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Title: A REVIEW OF ATRIAL FIBRILLATION FOR THE GENERAL PAEDIATRICIAN

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Abstract:
Paediatric atrial fibrillation is a rare entity in the absence of congenital heart disease as children are unlikely to have the structural and functional changes in their myocardium to sustain the arrhythmia. Any child presenting with this arrhythmia needs to be carefully evaluated for concealed cardiac pathology such as cardiomyopathy or inherited arrhythmia syndromes. Atrial fibrillation leading to a haemodynamically unstable patient is rare and should prompt synchronised cardioversion, while stable patients can be discussed with a paediatric cardiologist. Tachycardia-induced cardiomyopathy and thromboembolism are possible complications of sustained atrial fibrillation and anticoagulation is usually indicated to prevent the latter. Risk of atrial fibrillation increases with age and body mass index.

Obesity and athletics are known risk factors and recurrence can be seen even in the absence of any identifiable underlying pathology.

Key Points:

- Every child presenting with atrial fibrillation needs discussion with a paediatric cardiologist and careful evaluation for underlying cardiac pathology.
- Atrial fibrillation in Wolff-Parkinson-White syndrome can lead to ventricular fibrillation.
- Atrial fibrillation may be associated with supraventricular tachycardia due to both tachycardia-induced remodelling, and because it can be triggered by adenosine administration.

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Hypothetical Case Study 1

A 16-year-old overweight boy attending a general paediatric clinic for difficulties at school takes no medications and has no significant past medical history. On examination he looks well, is breathing comfortably, his pulse rate is 86 beats per minute and irregular, he is slightly hypertensive and well perfused. Further physical examination is unremarkable. An electrocardiogram (ECG) is performed and demonstrates atrial fibrillation with narrow QRS complexes except one with slightly different morphology (Figure 1).

Hypothetical Case Study 2

A 13-year-old girl with history of supraventricular tachycardia (SVT) presents to the emergency department with 90 minutes of palpitations. She is on no medications and can usually revert her SVT with vagal manoeuvres at home. Her heart rate is 200, she is mildly hypotensive, and focussed physical examination is normal. An ECG shows a narrow complex tachycardia (Figure 2). Escalating doses of adenosine transiently slow the heart rate though are unsuccessful in terminating the tachycardia.

Hypothetical Case Study 3

A 14-year-old previously well boy is admitted to the paediatric ward of a hospital for observation and workup after a first episode of seizure. During the admission he has a brief syncope after which an ECG is performed and demonstrates atrial fibrillation, which two hours later spontaneously reverts to sinus rhythm. Unfortunately, he was not followed up, and the child later presented to hospital with a dilated cardiomyopathy and a combination of atrial fibrillation, atrial flutter, and multifocal atrial tachycardia (Figures 3-5).

Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia in adults, though remains very rare in children (1, 2). When seen in childhood it is more common in adolescents than in younger children and is often associated with a predisposing underlying cardiovascular condition. It can be seen in children with structurally and functionally normal hearts, termed “lone AF” (3). This descriptor is potentially misleading and strict definition difficult as in patients with traditionally lone AF, there may be a latent underlying trigger that can be identified and treated (2, 4). Untreated atrial fibrillation can lead to tachycardia-induced cardiomyopathy (TIC), thromboembolism, and if associated with Wolff-Parkinson-White (WPW) syndrome, has the potential to cause sudden cardiac death (5).

Given its rarity in the paediatric population there is lack of large-scale data to direct investigation and treatment of children with AF, with many management recommendations based on expert consensus and extrapolated from data in adult populations. With increasing numbers of children with congenital heart disease and with the high prevalence of childhood obesity which is a known risk factor for paediatric lone AF (6), it is possible we may encounter this arrhythmia more commonly in paediatric practice. While early involvement of the paediatric cardiologist is necessary, initial recognition and immediate management may be required by the general paediatrician. Additionally, knowledge of risk factors for paediatric AF is essential to know in which patients a rhythm evaluation may be recommended, and primary prevention instituted.

Classification & Clinical Presentation

Atrial fibrillation is classified clinically by its duration (7-9), being divided into:

1. Paroxysmal (self-terminating, usually within 48 hours though can last up to 7 days)
2. Persistent (lasting more than 7 days though can be terminated by pharmacological or electrical cardioversion)
3. Long-standing persistent (Continuous AF lasting for ≥ 1 year)
4. Permanent (reversion to sinus rhythm is impossible)

Strict classification in practice may be impossible as duration prior to presentation may be ambiguous, and after cardioversion, it is impossible to know if it would have persisted more than 7 days. The classification of AF relates to the underlying pathophysiology with patients initially likely to experience paroxysmal AF that with time progresses to persistent and ultimately permanent. Children who present with AF are much more likely to have paroxysmal forms (10). Young people are also more likely to present with symptoms (mostly palpitations or atypical chest pain in > 95-98%) (3, 10) at the time of diagnosis, possibly related to their better atrioventricular (AV) conduction and therefore faster ventricular response (11), though asymptomatic presentations are possible.

Pathophysiology

Atrial fibrillation is a supraventricular arrhythmia defined by rapid uncoordinated atrial activation leading to ineffective atrial contraction with variable AV conduction (12). Focal ectopic firing and reentrant activity are the primary AF arrhythmia mechanisms (13). The

intricate details of these mechanisms are beyond the scope of this review. For a summary the reader is directed towards reviews by Iwasaki and colleagues (8) and Wijesurendra and Casadei (14). Here we will instead focus on those aspects that are essential towards understanding of disease presentation, aetiology, and management.

The existence of atrial fibrillation requires two basic components: a trigger (or driver) to initiate the arrhythmia, and an atrium (substrate) that is susceptible to its maintenance (14). AF can be induced in a child with a normal heart during an electrophysiology study by very rapid repetitive stimulation of the atrium from a catheter within the atrium. Without underlying pathology, the child's heart will usually sustain AF for a short time (a few seconds to five minutes) before spontaneously reverting back to normal sinus rhythm. Thus, a rapidly firing ectopic focus within the atrium can initiate AF, though is not sustained without structural or functional changes within the atrium. In adults the pulmonary veins are the usual location of focal ectopic firing driving AF initiation, and this has also been demonstrated in children though other foci are possible (15). Electrical isolation of areas of ectopic firing by catheter ablation is a potential treatment to prevent AF recurrence, and though very successful in younger populations with paroxysmal AF (10, 15), rarely recommended in children.

Reentry maintenance can result from either a shortened refractory period, or slowed conduction velocity (16). Functionally altered conduction is seen in patients with inherited channelopathies and under the influence of certain drugs or medications, while structural causes of altered conduction include atrial inflammation, previous surgery, and atrial fibrosis. Atrial fibrosis plays an important role in creating conduction speed heterogeneity downstream in the disease course regardless of initial aetiology and has become a target of "upstream therapy" for AF in adults. In addition to altered conduction, atrial enlargement permits the existence of more reentry circuits, hence structural heart disease resulting in atrial enlargement predominates in the causes of AF. Lastly, AF itself (as well as other atrial tachycardias) produces remodelling of atrial electrical properties that promote functional reentry which in part explains why atrial fibrillation becomes harder to revert with time (16).

The autonomic innervation of the heart also contributes to AF induction with simultaneous sympathovagal nerve discharges in dogs immediately preceding AF paroxysms. The pulmonary vein and left atrial junction is rich in autonomic innervation with stimulation of

these ganglionic plexi resulting in AF and their destruction via radiofrequency ablation reducing AF inducibility (17). Adenosine administration can also induce atrial fibrillation in children with its mechanism related to its autonomic actions (18).

Aetiology and Risk Factors

While the list of causes and risk factors for AF in children (see Table 1) may appear similar to that for adults, the probability of a particular aetiology being responsible is vastly different (11, 19). Most paediatric cases of AF are associated with congenital heart disease (CHD), a cardiomyopathy or WPW. So-called "lone AF" in children can be an early manifestation of a cardiomyopathy or unrecognised channelopathy, caused by isolated atrial myocarditis, triggered by an alternative supraventricular tachycardia, or be associated with any of the genetic and extracardiac causes of AF. True lone AF remains a diagnosis of exclusion that is only made after careful workup and evaluation (10).

Diagnosis and Acute Management

The diagnosis of atrial fibrillation is made on ECG: Irregularly irregular RR intervals and no discernible, distinct P waves. By convention an episode lasting more than 30 seconds is diagnostic (7). If the diagnosis is suspected on cardiac monitoring or telemetry, it is imperative to perform a full 12 or 15-lead ECG to establish the diagnosis unless there is haemodynamic instability requiring immediate treatment. Unstable patients should be promptly treated with synchronised DC cardioversion 1 J/kg, followed by 2 J/kg if necessary, preferably with anterior-posterior positioning of the pads (20, 21). If cardioversion is unsuccessful and haemodynamic instability remains, immediate paediatric intensive care and paediatric cardiology consultation should be obtained, and the alternative diagnosis of chaotic multifocal atrial tachycardia (MAT) should be considered. Regardless of cardioversion success, acute non-cardiac conditions associated with AF should be promptly considered, identified and treated (hypertension, hyperthyroidism, pulmonary embolism, viral infections, sepsis, drug overdose) (10).

The usually encountered scenario is for the child to appear well and haemodynamically stable. In this case, a full assessment should be performed including history, examination, and initial investigation, followed by paediatric cardiology consultation. Symptoms such as chest pain, palpitations, dyspnoea, presyncope, and syncope should be elicited in older children and pallor, increased work of breathing, poor feeding, and cyanosis in neonates, with emphasis on

onset to estimate the likely duration of AF prior to presentation. If there is no condition in the child's past medical history to suggest a predisposition to develop atrial fibrillation (see Table 1), then symptoms suggesting a possible aetiology should be elucidated. Precipitating factors such as recent or current infection, caffeine intake, alcohol or drug use, and medication ingestion should be screened for, and predisposing factors such as obesity and excessive endurance exercise training should be considered. A family history of AF, inherited cardiac arrhythmia, syncope, and sudden unexplained death (including car accidents on a straight road, or drowning in competent swimmers) should be obtained. A full examination should be performed with focus on signs of heart failure and thromboembolism. Suspicion for thromboembolism should be heightened in patients with structural heart disease as they are at increased risk.

ECG should be performed both in AF and in sinus rhythm if spontaneous reversion occurs or cardioversion is performed. The ECG in sinus rhythm should be assessed for: pre-excitation, early repolarisation, Brugada syndrome, long QT, short QT, and frequent ectopy. An ECG with elevated anterior chest leads can be performed in sinus rhythm to look for evidence of Brugada syndrome (22). If a child has a history of WPW or if pre-excitation is seen on a past or current ECG, then continuous monitoring should be performed due to the potential for AF to precipitate ventricular fibrillation (VF) if the accessory pathway has rapid antegrade conduction (Figure 6) (5). It is possible for a wide QRS complex to be seen in AF with rapid AV conduction due to rate-related bundle branch block, or due to ventricular ectopy, so not all wide QRS complexes are necessarily pre-excited. If a wide QRS complex follows a short RR interval and was preceded by a long RR interval, it is likely of supraventricular origin with rate-related bundle branch block, which is benign and termed Ashman phenomenon. However if wide QRS complexes are seen and are considered of ventricular origin, the concern for a cardiomyopathy or channelopathy should be high. Lastly, adenosine is not indicated in AF, though given that reentrant SVT is much more common in paediatrics, AF with rapid ventricular response may be misdiagnosed as such. It is important in any child with SVT to obtain a 12-lead ECG any time adenosine is given if possible.

Initial blood investigation should ideally include a blood gas, full blood count, electrolytes urea and creatinine, calcium magnesium phosphate, liver function tests, thyroid function studies, and a blood culture if history of fever. A chest x-ray may be appropriate if there is suspicion of lower respiratory tract infection or structural cardiac pathology. After initial

investigation has been performed consultation with a paediatric cardiologist is required for further management.

Further Management

A detailed summary of further managing paediatric AF is beyond the scope of this review, however a general approach is outlined. An echocardiogram, exercise testing, Holter monitoring, and consideration of electrophysiology study and/or cardiac MRI will usually be undertaken to determine an underlying cause with disease-specific management instigated if pathology found (10). Paediatric patients will usually be managed with rhythm control, so consideration of pharmacological versus electrical cardioversion will be made (11). Prevention of thromboembolism is necessary if the arrhythmia has persisted for more than 48 hours or the duration is unknown, or alternatively and more commonly done in paediatrics, a transoesophageal echocardiogram can be performed prior to cardioversion to rule out intracardiac thrombus (7, 9, 12). The risk of recurrence for AF is related to the underlying pathology if found. In an analysis of over 1500 cases of lone paediatric AF, El-Assaad and colleagues found a recurrence rate of 15% within 1 month, 19% within 3 months, 21% within 6 months, and 23% within 12 months, with recurrence rates increasing with age (6). Therefore, even in lone AF careful follow up is essential.

Case Study Discussion

The case studies presented at the beginning are examples of first presentation of AF in childhood. In all three cases the efforts should be directed towards finding signs of an underlying aetiology, assessing potential complications and involving a paediatric cardiologist or electrophysiologist.

First case: Pre-excitation is important to consider when AF is associated with broad QRS complexes given the potential for VF if the accessory pathway conducts rapidly. However, this broader QRS complex is likely due to rate related aberration given the overall rate is slow and that particular QRS complex follows shortly after the preceding complex (Ashman phenomenon). It is also important to recognise that obesity and hypertension are risk factors for AF and lifestyle modification should be encouraged.

The second case demonstrates the association of paediatric AF with other supraventricular tachycardias and serves as a reminder to both examine the ECG during adenosine administration (as adenosine might convert a reentrant tachycardia to AF), as well as

reconsider the diagnosis if routine management fails. ECG performed during adenosine administration revealed the previously concealed atrial fibrillation (see Figure 7).
The third case study is a reminder that paediatric AF can be the initial presentation of inherited arrhythmia syndromes such as CPVT, the diagnosis of which must not be missed as the next manifestation of the disease may be with tachycardia-induced cardiomyopathy or even sudden cardiac death.

Conclusion

While the incidence of paediatric AF remains very low, its recognition and knowledge about underlying diseases are increasingly important. Previously unrecognised cardiomyopathies, inherited cardiac arrhythmia syndromes, myocarditis and WPW are potentially lethal if not adequately treated. Children may present well-looking with brief paroxysmal AF, however all need paediatric cardiology/electrophysiology consultation. The final point to emphasise is that lone AF remains a diagnosis of exclusion and in itself has a relatively high recurrence rate so follow up is essential.

References

1. Strieper MJ, Frias P, Fischbach P, Costello L, Campbell RM. Catheter ablation of primary supraventricular tachycardia substrate presenting as atrial fibrillation in adolescents. Congenit Heart Dis. 2010;5(5):465-9.
2. Calkins H, Hindricks G, Cappato R, Kim YH, Saad EB, Aguinaga L, et al. 2017 HRS/EHRA/ECAS/APHRS/SOLAECE expert consensus statement on catheter and surgical ablation of atrial fibrillation. Heart Rhythm. 2017;14(10):e275-e444.
3. Mills LC, Gow RM, Myers K, Kantoch MJ, Gross GJ, Fournier A, et al. Lone atrial fibrillation in the pediatric population. Can J Cardiol. 2013;29(10):1227-33.
4. Ceresnak SR, Liberman L, Silver ES, Fishberger SB, Gates GJ, Nappo L, et al. Lone atrial fibrillation in the young - perhaps not so "lone"? J Pediatr. 2013;162(4):827-31.
5. Lee PC, Hwang B, Tai CT, Chiang CE, Yu WC, Chen SA. The different electrophysiological characteristics in children with Wolff-Parkinson-White syndrome between those with and without atrial fibrillation. Pacing Clin Electrophysiol. 2004;27(2):235-9.
6. El-Assaad I, Al-Kindi SG, Saarel EV, Aziz PF. Lone Pediatric Atrial Fibrillation in the United States: Analysis of Over 1500 Cases. Pediatr Cardiol. 2017;38(5):1004-9.
7. Kirchhof P, Benussi S, Kotecha D, Ahlsson A, Atar D, Casadei B, et al. 2016 ESC Guidelines for the management of atrial fibrillation developed in collaboration with EACTS. Eur Heart J. 2016;37(38):2893-962.
8. Iwasaki YK, Nishida K, Kato T, Nattel S. Atrial fibrillation pathophysiology: implications for management. Circulation. 2011;124(20):2264-74.
9. Brieger D, Amerena J, Attia J, Bajorek B, Chan KH, Connell C, et al. National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand: Australian Clinical Guidelines for the Diagnosis and Management of Atrial Fibrillation 2018. Heart Lung Circ. 2018;27(10):1209-66.
10. Gourraud JB, Khairy P, Abadir S, Tadros R, Cadrin-Tourigny J, Macle L, et al. Atrial fibrillation in young patients. Expert Rev Cardiovasc Ther. 2018;16(7):489-500.
11. Wasmer K, Breithardt G, Eckardt L. The young patient with asymptomatic atrial fibrillation: what is the evidence to leave the arrhythmia untreated? Eur Heart J. 2014;35(22):1439-47.
12. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, Cleveland JC, Jr., et al. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines and the Heart Rhythm Society. Circulation. 2014;130(23):e199-267.
13. Zipes DP, Jalife J, Stevenson WG. Cardiac electrophysiology : from cell to bedside. Seventh edition. ed. Philadelphia, PA: Elsevier; 2018.
14. Wijesurendra RS, Casadei B. Mechanisms of atrial fibrillation. Heart. 2019;105(24):1860-7.
15. Nanthakumar K, Lau YR, Plumb VJ, Epstein AE, Kay GN. Electrophysiological findings in adolescents with atrial fibrillation who have structurally normal hearts. Circulation. 2004;110(2):117-23.
16. Wakili R, Voigt N, Kaab S, Dobrev D, Nattel S. Recent advances in the molecular pathophysiology of atrial fibrillation. J Clin Invest. 2011;121(8):2955-68.

17. Chou CC, Chen PS. New concepts in atrial fibrillation: neural mechanisms and calcium dynamics. Cardiol Clin. 2009;27(1):35-43, viii.
18. Kaltman JR, Tanel RE, Shah MJ, Vetter VL, Rhodes LA. Induction of atrial fibrillation after the routine use of adenosine. Pediatr Emerg Care. 2006;22(2):113-5.
19. Sankaranarayanan R, Kirkwood G, Dibb K, Garratt CJ. Comparison of Atrial Fibrillation in the Young versus That in the Elderly: A Review. Cardiol Res Pract. 2013;2013:976976.
20. Liberman L, Hordof AJ, Altmann K, Pass RH. Low energy biphasic waveform cardioversion of atrial arrhythmias in pediatric patients and young adults. Pacing Clin Electrophysiol. 2006;29(12):1383-6.
21. Tibballs J, Carter B, Kiraly NJ, Ragg P, Clifford M. Biphasic DC shock cardioverting doses for paediatric atrial dysrhythmias. Resuscitation. 2010;81(9):1101-4.
22. Vohra J, Rajagopalan S, Group CGCW. Update on the Diagnosis and Management of Brugada Syndrome. Heart Lung Circ. 2015;24(12):1141-8.
23. Radford DJ, Izukawa T. Atrial fibrillation in children. Pediatrics. 1977;59(2):250-6.
24. Pieroni M, Chimenti C, Frustaci A. Letter regarding article by Nanthakumar et al, "Electrophysiological findings in adolescents with atrial fibrillation who have structurally normal hearts". Circulation. 2005;111(3):e27.
25. Collins KK, Thiagarajan RR, Chin C, Dubin AM, Van Hare GF, Robbins RC, et al. Atrial tachyarrhythmias and permanent pacing after pediatric heart transplantation. J Heart Lung Transplant. 2003;22(10):1126-33.
26. Parvez B, Darbar D. Lone AF - Etiologic Factors and Genetic Insights into Pathophysiolgy. J Atr Fibrillation. 2010;3(1):236.
27. Enriquez A, Antzelevitch C, Bismah V, Baranchuk A. Atrial fibrillation in inherited cardiac channelopathies: From mechanisms to management. Heart Rhythm. 2016;13(9):1878-84.
28. Krishnamoorthy S, Lip GY, Lane DA. Alcohol and illicit drug use as precipitants of atrial fibrillation in young adults: a case series and literature review. Am J Med. 2009;122(9):851-6 e3.
29. Di Rocco JR, During A, Morelli PJ, Heyden M, Biancaniello TA. Atrial fibrillation in healthy adolescents after highly caffeinated beverage consumption: two case reports. J Med Case Rep. 2011;5:18.
30. Lawless CE, Briner W. Palpitations in athletes. Sports Med. 2008;38(8):687-702.
31. Trivedi SJ, Claessen G, Stefani L, Flannery MD, Brown P, Janssens K, et al. Differing mechanisms of atrial fibrillation in athletes and non-athletes: alterations in atrial structure and function. Eur Heart J Cardiovasc Imaging. 2020;21(12):1374-83.
32. Mainzer G, Khoury A, Gelernter-Yaniv L, Lorber A. Neonatal atrial fibrillation after surgical repair of tracheoesophageal fistula with esophageal atresia. Pediatr Cardiol. 2008;29(1):150-2.

Tables:

Table 1. Causes and risk factors for paediatric atrial fibrillation

Causes and risk factors	References
Structural heart disease	
Congenital heart disease (particularly left heart obstructive lesions, palliated single ventricles, and mitral valve disease)	(13, 23)
Valvular heart disease	(23)
Cardiomyopathies (HCM, DCM, RCM, ARVC, LVNC)	(23)
Rheumatic heart disease	(23)
Infective endocarditis	(23)
Atrial tumours	(23)
Peri-/myocarditis	(24)
Supraventricular tachyarrhythmia (AET, MAT, atrial flutter, AVRT including WPW, AVNRT)	(1, 3, 4, 23)
Cardiac surgery	(23)
Cardiac transplantation	(25)
Genetic	
Familial AF (monogenic, polygenic)	(26)
Channelopathies (LQTS, SQTS, CPVT, BrS, ERS)	(27)
Extracardiac causes	
Autonomic imbalance	(26)
Obesity	(6)
Obstructive sleep apnoea	(19)
Hypertension	(11)
Alcohol	(28)

Caffeine	(19, 29)
Nicotine	(19)
Illicit drugs	(28)
Medications (adenosine, cardiac stimulants, antiarrhythmics, cholinergics, sympathomimetic inhalants, xanthines, chemotherapy, central nervous system drugs)	(3, 18, 26)
Thyroid dysfunction	(1, 19)
Phaeochromocytoma	(19)
Diabetes mellitus	(10)
Venous thromboembolism	(10)
Sports & physical activity (especially endurance)	(26, 30, 31)
Non-cardiac thoracic surgery	(32)

HCM, hypertrophic cardiomyopathy; DCM, dilated cardiomyopathy; RCM, restrictive cardiomyopathy, ARVC, arrhythmogenic right ventricular cardiomyopathy; LVNC, left ventricular non-compaction cardiomyopathy; AET, atrial ectopic tachycardia; MAT, multifocal atrial tachycardia; AVRT, atrioventricular reentrant tachycardia; WPW, Wolff-Parkinson-White syndrome; AVNRT, atrioventricular nodal reentrant tachycardia; LQTS, long QT syndrome; SQTS, short QT syndrome; CPVT, catecholaminergic polymorphic ventricular tachycardia; BrS, Brugada syndrome; ERS, early repolarisation syndrome

Figure legends:

Figure 1. Atrial fibrillation with variable ventricular response (beat to beat heart rate varies 50 – 130 beats per minute). The fifth QRS complex is broader, likely due to rate related aberration (incomplete right bundle branch block).

Figure 2. Narrow complex tachycardia with heart rate ~210 beats per minute. Irregular RR intervals suggest atrial fibrillation with rapid ventricular response rather than supraventricular tachycardia.

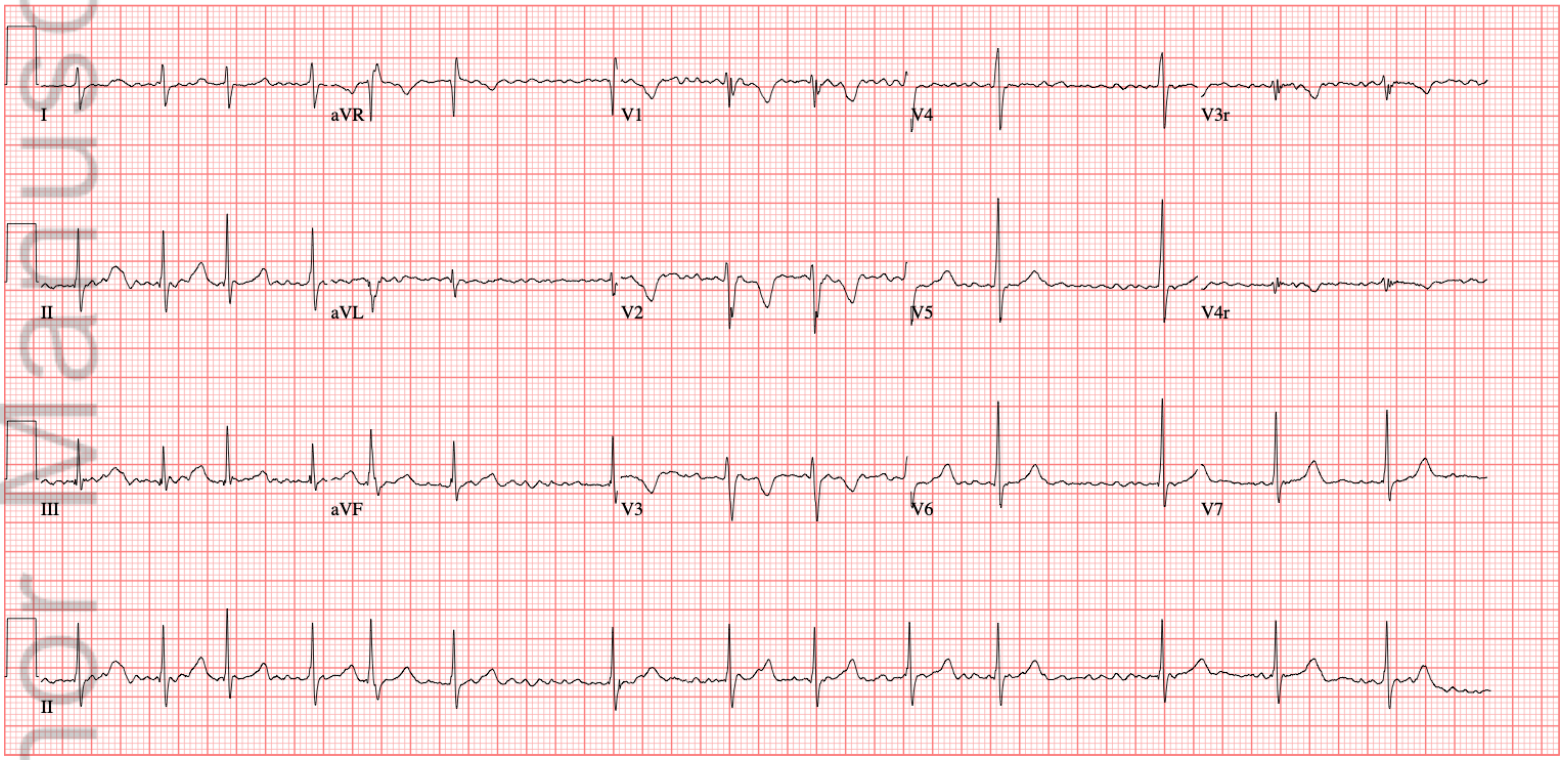
Figure 3. Atrial fibrillation with rapid ventricular response in a patient who presented with dilated cardiomyopathy and later found to have catecholaminergic polymorphic ventricular tachycardia.

Figure 4. Atrial flutter with variable atrioventricular conduction (2:1 – 4:1) in the same patient from Figure 3.

Figure 5. Multifocal atrial tachycardia with irregular RR intervals, again in the same patient from Figure 3.

Figure 6. Atrial fibrillation with rapid ventricular response in a patient with Wolff-Parkinson-White syndrome.

Figure 7. Unmasking of fast conducting AF with Adenosine (left part of the strip, with weaning of Adenosine effect then again fast conduction over the AV node.



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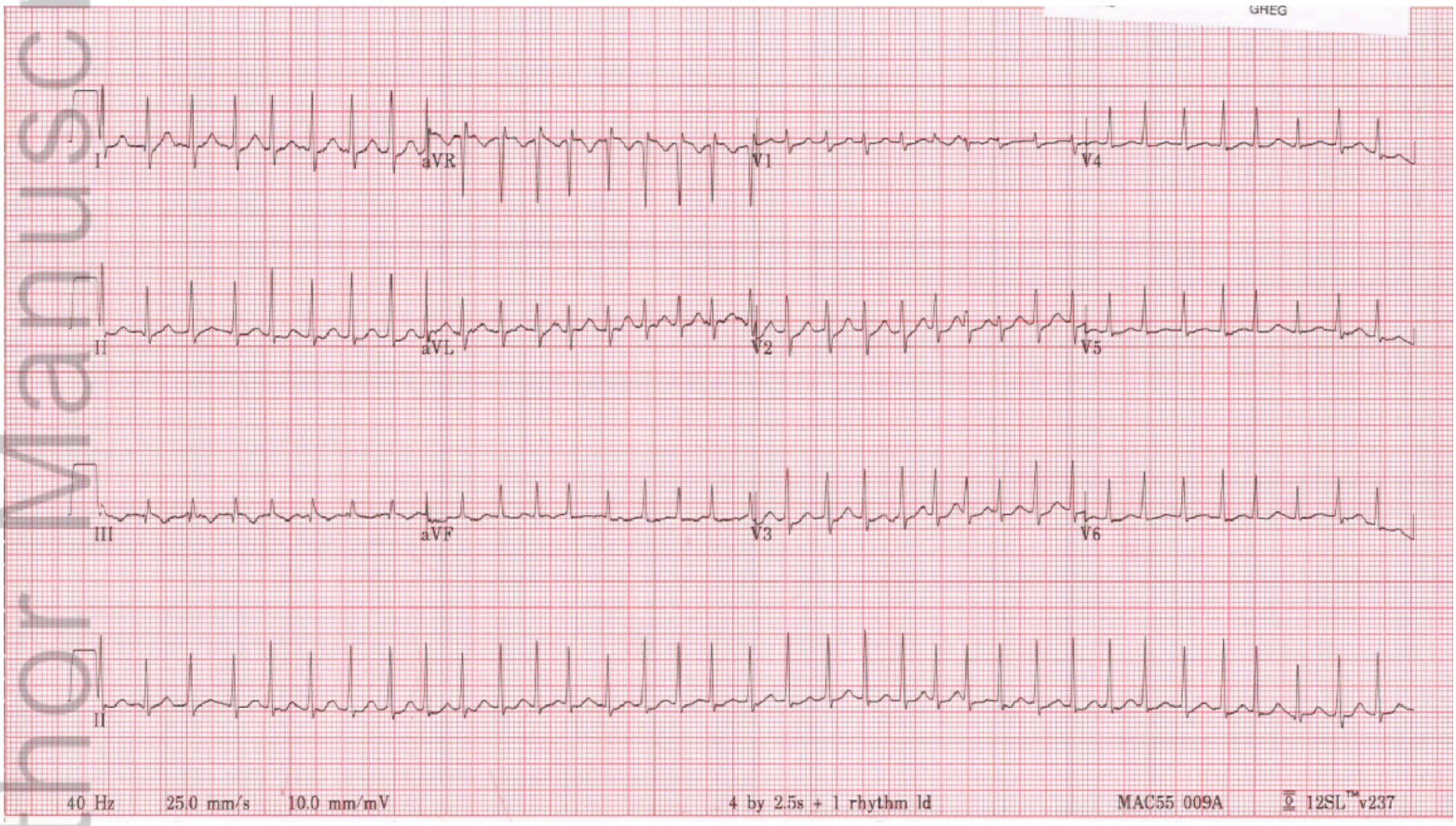

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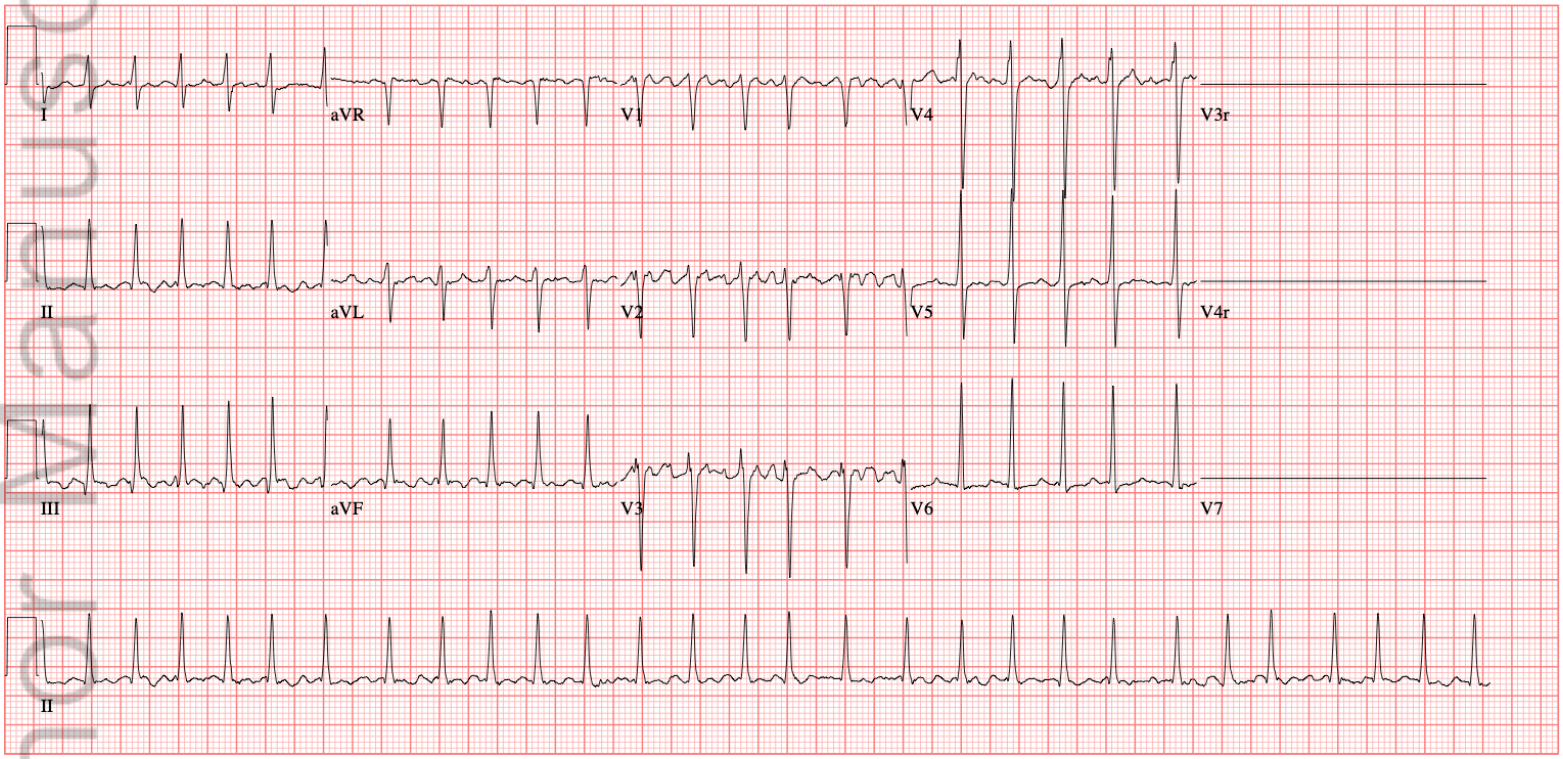

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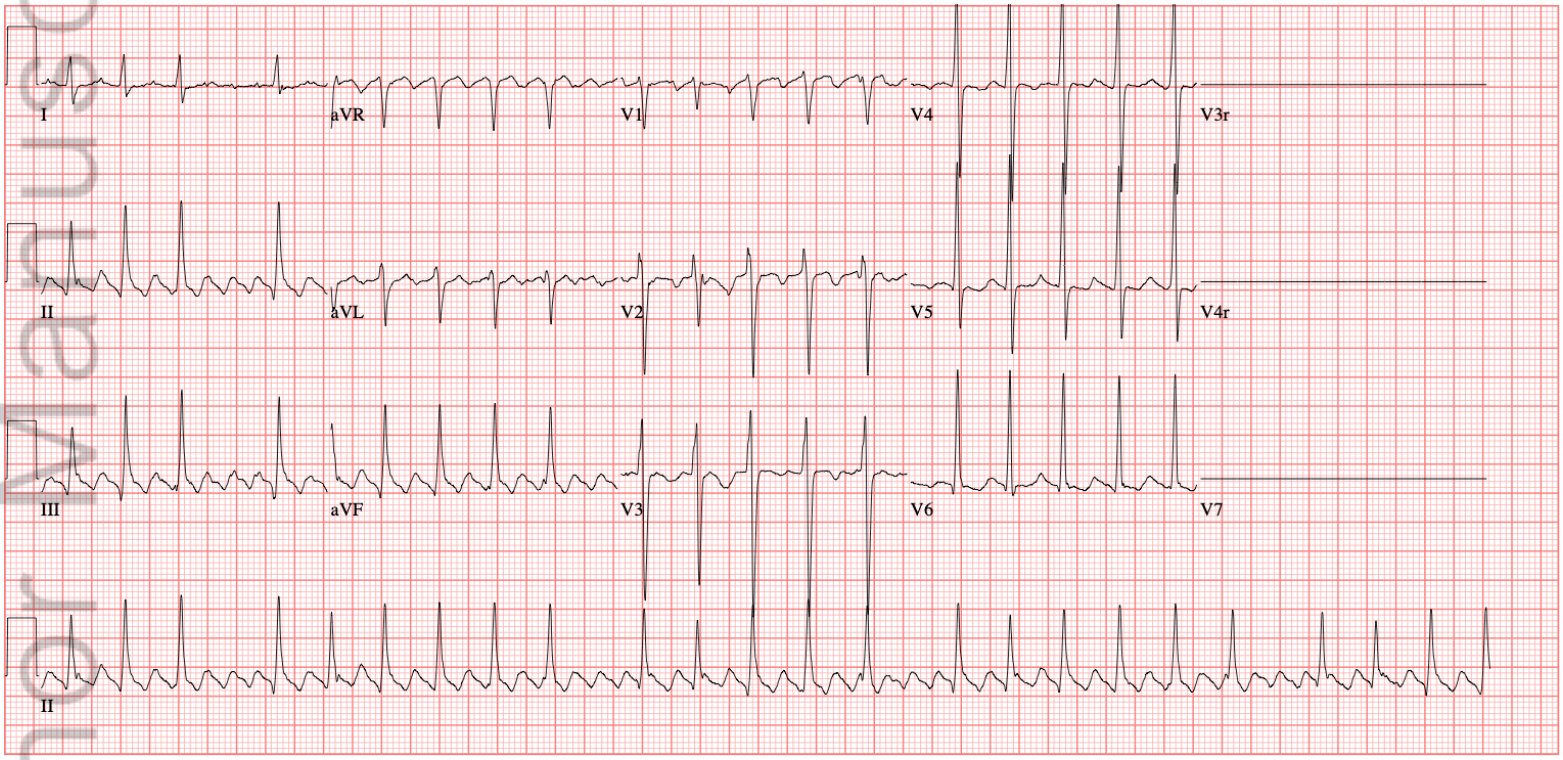

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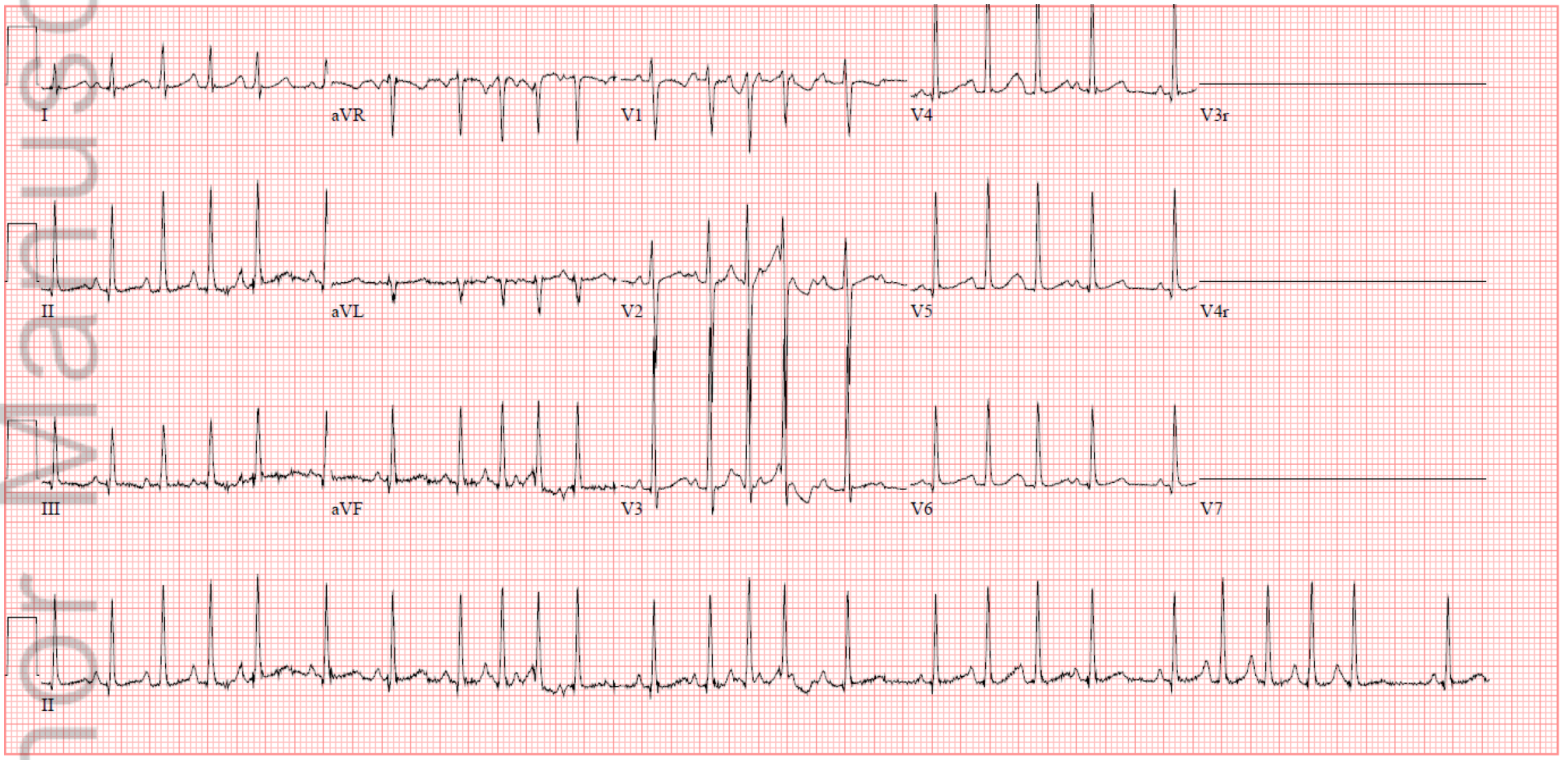

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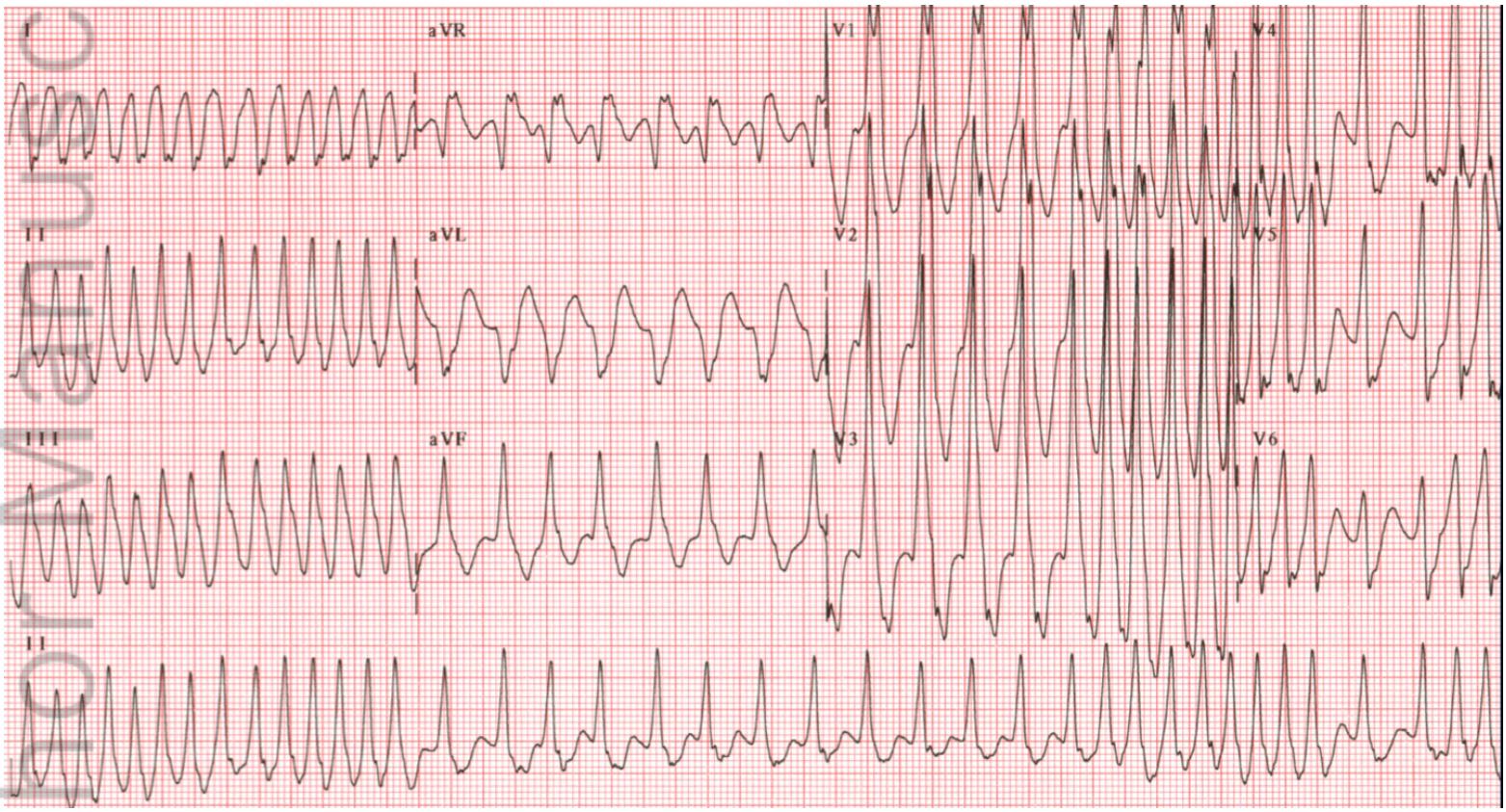

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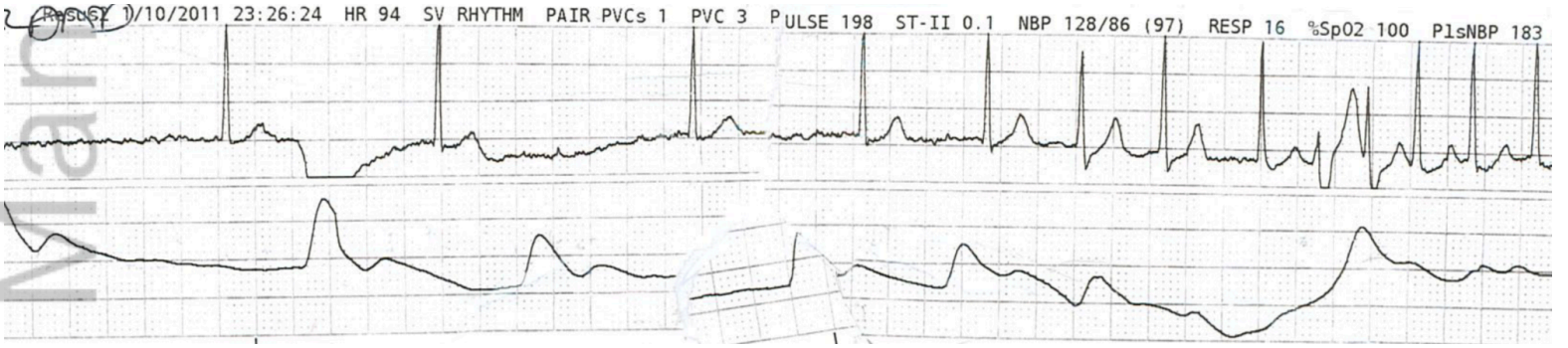

JPC_15714_Figure 7.png

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