



Review Article

ARRHYTHMIA'S: TYPES, PATHOPHYSIOLOGY AND THERAPY: A REVIEW

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ABSTRACT

Arrhythmia is irregular heartbeat, too slow or too fast. Heart function is regulated by electrical impulse which initiate from the sinus node and deputed over heart. Rhythmic change occurs in heart due to abnormal conduction of impulse results in arrhythmia Origin and mechanism of the arrhythmia is different and the electrocardiogram is used to have complete knowledge of arrhythmias, methodology and its construction of electrical impulse. Tele ECG is one among, the recent invention which has reduced the workload of recording heartbeat in the diagnosis of cardiovascular disease. According to the impulse disorder in heart, different arrhythmia were categorised. Mainly two, disorder of impulse occur in atrium is a called atrial arrhythmia and irregular impulse occur in ventricle is a known as ventricular arrhythmia. Anti-arrhythmic agents are classified based on changes in the phases of myocardial cell to treat the various arrhythmia. Herbal medicine has a minimized in side effects.

Keywords: Arrhythmia, Electrocardiogram, Tele ECG, Anti-arrhythmic

INTRODUCTION

Cardiac arrhythmias usually persist with a slow or a fast heartbeat consisting of irregular rhythm. The necrosis of the cells of a heart can be due to a long-term ischemic effect from a complete absence of oxygen¹. This causes an abnormal re-entry of the electrical impulses, which causes arrhythmia. In the case of fibrosis, ageing and cardiac channel mutation the irregular cardiac action potential is developed which may make the patient susceptible to cardiac arrhythmias. An electrical device which activates the electrical signal in the presence of specific cardiac structure for generating, conduction, and distribution to the contractile myocardium². In heart, Electric impulse is generated from the sinus node and deputed to specific ionic channels explicitly situated in cells and another part of the heart. Secondary pacemaker show activity in crisis condition³.

Capacity to generate a frequent depolarisation's reduces the SA node, and is minimum in ventricle. The primary pacemaker receives essential neuro vegetative inputs, were depolarisation are frequently regulated change it to needs of the organism for the metabolic⁴. By conduction system, impulse from sinus node is deputed to the heart; the sinus node and atrioventricular node are connected through the inter-nodal tracts that constitute the physiologically decelerator the electromechanical delay between the atrial and ventricular conduction. One of the major cause of casualties in CVD is the arrhythmia, thus monitoring the patient periodically by using an ECG tool can help to restraint such casualty provoked by arrhythmia⁵

The development of Tele-ECG has reduced the casualties caused by the arrhythmia, which is a disorder that takes place in the normal rhythm of the heart. The ventricular fibrillation is very fatal and can lead death in the victims. The availability of the ECG in the rural hospital had reduced the time for detection and has been an effective way to analyse the signals produced by the arrhythmia. This signal processing can be applied to improve the diagnosis through cardiac electrical signals⁷.

TYPES OF ARRHYTHMIA

Atrial flutter⁹

In the atrium, fibrillation comprises a numerous irregular and diverse quivers, atrial flutter is from part of the atrium is not directly legitimately, and because of this, the anomalous heart conduction is occurring in the heart. Nor a perfect directing blood through the heart. The few patients might experience flutter and fibrillation. Atrial flutter beats 250 to 350 beats/min, and Untreated of atrial flutter leads to fibrillation (table no: 2)³.

Atrial fibrillation

It is sporadic beats of the atrial chamber - almost rapid in every case. Atrial fibrillation is mostly influencing more in geriatric patients. Atrial chamber rather than the single and robust contraction, chamber fibrillates (shudder). The chamber fibrillate 350 beats/min in some cases and 600 beats/min in extreme cases.

Supraventricular tachycardia (SVT)

Patient experiences the burst of a rapid pulse which last for seconds to hour.it is regular and sporadic heartbeats. Heart rate range between 160-200 beats/min, from the supraventricular tachycardia (SVT) only atrial fibrillation and flutter are classified.

Ventricular tachycardia

It is origin from ventricles and causes a sporadic and rapid heartbeat. It frequently happens if the heart has experienced the prior heart attack. Ventricle contract more than 200 times/min.

Ventricular fibrillation

Ventricular fibrillation is uncoordinated fluttering contraction and very fast sporadic rhythm of the ventricles. Pumping ability of blood in ventricle will be improper shudder. VF is life threatening it was triggered by a heart attack (Fig: 2)⁶.

SYMPTOMS AND SIGNS ⁹

Symptoms a person will experience

- Palpitations
- A slow heartbeat
- Sporadic heartbeat
- A feeling of pauses between heartbeats

More serious signs and symptoms include:

- Anxiety
- Weakness
- Dizziness and light-headedness
- Fainting or nearly fainting
- Sweating
- Shortness of breath
- Chest pain

The condition that weakens the heart, such as:

- Heart attack
- Heart failure or cardiomyopathy
- Heart tissue that is too thick or stiff
- Leaking or narrowed heart valves,
- Congenital problems

Other conditions also can increase the chances of arrhythmia, such as:

- High blood pressure
- Damages of the heart muscle or sac around the heart due to infection
- Diabetes
- Sleep apnoea
- Overactive or underactive thyroid
- Drugs for heart surgery like cocaine or amphetamine
- Imbalance of chemicals in a body (potassium, magnesium)

PATHOPHYSIOLOGY ⁶

An electrical system of the heart

The heart is present in the centre of the thoracic cavity, and its function is to pump blood throughout the body by the continuous rhythmic contractions. An increase in the intracellular potentials is accompanied by the electrical activation of the myocardial cells which occurs through the contraction of the heart. The membrane of the myocardial cells has a specific ion channel which generates a sequence of ion fluxes known as the action potential. SA node is located on the right atrium act as the pacemaker in a healthy heart. Electrical impulse proliferates through atrial wall and reaches a ventricle for the contraction respectively¹⁰. The electrical activity can be measured indirectly by electrocardiogram (ECG). It reflects the many deflections of the electrical activity of the heart chamber (Fig: 4)⁷.

P wave – Electrical activation of the atria
QRS wave – ventricular depolarization
T wave – ventricular repolarization

Disorder of Impulse Conduction

Delay and block of the conduction results in tachyarrhythmia's, Brady arrhythmia's and re-entrant excitation (Fig: 1)⁸.

Re-entry

The whole heart is activated through the electrical activity which begins in the sinus node, and thus each cardiac cell are interconnected electrically, and if the impulse perishes, all fibres have been discharged and are entirely refractory. At the period of

refractory, the cardiac impulse has no place to go, and it must be extinguished¹⁰.

Reflection

It is subclass of re-entry, however, varies in the impulse seems to go laterally a similar alleyway in the two bearings. It ventures and meets a region in disabled conduction, where delays in active transmission. Electronically, the debilitation motivation traverses, initiates the distal portion, to re-excite the proximal section it returns the electronically over the area of impeded (Fig: 3)¹³.

Immature complex are caused by solitary reflection. While proceeding with the forward reflection and backwards over inexcitable area may cause the tachycardia. Re-entry is caused by tachyarrhythmia, it include different flutter, fibrillation, ventricular tachycardia's and supraventricular ¹².

Sinus and atrial re-entry

Re-entry is parts of the chamber has accounted for a few exploratory models, and additionally in people. The SA node imparts to the Atrio ventricle node electrophysiological highlights, for example, the potential for separation of conduction¹². For instance, untimely incitement can deliver moderate spread in the sinus node, hinder in a few regions, and the improvement of dreary reactions in all likelihood caused by re-entry. Proof from concentrates in humans²³ and animals²⁴ underpins the idea that maintained re-entry in the SA node can happen and cause SVT (supra ventricular tachycardia) ^{14,15}.

THERAPY

Class I (anti-arrhythmic agents)

Class I agent includes the most class of antiarrhythmic agent. This agent reduces the sodium influx which is responsible for conduction of cardiac action potential. Also they reduce and block intra cardiac conduction (table: 3 and Fig: 5)^{7,16}.

Class IA drugs (quinidine, disopyramide and procainamide)

Quinidine is used most widely upon these drugs because it has same electrophysiology property in both *in-vivo* and *in-vitro*. It blocks the influx of the sodium current (responsible for the repolarization of the cardiac potential action) and efflux of potassium current (liable to the repolarization of the Cardiac Potential Action) at the concentration near to the therapeutic level ¹⁶.

Class I B Drugs (Lidocaine, phenytoin, mexiletine)

These agents diminish automaticity and conduction; though, they quick repolarization diminished APD even though ERP is increased and Lidocaine is generally utilized agent in intense VA (ventricular arrhythmia) due to its high effectiveness and negligible side effects. Also, it has a two mechanism of action to diminishing the automaticity (phase 4) and ensuing ectopic, re-entry arrhythmia caused by conduction of impulse (phase 0). Recalcitrance (phase 3) is insignificantly influenced, with a decline in APD (action potential duration) counterbalanced by an elevate ERP (effective refractive period) ¹⁸.

Class IC Drugs (Flecainide, Propafenone, Moricizine)

Class IC agents are particularly reduced (phase 4) and conduction (phase 0) in a nonselective equivalent in ordinary and ischemic tissue, whereas action potential duration and the effective refractory period is unaffected ¹⁹. Encainide not effect on the effective refractory period but slows the conduction velocity and diastolic depolarisation (phase 4), that elevates the PR, QRS, and QT periods. Encainide is also (30-60%) successful to treat ventricular tachycardia or Atrial fibrillation.

Class II - Sympathetic Antagonists (propranolol, Timolol, metoprolol, atenolol, esmolol)

In 1958 this class of drug used as an antagonist for isoproterenol and a multitude of effects. Beta antagonist have diminished the action potential of phase 4, phase 0 and phase 3 by the impact on exogenous autonomic induce to maintenance of internal membrane^{20,21}. Some equal characterisation on this agents is SA node reduction, Atrio ventricle conduction and inotropic state this results in a decrease of myocardial oxygen utilisation. Each drug has specific characterisation like lipid solubility relational to the side effects in CNS.

Class III - Antifibrillatory agents (Amiodarone, sotalol, Ibutilide, dofetilide)

This agents is to re-establish automaticity (Phase 4), Covert the multiple to uniform morphology of heterogeneous action potential, and moderate re-entry recuperation by delaying the Phase 3²².

Amiodarone is examined first as anti-anginal coronary artery vasodilator, this agent comprises of a procainamide-Lidocaine congener part, physiological effect on diethyl amine group. And

thyroid function effects on iodine group²³. Amiodarone inhibits the calcium and sodium channel in SA and AV nodes and purkinje's fibres respectively, it leads to falls of conduction in Phase-0. These action extent the APD (action potential duration) and ERP(Effective refractive period), showed as differed bundle of His atrial (AH) and ventricle's (HV) intervals associating in elevating the intervals between PR and QT in ECG^{24,25}.

Class IV-Calcium channel rival (Nifedipine, Verapamil, and Diltiazem)

This type of agent act as calcium channel blocker, which inhibits the calcium entry in SA and AV nodes. To treat specifically conduction disorder like Supra-Hissian (Verapamil > Diltiazem > Nifedipine)^{26,27}. The cardiovascular effect are decreases diastolic depolarisation (Phase-4), activity potential incline and sufficiency (phase-0).cardiovascular sequence unwind (N>D>V), reduces contraction in intracellular calcium, and bad chronotropic effect AV > SA nodes. These class drug are used to treat violent Supra ventricular tachycardia 90% and atrial fibrillation 10% typical sinus rhythm. (Wolff- Parkinson- white disorder)^{28,29}.

HERBAL APPROACHES FOR CARDIAC ARRHYTHMIA

Table 1: Medicinal plants for arrhythmia

Name of the plant	Family	Chemical constituent	Uses
<i>Tinospora cordifolia</i>	Menispermaceae	Alkaloids, cardiac glycosides, diterpenoids, sesquiterpenoids, steroids, phenolics, terpenoids.	Cardio protective, chronic diarrhoea, anti-cancer, anti-diabetic, anti-tumour, anti-tussive, anti-inflammatory, anti-ulcer ³⁰ .
<i>Parkia biglobosa</i>	Fabaceae	Saponian, tannins, cardiac glycosides, flavonoids, phenolic compounds, gallic acid, ellagic acid.	Cardio protective, antioxidant, anti-hyperlipidemic, anti-diarrhoeal, treat infectious diseases ³¹ .
<i>Nelumbo nucifera</i>	Nymphaeaceae	Nuciferine, alkaloids, glycosides, myricetin, quercetin, flavonoids, adenine, sitosterols.	Cardio protective, treat digestive problems, enteritis, cough, poisoning antidote, anti-cancer, spermatorrhoea, insomnia, diuretic ³² .
<i>Terminalia arjuna</i>	Combretaceae	Triterpenoids, arjunic acid, arjunolic acid, tannins, flavonoids, gallic acid, ellagic acid, arjuna glycosides, phytosterols.	Cardio protective, inotropic, anti-ischemic, antioxidant, anti-hypertensive, anti-atherogenic, anti-hypertropic, hypolipidemic, anti-platelet ³³ .
<i>Ficus racemosa</i>	Moraceae	Lupeol, lupeol acetate, amyrin, sitosterol, stigmasterol, coumarin, psoralen, friedelin, kaempferol, gallic acid, bergenin, bergapten, rutin, ellagic acid, arabinose, racemosic acid.	Cardio protective, anti-hyperglycemic, antioxidant, analgesic anti-diarrheal, anti-bacterial, anti-fungal, anti-filarial, gastro protective, larvicidal, anti-inflammatory ³⁴ .
<i>Trichopus zeylanicus</i>	Trichopodaceae	Glycosides, alkaloids, flavonoids, terpenoids, saponins phytosterols	Cardioprotective, to increase stamina, hepatoprotective, weight loss, peptic ulcer, sexual dysfunction, fatigue ³⁵ .
<i>Pithecellobium dulce</i>	Fabaceae	Tannin, fixed oil, olein, glycosides, steroids, saponins, lipids, phospholipids, glycolipids, polysaccharides	Cardio protective, anti-hyperlipidemic, antioxidant, antiseptic, antibacterial, toothache, gum ailments, hemorrhage, anti-tuberculosis, anti-inflammatory, anti-diabetic, hepatoprotective, anti-diarrheal, CNS depressant ³⁶ .
<i>Mangifera indica</i>	Anacardiaceae	Xanthone C glycoside, urushiols, kaempferol, quercetin, tnnins, flavonoids, polyphenolic compounds ³⁸	Cardio protective, anti-viral, antioxidant, anti-diabetic, anti-allergic, anti-tumor, anti-inflammatory, anti-parasite, anti-pyretic, hepatoprotective, antidiarrheal, antispasmodic, gastroprotective, anti HIV, antimmunomodulator, anti- bone resorption ³⁷
<i>Nigella sativa</i>	Ranunculaceae	Omega-6-fatty acid, alkaloid, omega-3-fatty acid, vitamins, saponins, proteins, mono terpenoids, phenols	Cardio protective, anti-cancer, anti- diabetic, gastroprotective, bronchodilatory, anti-inflammatory, anti-microbial ³⁹
<i>Momordica charantia</i>	cucurbitaceae	Triterpenoids, monordenol, momordicilin, momodincin, charantidiol, saponins, cucurbitins	Cardioprotective, analgesic, anti-diabetic, anti-inflammatory, anti-microbial, larvicidal, antioxidant, hepatoprotective ⁴⁰

Table 2: Types of Arrhythmia³

Class	Arrhythmia can be any of the following
1	Ischemic changes
2	Old frontal myocardial
3	Old lower myocardial
4	Sinus tachycardia
5	Sinus bradycardia
6	Ventricular premature contraction
7	Supraventricular premature contraction
8	Left bundle branch block
9	1 st Degree Atrioventricular Block
10	2 nd Degree Atrioventricular Block
11	3 rd Degree Atrioventricular Block
12	Left ventricle hypertrophy
13	Atrial fibrillation

Table 3: Classification of arrhythmia¹⁷

No	Class	Mechanism of action	Drugs
1	Class Ia	Blocks fast sodium channel, Depresses phase 0 depolarisation, Prolong action potential	Quinidine, Procainamide, Disopyramide
2	Class Ib	Sodium channel blockers	Lidocaine, Phenytoin, mexiletine
3	Class Ic	Most potent sodium channel blockers, Markedly depress phase 0 depolarisation	Flecainide, Propafenone, moricizine
4	Class II	Beta-blockers	Propranolol, Timolol, metoprolol, Atenolol, esmolol
5	Class III	Block potassium efflux	Amiodarone, Sotalol, Ibutilide, Dofetilide
6	Class IV	Slow calcium channel blocker	Verapamil, Diltiazem, Nifedipine
7	Class V	Variable mechanism	Adenosine, Digoxin, Magnesium sulphate

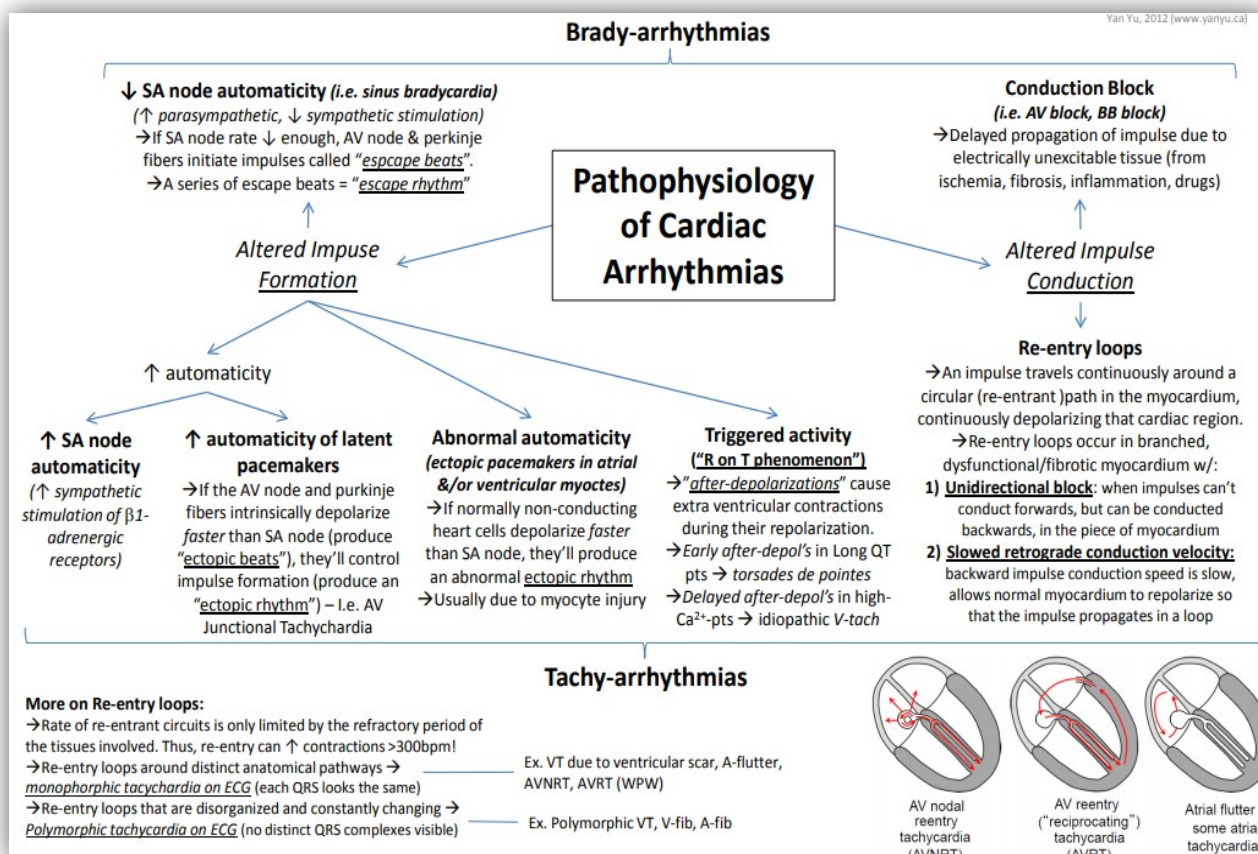


Figure 1: Pathophysiology of cardiac Arrhythmia⁸

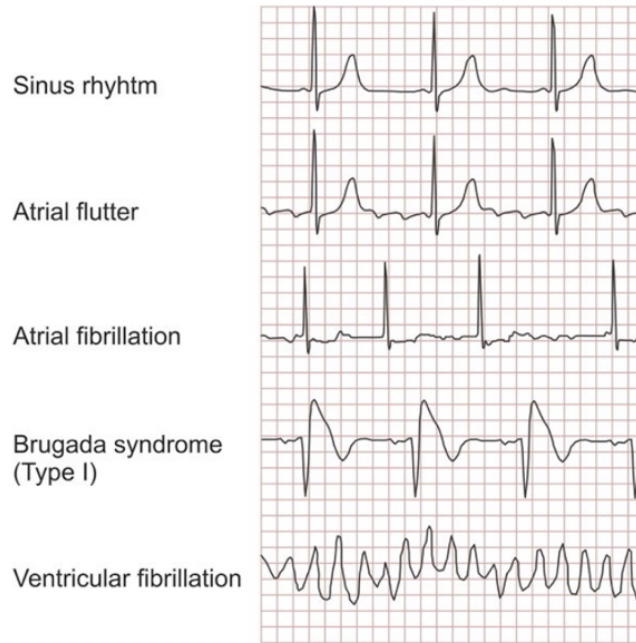


Figure 2: ECG of various arrhythmia ⁶

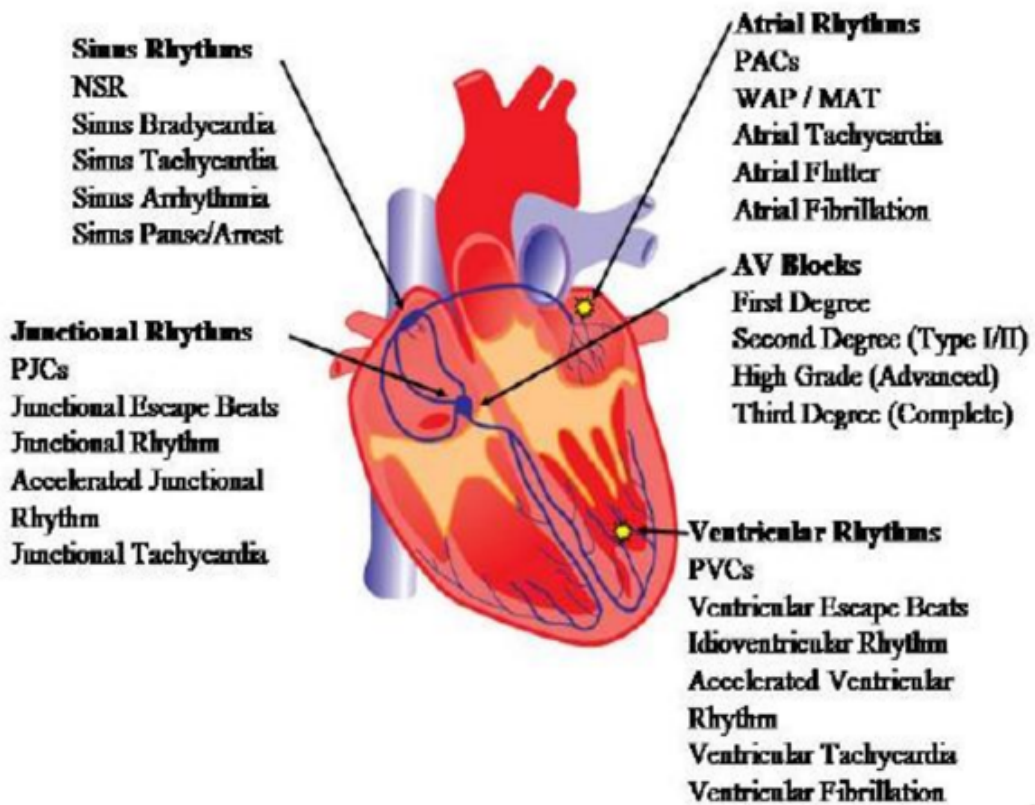


Figure 3: Occurrence of arrhythmia ¹³

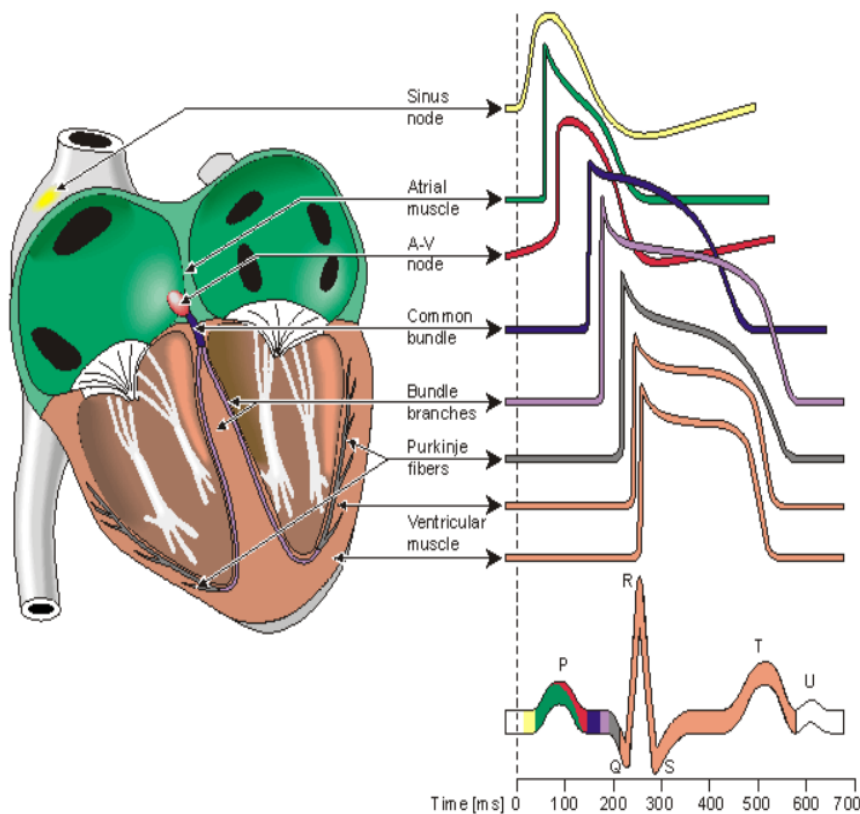


Figure 4: ECG of healthy Heart⁷

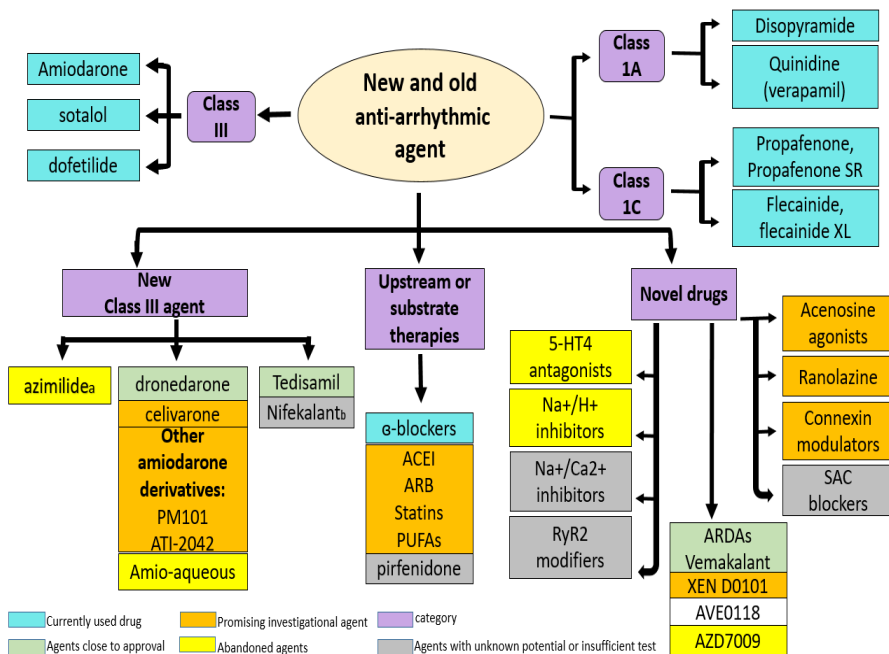


Figure 5: Classification of arrhythmia¹⁶

CONCLUSION

Arrhythmia is diagnosed by ECG and they are treated with anti-arrhythmic agent, pacemaker implantation and by herbal medicines. Allopathic treatment has various side effects, were

herbal plants have no adverse effect. In recent therapy pacemakers are implanted to regulate the normal heart beat in arrhythmia patients. Different classes of anti-arrhythmic agents are classified for treatment and ECG is used to diagnose the arrhythmia.

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