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Review Article

ECG Easy as 123

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Abstract

This manuscript provides a novel, simplified approach to rhythm interpretation and management. It outlines an algorithm that asks the physician to recognize simple aspects of the ECG: rate, regularity, complex width, and P-waves relationships. The physician arrives at broad diagnostic groupings that share common treatment pathways based on heart rate or rhythm treatments. Cardiac medications are grouped according to their rate and/or rhythm-modifying characteristics, rather than molecular site and mode of action. In our opinion safe and effective management is more important than precise ECG diagnosis, and targeted drug therapy. This algorithm provides a safe, effective and intuitive approach to arrhythmia management.

Keywords

Arrhythmia; Arrhythmia Cardiac Care; ECG Interpretation; ECG Training

Introduction

Traditional ECG interpretation as taught in textbooks, study guides, and published algorithms may be based on detailed anatomy, electrical conduction patterns, and complex vector analysis. Common algorithmic treatment protocols for dysrhythmia are predicated on precise rhythm recognition and clinical parameters [1-7]. These method can prove challenging, particularly when a definite rhythm diagnosis is not immediately apparent. Rhythm interpretation is often the most difficult step in the process. We propose a simplified approach to management of dysrhythmia that requires recognition of only a few simple factors: fast or slow, regular or irregular, wide or narrow complex, and present/absent P-waves. We also present a pharmacologic table of anti-arrhythmic medications grouped by site of action and cardiac rate and/or rhythm-modifying characteristics.

Overview

The ECG is a static picture of the cardiac electrical conduction system. It is inexpensive, painless, and reproducible. The conduction pathways can be affected by many different factors, therefore ECG interpretation must include clinical context. In the emergent setting, the exact rhythm diagnosis may not be as important as simply recognizing that the rate is slow or fast.

Along with a rhythm strip, the 12-lead ECG is crucial in identifying the heart rhythm. It is important to remember a few rules when determining the origin of a rhythm. A "P" wave identifies organized atrial activation. The QRS represents ventricular activation. The QRS width (normal < 120 ms) and morphology describe the origin and pattern of ventricular activation. Recognizing P-QRS association, and QRS width and regularity differs in importance between bradycardia and tachycardia rhythms. This results in necessary decision point asymmetry between the "fast" and "slow" algorithms.

In tachycardia the priority is recognizing VT. P waves and their association to QRS complexes is likely to be too difficult at rapid ventricular rates; QRS width takes higher importance. P-QRS relationships can be very difficult to identify in all arrhythmias. In bradycardia P waves are more obvious yet their association to QRS complexes may be equally uncertain. When the P-QRS relationship is uncertain in slow rhythms, QRS regularity rather than width becomes a key decision point.

Treatment should be based primarily on patients' overall clinical picture. Using the simple flowcharts in Figure 1 and

Figure 3, a practitioner can rapidly employ a safe and effective treatment strategy for every dysrhythmia regardless of precise diagnosis. Pharmacologic options may vary depending on local availability and expertise. Prompt electrical cardio version is indicated for any tachycardia patient with severe cardiac chest pain, hemodynamic compromise, or decreased mental status when the symptoms of instability are directly related to their rate or rhythm.

Ventricular fibrillation and Pulseless Electrical Activity (PEA) do not appear in our modified flowcharts. VF is easily recognized by the characteristic chaotic tracing and severely compromised patient. These patients require prompt defibrillation. A patient with PEA, will also present in extremis and should be treated immediately. Littman, Bustin, and Haley describe the management of PEA based on the width of the QRS complex: narrow is a mechanical problem (Pseudo-PEA) and wide is a metabolic problem (True-PEA) [8]. This algorithm needs to be used while keeping in mind the "H's and "T"s of PEA: Hypovolemia, Hypoxia, Hydrogen ions (acidosis), Hyper/Hypokalemia, Hypothermia, Toxins, Tamponade, Thrombosis of lungs or heart and Tension pneumothorax.

Pharmacology

Medication classes listed in the flow diagrams are based on their rate and/or rhythm-controlling characteristics. We place less emphasis on the biochemical mode of action. Treatment is dictated as much by patient condition as by the arrhythmia mechanism. Frequently, a dysrhythmia is a bystander to concomitant illness. In this instance treatment should focus on the primary problem and not the dysrhythmia e.g. antibiotics and fluid resuscitation of sepsis, diuresis and preload reduction for heart failure, thrombolysis for pulmonary embolism, and angioplasty or thrombolysis for urgent Acute Coronary Syndrome (ACS) [9-11].

When targeted treatment of the arrhythmia is appropriate, there are a number of medications available. The traditional system of classifying anti arrhythmic medications can be confusing and impractical for non-cardiologists [12-14]. We have organized these medications according to their most basic function: rate control or rhythm control. There is overlap in the functions of these agents and Table 1 lists common available drugs that would be used in the different clinical situations.

Chronotropes

Chronotropes increase heart rate. They are adrenergic stimulants that enhance all rhythm foci and circuits, and accelerate AV node conduction. Atropine is also a chronotrope but accelerates heart rate by inhibiting parasympathetic input to the sinus and AV nodes.

Rate controlling agents

The signs and symptoms of tachycardia are dictated mostly by the ventricular (pulse) rate rather than rhythm. Rate control refers to slowing of the ventricular rate. For supraventricular tachycardia, this is achieved by slowing conduction in the AV node. Calcium channel blockers (non-dihydropyridine class), beta blockers, and digoxin have this property.

Chronotropes
Isoprenaline Isoproterenol Adrenaline (epinephrine) Dopamine Atropine
Rate-controlling agents
Beta Blockers (Class II) Calcium Channel Blockers (Non-dihydropyridine) (Class IV) Digoxin
Rhythm-control agents
Procainamide (Class I a) Lignocaine (lidocaine) (Class I b) Flecainide (Class I c) Amiodarone (Class III) Sotalol (Class III) Adenosine Magnesium

Table1: Drug Classifications (Vaughan Williams classification).

Rhythm controlling agents

Rhythm controlling agents aim to terminate the underlying tachycardia mechanism. They act both in the atria and the ventricles and are classified by their site of action on the cardiac action potential. In accordance with the Vaughan Williams classification, Class 1 agents such as procainamide, lignocaine (lidocaine), and flecainide block sodium channels. Class 3 agents like amiodarone and sotalol act primarily on the potassium channel. Both of these drugs have beta-blocking properties useful for rate as well as rhythm control.

It is important to note that in AV Node Reentry Tachycardia (AVNRT) and AV Reentry Tachycardia (AVRT), so-called "rate control agents"(beta blockers and calcium channel blockers) will terminate the tachycardia mechanism. Adenosine which uniquely works at the adenosine type 1 receptor, also has this property [15]. Adenosine can be very effective at terminating node-dependent tachycardia, and is therefore a useful diagnostic tool in unmasking Atrial Flutter (AF) or Atrial Tachycardia (AT) when the tachycardia mechanism is unclear.

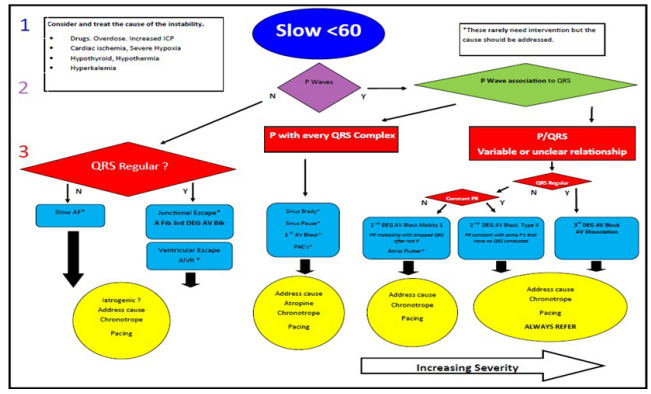

Figure 1: Slow algorithm.

In the setting of undifferentiated Broad Complex Tachycardia (BCT) that is not clearly Ventricular Tachycardia (VT), its short half-life allows safe administration even when the diagnosis is VT [16-20].

Magnesium is also included in this class. Unless contraindicated, specifically in severe renal impairment, it may have some efficacy at treating polymorphic VT and AF [21,22].

Treatment Algorithm: Bradycardia

Slow

The nature and urgency of treatment for bradycardia is determined by symptoms and rhythm. Prompt treatment is required if there is hemodynamic compromise, decreased mental status, or severe cardiac symptomology. Marked sinus bradycardia is common in athletes, is typically asymptomatic, and rarely warrants treatment. Asymptomatic bradycardia in other clinical settings may signal clinical deterioration warranting prompt attention to the underlying cause, rather than to the heart rate or rhythm. Common scenarios include drug overdoses, hypoxia, hypothermia, hypothyroid, and ACS. Other important clinical situations to consider are increased intracranial pressure as part of a Cushing reflex and hyperkalemia.

P Waves present?

The presence of P waves represents atrial depolarization. If P waves are connected to QRS complexes the rhythm is atrial in origin, either sinus or an alternative (ectopic) atrial focus. The absence of clear P waves may represent a rhythm originating at the level of the AV node (junctional rhythm), or AF. Absence of P waves is the hallmark of AF, typically associated with an irregularly irregular QRS rate.

In bradycardia QRS regularity is more important than QRS width and is discussed in more details below. A wide QRS maybe one of four things: a ventricular rhythm, supraventricular rhythm with bundle branch aberrancy, ventricular pacing, or a supraventricular rhythm with pre-excitation (delta wave), but is seldom useful in determining the acute management of bradycardia. Aberrant conduction in the Purkinje system is structural or metabolic as in electrolyte disturbance or digitalis toxicity. Lastly consider the reperfusion rhythm of accelerated idioventricular rhythm in the post thrombolytic patient [23].

Regularity and relationship of P waves to the QRS complexes

The next step in determining the diagnosis and treatment depends on the association and frequency of the P wave to the QRS complex. The relationship of the P waves to the QRS describes the flow of the electrical impulse from the atria, through the junction and into the ventricles. If every QRS is consistently preceded by a P wave at the same rate then the bradycardia has a supraventricular origin like sinus bradycardia, sinus pause, or first degree AV block.

If every P wave is not followed by a QRS complex the rhythm is either not arising from the atrium, or it is an atrial rhythm with AV conduction disturbance. The AV node is exquisitely sensitive to autonomic modulation, and physiological AV block from excessive vagal stimulation is common. It typically occurs at night, can range from PR prolongation to AV dissociation, and almost never requires treatment. Pathological AV block is commonly due to atherosclerotic heart disease. First degree and Type 1 second degree AV block is disease limited to the AV node and is typically benign.

The priority in bradycardia is identifying complete (third degree) Atrial Ventricular (AV) block. A hallmark of complete AV block is the "regularity" of QRS complexes.

Group clustering of QRS complexes can identify second degree AV block. Type 1 block is characterized by progressive PR lengthening and RR shortening prior to non-conduction of a P wave; the resultant clustering of QRS complexes gives rise to the so-called "Wenckebach footprint" (Figure 2). In Type 2 second degree block the PR interval is stable. The non-conducted P appears as a QRS "gap" with groups of conducted beats either side (Table 2).

Broadening of the QRS width, Type 2 second degree or third degree AV block, signal involvement of the distal His-Purkinje conduction system and warrant consideration for a pacemaker [24,25]. Third degree AV block or "complete heart block" is particularly important to recognize. The hallmark of third degree block is complete AV dissociation, where P waves are independent of QRS complexes, with a ventricular escape

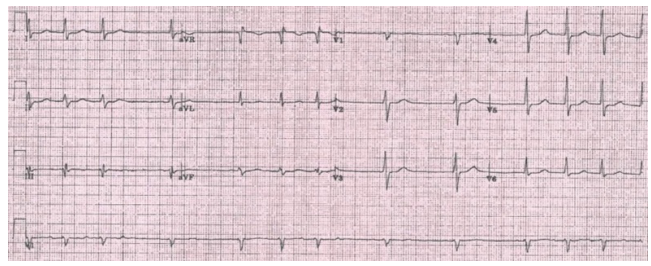

Figure 2: Wenckebach footprint and example of small P waves.

rhythm that can be slow and unreliable. In atrial fibrillation the only clue will be slow, QRS complexes. Acute transcutaneous pacing or chronotropic medication like isoproterenol can be effective. Adrenergic stimulants should be used with caution in ACS. Atropine, acting on the AV node, will not improve block due to disease in the distal His-Purkinje system. Cardiology referral is mandatory with Type 2 second degree AV block or third degree AV block.

Treatment Algorithm: Tachycardia

Fast

Tachycardic rhythms are those > 100 BPM, however they are seldom symptomatic and rarely life-threatening at rates < 120 BPM. It is important to consider that most tachycardias

Figure 3: Fast Algorithm.

are secondary to non-cardiac causes. Common secondary causes include life-threatening emergencies such as cardiac tamponade and tension pneumothorax, hypoxia, pulmonary embolus, acute coronary syndrome, hypovolemia or blood loss. Also consider fever and infections, anemia, sympathomimetic syndromes, phaeochromocytoma, andhyperthyroidism. More benign conditions such as pain and anxiety will be diagnoses of exclusion. In an unwell patient, tachycardia <150 BPM is unlikely to be the root cause of the instability; rate or rhythm control is unlikely to be helpful and may be harmful [9-11].

Is the QRS wide or narrow?

After determining clinical stability, the next priority is recognizing QRS width. Recognising ventricular tachycardia is of utmost importance. If the QRS is narrow the diagnosis is unlikely to be VT.

Is the QRS regular and are P waves present?

The next question is determining QRS regularity and the presence of P waves. This determines the origin of the cardiac impulse. A narrow, regular QRS is most likely to be SVT. Identification of P waves is of secondary importance; they can be difficult to see and seldom influence acute management. Treatment with AV blockade such as vagal stimulation by carotid sinus massage or a valsalva manoeuvre may be effective. If this is unsuccessful rate-control medication is the next appropriate step. This may terminate node-dependent processes (AVNRT or AVRT), or halt QRS complexes long enough to uncover rapid P waves in the case of atrial tachycardia or flutter.

Irregular narrow complex tachycardia is likely to be multifocal AT or AF. The presence of P waves may differentiate AT from AF but the priority for both is using rate control agents. Rhythm control may carry thromboembolic risks in the non-anticoagulated patient and additional precautions may be required but is beyond the scope of this manuscript. Rhythm control is of secondary importance in this setting.

Broad Complex Tachycardias (BCT) may be ventricular or supraventricular. In supraventricular tachycardia (SVT) the cause of broad QRS complexes is bundle branch aberrancy, ventricular pacing, or pre-excitation. In a regular BCT, VT can be difficult to distinguish from SVT with aberrancy. There is a significant amount of literature that describes specific ECG criteria and clinical characteristics to differentiate these rhythms [26-32] Adenosine can be helpful in this setting, and is a notable inclusion in our "fast" algorithm. Adenosine will not perturb VT, but is likely to terminate node-dependent tachycardia, or reveal the underlying atrial oscillations of AT or AF. If the diagnosis of VT is strongly suspected or remains uncertain rhythm agents such as procainamide or amiodarone should be used [32-34] Calcium channel blockers should be avoided because of a risk of enhancing conduction in an accessory pathway (thereby exacerbating antidromic AVRT or pre-excited AF), or causing hypotension in VT [35,36].

An irregular BCT may be multifocal AT or AF with pre-excitation, bundle branch aberrancy or ventricular pacing, or polymorphic VT. If the diagnosis cannot be clarified amiodarone is reasonable. Examination of the baseline, non-tachycardic ECG for pre-existing bundle branch aberrancy, AF, ventricular pacing, or pre-excitation can provide valuable clues. Cardioversion of AF to sinus rhythm (with anti arrhythmics or electrical cardio version) may carry thromboembolic risks in the non-anticoagulated patient as mentioned previously.

Summary

Arrhythmia management in the acute setting is based on rate, rhythm and clinical stability. Textbooks and standard published algorithms rely on the physician's ability to correctly identify a cardiac rhythm. "ECG 123" algorithms aim to identify key diagnostic groupings with shared treatment options. These new algorithms allow for safe and effective dysrhythmia treatment based upon easily identifiable ECG characteristics. The medication classifications of chronotropes, rate controlling agents, and rhythm controlling agents provide easy and usable classification categories.

References

1. Nolan J, Soar J, Lockley A (2011) Advanced life support. (6thedn), Resuscitation Council (UK).
2. Hampton JR (2013) The ECG Made Easy. 8thedn, Elsevier - Churchill Livingstone, London, UK.
3. New Zealand (2012) Resuscitation Guidelines Level 7 ACLS. Pg no: 84-95.
4. Durham D, Worthley LI (2002) Cardiac arrhythmias: diagnosis and management. The tachycardias. Crit Care Resusc 4: 35-53.
5. Lewalter T, Lickfett L, Schwab J, Yang A, Lüderitz B (2007) The Emergency Management of Cardiac Arrhythmia. Dtsch Arztebl 104: 1172-1180.
6. January CT, Wann LS, Alpert JS, Calkins H, Cigarroa JE, et al. (2014) 2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation: Executive Summary. J Am Coll Cardiol 64: 2246-2280.
7. Littmann L, Bustin M, Haley MW (2014) A Simplified and Structured Teaching Tool for the Evaluation and Management of Pulseless Electrical Activity. Med Princ Pract 23: 1-6.

8. Scheuermeyer FX, Pourvali R, Rowe BH, Grafstein E, Heslop C, et al. (2015) Emergency Department Patients With Atrial Fibrillation or Flutter and an Acute Underlying Medical Illness May Not Benefit From Attempts to Control Rate or Rhythm. Ann Emerg Med 65: 511-522.

9. Yeh Y, Li A, Wu Y (2015) Patient Selection Is Critical in Rate or Rhythm Control of Atrial Fibrillation or Flutter. Ann Emerg Med 66: 562-563.

10. Atzema C, Lam K, Young C, Kester GN (2013) Patients with atrial fibrillation and an alternative primary diagnosis in the emergency department: a description of their characteristics and outcomes. Acad Emerg Med 10: 193-199.

11. [No Authors Listed] (1991) The Sicilian gambit. A new approach to the classification of antiarrhythmic drugs based on their actions on arrhythmogenic mechanisms. Task Force of the Working Group on Arrhythmias of the European Society of Cardiology. Circulation 84: 1831-1851.

12. Cobbe SM (1987) Clinical usefulness of the Vaughan Williams classification system. Eur Heart J 8: 65-69.

13. Lau W, Newman D, Dorian P (2000) Can antiarrhythmic agents be selected based on mechanism of action. Drugs 60: 1315-1328.

14. Engelstein ED, Lippman N, Stein KM, Lerman B (1994) Mechanism-Specific Effects of Adenosine on Atrial Tachycardia. Circulation 89: 2645-2654.

15. Rankin AC, Oldroyd KG, Chong E (1989) Value and limatations of adenosine in the diagnosis and treatement of narrow and broard complex tachycardias. Br Heart J 62: 195-203.

16. Sharma AD, Klein GJ, Yee R (1990) Intravenous adenosine triphosphate during wide QRS complex tachycardia: safety, therapeutic efficacy, and diagnostic utility. Am J Med 88: 337-343.

17. Ilkhanipour K, Berrol R, Yealy DM (1993) Therapeutic and diagnostic efficacy of adenosine in Wide-Complex Tachycardia. Ann Emerg Med 22: 1360-1364.

18. Richmond HC, Taylor L, Monroe MH, Littmann L (2006) A new algorithm for the initial evaluation and management of supraventricular tachycardia. Am J Emerg Med 24: 402-406.

19. Marill KA, Wolfram S, DeSouza IS, Nishijima DK, Kay D, et al. (2009) Adenosine for wide complex tachycardia: efficacy and safety. Crit Care Med 37: 2512-2518.

20. Kulick DL, Hong R, Ryzen E, Rube RK, Nishi, et al. (1988) Electro physiologic effects of intravenous magnesium in patients with normal conduction systems and no clinical evidence of cardiac disease. Am Heart J 115: 367-373.

21. Brugada P (2000) Magnesium: an antiarrhythmic drug, but only against very specific arrhythmias. Eur Heart J 21: 1116.

22. Goldberg S, Greenspon AJ, Urban PL, Muza B, Berger B, et al. (1983) Reperfusion arrhythmia: A marker of antegrade flow during intracoronary thrombolysis for acute myocardial infarction. Am Heart J 105: 26-32.

23. Langendorf R, Pick A (1983) Atrioventricular block, type II (Mobitz) - its nature and clinical significance. Circ; 38: 819-821.

24. Haft JI (1977) Clinical implications of atrioventricular and intraventricular conduction abnormalities. Cardiovasc Clin; 8: 41-64.

25. Brugada P, Brugada J, Mont L, Smeets J, Andries EW (1991) A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circ; 83: 1649-1659.

26. Lau EW, Pathamanathan RK, Ng GA, Cooper J, Skehan JD, et al. (2000) The Bayesian approach improves the electrocardial diagnosis of broard complex tachycardia. Pacing Clin Electrophysiol 23: 1519-1526.

27. Vereckei A, Duray G, Szénási G, Altemose GT, Miller JM, et al. (2007) Application of a new algorithm in the differential diagnosis of wide QRS complex tachycardia. Eur Heart J 28: 589-600.

28. Pava LF, Perafán P, Badiel M, Arango JJ, Mont L, et al. (2010) R-wave peak time at DII: a new criterion for differentiating between wide complex QRS tachycardias. Heart Rhythm 7: 922-926.

29. Szelényi Z, Duray G, Katona G, Frituz G, Szego E, et al. (2013) Comparison of the "Real-Life" Diagnostic Value of Two Recently Published Electrocardiogram Methods for the Differential Diagnosis of Wide QRS Complex Tachycardias. Acad Emerg Med 20: 1121-1130.

30. Jastrzebski M, Kukla P, Czarnecka D, Kawecka-Jaszcz K (2012) Comparison of five electrocardiographic methods for differentiation of wide QRS-complex tachycardias. Europace 14: 1165-1171.

31. Vereckei A (2014) Current algorithms for the diagnosis of wide QRS complex tachycardias. Curr Cardiol Rev 10: 262-276.

32. Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, et al. (2010) Part 8: Adult Advanced Cardiovascular Life Support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 122: 729-767.

33. Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, et al. (2006) ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death--executive summary: A report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death) Developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. Eur Heart J 27: 2099-2140.

34. Garmel G (2008) Wide Complex Tachycardias: Understanding this Complex Condition Part 2 - Management, Miscellaneous Causes, and Pitfalls. West J Emerg Med 9: 97-103.

35. Buxton AE, Marchlinski FE, Doherty JU, Flores B, Josephson ME (1957) Hazards of intravenous verapamil for sustained ventricular tachycardia. Am J Cardiol 59: 1107-1110.