fragments in patients with digitalis poisoning was associated with a low mortality rate¹. The most common side effect is rapid development of hypokalemia and exacerbation of congestive heart failure². Purified digoxin-specific Fab fragments are capable of rapid reversal of advanced digoxin toxicity. MgSO₄ is an useful therapeutic tool for the treatment of various ventricular tachycardias. Given at low rate (50 mg/min) and continued 2 hours after disappearance of arrhythmia, and infusion repetition at the same rate for 60-90 min twice daily for the next 3-4 days³. Lignocaine can be useful in the treatment of digitalis-induced bidirectional tachycardia. The drug is superior to other class I or class IV agents⁴.

1. Lapostolle F, Borron SW, Verdier C, Taboulet P, Guerrier G, Adnet F, et al. Digoxin-specific Fab fragments as single first-line therapy in digitalis poisoning. *Crit Care Med.* 2008 Nov;36:3014-18.
2. Antman EM, Wenger TL, Butler VP Jr, Haber E, Smith TW. Treatment of 150 cases of life-threatening digitalis intoxication with digoxin-specific Fab antibody fragments. Final report of a multicenter study. *Circulation.* 1990 Jun;81:1744-1752.
3. Perticone F, Pintaudi C, Cuda G, Spadea F, Tassone P, Mattioli PL. Magnesium salts in the treatment of ventricular tachycardia. *G Ital Cardiol.* 1987 Oct;17:857-864
4. Castellanos A, Ferreiro J, Pefkaros K, Rozanski JJ, Moleiro F, Myerburg RJ. Effects of lignocaine on bidirectional tachycardia and on digitalis-induced atrial tachycardia with block. *Br Heart J.* 1982 Jul;48:27-32.

BVT of Herbal Aconitine Poisoning

The rootstocks of aconitum plants have been commonly employed in traditional Chinese herbal recipes for "cardiotonic" actions and for relieving "rheumatism". Bidirectional tachycardia can be induced by aconites present in a Chinese herbal decoction consume. The tachycardia show marked susceptibility to vagotonic maneuvers, cholinesterase inhibition, and adenosine triphosphate. The incessant nature of the tachycardia, rapid recurrence after transient suppression, and failure to respond to direct current cardioversion suggested an automatic tachycardia mechanism consistent with known data on the cellular electrophysiological mechanism of aconitine-mediated arrhythmogenesis.

A fascicular or ventricular myocardial origin of the tachycardia with alternating activation patterns, or dual foci with alternate discharge, appeared most plausible. Multiple pitfalls could occur during the processing of these herbs that might have predisposed to aconite poisoning. The need for strict control and surveillance of herbal substances with low margins of safety is highlighted.

According to the fact that aconitine acts as a agonist of Na-channel receptor, antiarrhythmic agents which belong to Vaughan-Williams' classification I might be the first choice for the therapy of aconitine induced arrhythmias.

1. Tai YT, Lau CP, But PP, Fong PC, Li JP. Bidirectional tachycardia induced by herbal aconite poisoning. *Pacing Clin Electrophysiol.* 1992 May;15:831-819.
2. Tsukada K, Akizuki S, Matsuoka Y, Irimajiri S. A case of aconitine poisoning accompanied by bidirectional ventricular tachycardia treated with lidocaine *Kokyu To Junkan.* 1992 Oct;40:1003-1006.

BVT of CPVT

1. Children or young people
2. Familial history of syncope or sudden death in 30%
3. mechanisms of arrhythmias associated to impaired Ca^{2+} regulation homeostasis¹
4. Unexplained syncope. Unmasking false epilepsy
5. Absence of structural heart disease
6. Autosomal dominant mutations in the cardiac ryanodine receptor (RyR2) or recessive Mutation on calsequestrin 2 (CASQ2) have been reported².
7. Normal QT interval³.
8. Unexplained syncopal episodes, or sudden death⁴ even when the resting ECG is normal.
9. Stress- or emotion-induced syncope related to PVT or BVT.
10. Induced by any form of increasing adrenergic stimulation (adrenergic-dependence) could be induced by increasing the heart rate by atrial pacing or isoprenaline administration.
11. His bundle electrography show that the arrhythmia is ventricular in origin.
12. Therapy: appropriate β -blocker associated with ICD

1. Cerrone M, Napolitano C, Priori SG. **Catecholaminergic polymorphic ventricular tachycardia: A paradigm to understand mechanisms of arrhythmias associated to impaired Ca^{2+} regulation.** Heart Rhythm. 2009 Nov;6:1652-1569.
2. Beery TA, Shah MJ, Benson DW. **Genetic characterization of familial CPVT after 30 years.** Biol Res Nurs. 2009 Jul;11:66-72.
3. Lee SY, Kim JB, Im E, Yang WI, Joung B, Lee MH, Kim SS. **A case of catecholaminergic polymorphic ventricular tachycardia.** Yonsei Med J. 2009 Jun 30; 50:448-451.
4. Jalife J. **Ventricular arrhythmias of catecholaminergic origin and sudden death** Arch Cardiol Mex. 2007 Oct-Dec; 77 Suppl 4:S4-175-177.



Femenine, white 20 years old. Repetitive stress- or emotion-induced syncope's episodes. Autosomal dominant mutations in the cardiac ryanodine receptor (RyR2) . ECG QRS axis from -60° to $+120^{\circ}$.beat to beat. QRS pattern with RBBB and LBBB patters in alternating fashion.

BVT of Andersen-Tawil-Syndrome (ATS)

1. It is a potassium channelopathy with autosomal-dominant inheritance
2. Mutation of KCJN2 gene which encodes the alpha-subunit of the strong inward-rectifier Ba²⁺-sensitive potassium channel I_{k1} coding for Kir 2.1 protein with important roles in maintaining membrane potential and during the terminal phase of cardiac action potential repolarization (phase 3 and 4).
3. includes a clinical triad: periodic paralysis (episodic muscle weakness), cardiac arrhythmia with syncope or sudden cardiac death¹ and usually mild but diagnostically useful dysmorphic facial/skeletal features.
4. Cardiac manifestations are variable and may include a long QT syndrome(LQT7), PVCs, complex ventricular ectopy, and polymorphic or BVT.
5. Potential neuropsychiatric system involvement.
6. Treatment with a combination of spironolactone, amiloride, and potassium supplements². The class IC antiarrhythmia drug flecainide has been shown to depress ventricular ectopy and improve exercise capacity in patients with ATS^{3;4}. Imipramine therapy had a positive effect on arrhythmia⁵.

1. Airey KJ, Etheridge SP, Tawil R, Tristani-Firouzi M. Resuscitated sudden cardiac death in Andersen-Tawil syndrome. *Heart Rhythm*. 2009 Dec;6:1814-1817.
2. Kim JB, Chung KW. Novel de novo mutation in the KCNJ2 gene in a patient with Andersen-Tawil syndrome. *Pediatr Neurol*. 2009 Dec;41:464-466.
3. Platt D, Griggs R. Skeletal muscle channelopathies: new insights into the periodic paralyses and nondystrophic myotonias. *Curr Opin Neurol*. 2009 Oct;22:524-531.
4. Pellizzón OA, Kalaizich L, Ptáček LJ, Tristani-Firouzi M, Gonzalez MD. Flecainide suppresses bidirectional ventricular tachycardia and reverses tachycardia-induced cardiomyopathy in Andersen-Tawil syndrome.
5. *J Cardiovasc Electrophysiol*. 2008 Jan;19:95-97.

Colleagues Commentaries

This could be a case of bidirectional VT typical arrhythmia of CMPVT and can be associated with digitalis intoxication maybe during hypokalemia during furosemide therapy.

Last time we described a fulminated myocarditis in Kardiol. Pol . in female with EF % of about 15% during digoxin therapy

(Fulminant myocarditis complicated with cardiogenic shock and various tachyarrhythmias)

Kukla P, Paruzel M, Plato A, Malarczyk-Zaher B, Bromblik A, Szczuka K. Kardiol Pol. 2006 Feb;64:208-211; discussion 212.

Best wishes from Poland

Piotr Kukla, MD, PhD

Dear Prof. Riera and All

This is a case of bidirectional ventricular tachycardia mostly due to Digoxin toxicity.

Immediate therapy with LIDOCAINE [others prefer PHENYTOIN] and evaluation of K level. FAB, if available, is the drug of choice for malignant arrhythmias induced by digoxina toxicity.

Digoxin toxicity is precipitated by hypokalemia and the toxicity by itself can cause hyperkalemia.

Raed Abu Sham'a, M.D

Querido Professor Riera e todos:

Este é um caso de taquicardia bidireccional em sua maioria causado por intoxicação digitálica.

Imediata terapia com lidocoaina (outros preferem fentoína) e avaliação do nível sérico de potássio. FAB se estiver disponível é a conduta de escolha em caso de arritmias malignas causadas por intoxicação digitálica.

A toxicidade digitálica é precipitada pela hipopotassemia e a toxicidade por sí mesma pode causar hiperpotassemia.

Andres, I will allow you to circulate this to the group with your usual elegant additional explanations. With the patient on digoxin and having a significant underlying cardiomyopathy, this is a bidirectional ventricular tachycardia consistent with digitalis intoxication. At least, this is my first diagnosis. There is an excellent example published recently in the Journal of the American College of Cardiology (Images in Cardiology) by Drs. Richter and Brugada, JACC 2009; 54: issue 13. I would request a serum digoxin level and also consider starting the patient on Digibind, if available, even before the serum digoxin level returns. Towards the end of the anterior precordial lead recordings, there are two other morphology PVCs as a couplet. With the multifocal PVCs in this setting, I believe that this is digitalis toxicity until proven otherwise and the patient is at a particularly high risk of degenerating to ventricular fibrillation.

If this was a young patient without identified structural heart disease, particularly if associated with exercise, consider CPVT. There are also reported cases in the absence of digitalis associated with a severe underlying cardiomyopathy. This can also be a supraventricular tachycardia with alternating rate-dependent bundle branch block. I have attached some brief power point presentations that can be shared with the group. ECG Case Study 0029 shows non-conducted APBs and both RBBB and LBBB functional (Ashman's) aberrant conduction. Case 0028 is various manifestations of Ashman's Aberration and Case 0080 is classic Tachycardia (Phase 3) dependent BBB.

If there was a baseline 12 lead ECG and one of the repetitive complexes was consistent with the patient's intrinsic sinus or supraventricular conducted QRS complex, then this could be ventricular bigeminy with a PVC alternating with a conducted QRS. It would help if I could identify P waves but with the very brief rhythm strips associated with each lead of the 12 lead tracing and the alternating morphology of the QRS complex, I am unable to identify a distinctive P wave or repetitive distortion in the ST-T wave to identify AV dissociation or even confirm that one of the QRS complexes is sinus and the other is a PVC.

Another cause of “electrical alternans” is a pericardial effusion with tamponade but this is usually seen best in one or two leads with minor variation in the other leads and the clinical history is likely to be different.

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Vice President, Medical Services
Tel: 1-818-493-2900 Fax: 1-818-362-2242
[plevine@sjm.com/](mailto:plevine@sjm.com)**

Andres,
Eu permitirei a você circular este para o grupo com seu elegante habituais explicações adicionais. É um paciente em uso de digoxina que tem como doença subjacente uma cardiomiopatia. Isto é uma taquicardia ventricular bidirecional compatível com intoxicação digitálica. Pelo menos, este é meu primeiro diagnóstico. Existe um exemplo excelente publicado recentemente no Diário da Academia Americana de Cardiologia (Imagens em Cardiologia) por os Drs. Richter e Brugada, JACC 2009; 54:13.

Eu solicitaria uma dosagem sérica de digoxina e também consideraria começar com Digibind, se disponível, mesmo antes de ter o resultado da digoxinemia. Nas derivações precordiais existem duas morfologias. Com a presença de extrasístoles polifocais o paciente encontra-se em um risco alto de degenerar para fibrilação ventricular.

Se fosse um paciente jovem sem doença estrutural, particularmente se associado com exercício, consideraria o diagnóstico de taquicardia ventricular polimórfica catecolaminérgica. Existem casos reportado na ausência de digital associada a severa cardiomiopatia subjacente.

Este evento poderia ser uma taquicardia supraventricular com bloqueio de rama alternante dependente da frequência (Ashman). Eu ajuntei algumas apresentações que poderão ser compartilhadas com o grupo condução aberrante.

Outra causa de “alternância elétrica” é o derrame pericárdico com taponamento mas isto é normalmente visto melhor em uma ou duas derivações com mínima variação em outras derivações e a história clínica seria diferente.

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Andres:

The patient has severe digitalis toxicity compounded by low K+.

Rx:

Discontinue all medications, start an IV and give intravenous K+ immediately, but cautiously, (no more than 40 mEq/hour), initiate cardiac monitoring with continuous professional observation. Prayer.

EKG Dx:

Patient has a full constellation of Digitalis toxicity effects. Supraventricular tachycardia approaching 300/minute...very dangerous in this severely compromised patient.

Bidirectional tachycardia, at least one source being ventricular and the other probably Junctional. The supraventricular origin shows Left Axis Deviation.

Occasional PAC's and multifocal ventricular extrasystole's. AV node only minimally blocked, SA node probably overdrive suppressed by the SV tachycardia.

Note: great ST elevation in extrasystole's of ventricular origin typical of Marriotts' ventricular extrasystole "snapshot effect"; the signs of ischemia most likely due to the high ventricular rate effect (i.e., relative ischemia due to pulse rate up to 300/minute) outstripping the metabolic demands on non-infarcted (somewhat normal) but severely stressed myocardium.

Dale Dubin M.D.

Dear Andres,

In principle it is a bidirectional TV, and using digoxine, the possible cause would be the digitalic poisoning. The question would be if the expanded cardiomyopathy is of well-known origin and the arrhythmia is originating of its same cardiopathy associated to the digitalic poisoning. If it were suspended to the digitalis and the arrhythmia it continued would be possible to be thought about the syndrome of Andersen-Tawil (QTL) that does not have expansion Generally I SAW. We had a case that the TVB generated a taquimiocardiopatía that reverted when flecainida dealing itself with. To this we published it case in JCE (Flecainide Suppresses Ventricular Bidirectional Tachycardia and Reverses Tachycardia-Induced Cardiomyopathy in Andersen-Tawil Syndrome Journal of Cardiovascular Electrophysiology. Volume 19, Issue 1, Date: January 2008, Pages: 95-97). This it is the small contribution that I can do. Greetings.

Estimado Andrés,

En principio es una TV bidireccional, e ingiriendo digoxina, la posible causa sería la intoxicación digitalic. La cuestión sería si la miocardiopatía dilatada es de origen conocido y la arritmia es proveniente de su misma cardiopatía asociada a la intoxicación digitalic. Si se suspendiera la digital y la arritmia continuara se podría pensar en el síndrome de Andersen-Tawil (QTL) que Generalmente no tiene dilatación VI. Nosotros tuvimos un caso que la TVB generó una taquimiocardiopatía que revirtió al tratarse con flecainida. Este caso lo publicamos en JCE (Flecainide Suppresses Bidirectional Ventricular Tachycardia and Reverses Tachycardia-Induced Cardiomyopathy in Andersen-Tawil Syndrome Journal of Cardiovascular Electrophysiology. Volume 19, Issue 1, Date: January 2008, Pages: 95-97).

Este es el pequeño aporte que puedo hacer. Saludos.

Oscar Pellizón MD.

Las veces que he visto este tipo de taquicardias es en libros, como ejemplo de arritmias provocadas por intoxicación digitálica (taquicardia bidireccional) lo cual condice con el cuadro de náuseas y vómitos, si tengo como medir digoxemia sería el primer estudio a hacer, evaluación iónica y función renal, suspensión de digoxina y si tengo disponible, anticuerpos antidigoxina. Si la arritmia le esta generando fallo hemodinamico, se puede probar con lidocaína o con difenilhidantoina (fenitoina).

Ignacio Retamal
Córdoba

Need to see previous ECG. Is this atrial fibrillation?
Somewhat atypical bidirectional VT, usually caused by Digital.
Blood dig level?

Boris Surawicz

Necessário ver ECG prévio. Estava em fibrilação atrial?
Um tanto Taquicaridia ventricular bidireccional, usualmente ocasionada por digital
Como estava o nível sérico?

Boris Surawicz.

Dear Andres.

In my two cents this is a beautiful demonstration of digitalis toxicity on ECG: bidirectional ventricular tachycardia alternating from regions nearby the anterior and posterior fascicles of left ventricle.

Many thanks to you and to Dr. Raimundo Barbosa Barros for sharing this very interesting ECG!

Li Zhang M.D.

Querido Andrés.

Na minha experiencia isto é uma bonita demonstração de toxicidade digitálica no ECG: A taquicardia ventricular bidirecional alternando a condução pelo fascículo anterior e posterior do ramo esquerdo.

Muito obrigado para você e para o Dr. Raimundo Barbosa Barros por compartilhar este muito interessante ECG!

Suspender la digital, administrar sales de potasio, dosar digoxinemia. Sólo si no cede el cuadro con estas medidas administraría amiodarona.

Saludos.

Luciano Pereira Ciudad del Este República del Paraguay

Suspender a digital, administrar sais de potássio, fazer a dosagem de digoxinemia. Apenas se não reverter o quadro com estas medidas administrar amiodarona.

Saudações

Luciano Pereira Cidade del Este República Oriental do Paraguai.

Suspend the digital, manage potassium salts, make the seric digoxin dosage. Just if do not revert the picture with these measured then to administer amiodarone.

Greetings

Luciano Pereira. del Este City Paraguay
