TACHYARRHYTHMIAS

SVT

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AF

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CLASSIFICATION OF TACYARRHYTHMIAS, MECHANISMS OF ARRHYTHMOGENESIS, AND ANTIARRHYTHMIC DRUGS

Definitions and Classification

Definitions

The term ***tachyarrhythmia*** (Greek *tachy* = fast and *arrhythmia* = rhythm disturbance) is used to describe conditions in which the heart rate exceeds the conventional number of 100 bpm, either in response to metabolic demand or other stimuli or due to disease. The term ***tachycardia*** requires more than 3 (or 5) consecutive beats, otherwise we are talking about premature beats or extrasystoles. A tachycardia is called ***sustained*** when lasting more than 30 sec, ***paroxysmal*** when it starts and stops abruptly and ***incessant*** or ***permanent*** when it occurs most of the time with annoying perseverance. The term ***chronic*** refers to incessant tachycardias which occur in a long-term basis and may result in cardiac dilatation, and impairment of ventricular contractility.

Classification of arrhythmias

Traditionally, tachyarrhythmias have been classified as ***ventricular*** or ***supraventricular***. The term supraventricular literally indicates tachycardias (atrial rates >100 beats per minute [bpm] at rest), the mechanism of which involves tissue from the His bundle or above.[1-3]
Traditionally, SVT has been used to describe all kinds of tachycardias apart from ventricular tachycardias and atrial fibrillation and has, therefore, included tachycardias such as atrioventricular re-entry due to accessory connections that is not, in essence, a supraventricular rhythm (Table 1). The term narrow QRS tachycardia indicates those with a QRS duration ≤120 milliseconds (ms). A wide QRS tachycardia refers to one with a QRS duration >120 ms.

Electrophysiologic Mechanisms of Arrhythmogenesis

The action potential

Pacemaker automaticity is due to spontaneous diastolic repolarization of phase 4 that generates rhythmic action potentials and determines the heart rate through various currents, including the I_f current.[4] There are no histologically specialized conduction tissues between the sinus and AV node, and the sinus impulse is transmitted via several exit pathways which electrically bridge the nodal tissue and atrial myocardium,[5] and then preferentially toward the AV node via muscle bundles with well aligned arrangement of cardiomyocytes.[6] By traveling through intercellular gap junctions (cell-to-cell connections), the excitation wave depolarizes adjacent atrial myocytes, ultimately resulting in excitation of the atria. The excitation wave

then propagates via the atrioventricular node and the Purkinje fibers to the ventricles, where ventricular myocytes are depolarized, resulting in excitation of the ventricles. Depolarization of each atrial or ventricular myocyte is represented by the initial action potential (AP) upstroke (phase 0), where the negative resting membrane potential (approximately -85mV) depolarizes to positive voltages (Figures 1 and 2, and Table 2).[7] The action potential is produced by transmembrane flow of ions (inward depolarising currents mainly through Na^+ and Ca^{2+} channels, and outward repolarising currents mainly through K^+ channels).[8] The resting potential of atrial and ventricular myocytes during AP phase 4 (resting phase) is stable and negative (-85 mV) due to the high conductance of the potassium channels. Excitation by electrical impulses from adjacent cells activates the inward Na^+ current that depolarises myocytes rapidly (phase 0). Transient outward K^+ current (phase 1) creates a notch during the early phase of repolarisation (Ito). Balance of the inward depolarising L-type Ca^{2+} current (I_{Ca-L}) and outward rectifier K^+ currents (slow-I_{Ks}, rapid-I_{Kr}, and ultra-rapid- I_{Kur}) forms a plateau phase (phase 2). Deactivation (closing) of the inward current I_{Ca-L} and increase of the outward currents creates phase 3 with final repolarization mainly due to potassium efflux through the inward rectifier I_{K1} channels, and the membrane potential returns to its resting potential (phase 4). The pacemaker current (I_f) contributes to action potential generation in the sinus node, and significantly determines heart rate.[9] I_f is a an inward current activated on hyperpolarization, and carried by Na^+ and K^+ current. It is called the funny current because despite being an inward current, it behaves like a pure K^+ current (due to superimposition of I_{k1}).[10] In Purkinje fibers, a similar behavior is expressed by current I_{k2}. I_f activation is accelerated by intracellular cyclic adenosine monophosphate (cAMP) levels, thus regulated by sympathetic and parasympathetic activity, which control synthesis and degradation of intracellular cAMP, respectively.

Opening and closing (gating) of ion channels enable transmembrane ion currents that consist of proteins called pore forming alpha (α)-subunits and accessory beta (β)-subunits. Terminology of genes encoding for these proteins describes their function. For example, the gene encoding the alpha-subunit of the cardiac sodium channel is called SCN5A: sodium channel, type 5, alpha-subunit. The alpha-subunit is termed Nav 1.5: Na^+ channel family, subfamily 1, member 5, and V means that channel gating is regulated by transmembrane voltage changes (voltage dependent).[1] Polymorphisms and mutations in genes encoding for ion channels are associated with slow conduction and QRS duration prolongation and, consequently, future development of cardiac arrhythmias.[11] [12] For a detailed description of acquired diseases due to mutations of genes affecting the action potential see Genetic Channelopathies and Bradyarrhythmias.

Mechanisms of arrhythmias

Although the initiation of tachycardias is a complex process depending on the interaction of several factors, the main mechanisms responsible for the initiation of tachycardias are thought to be cell membrane hyperexcitability ie enhanced automaticity or triggered activity, and reduction in cell to cell electrical coupling resulting in conduction block and reentry.[13] Clinical arrhythmias may have more than one mechanism for their initiation and maintenance.

Abnormal or enhanced automaticity is due to the maximum diastolic potential spontaneously becoming less negative (-40 to -60mV; phase 4 of the AP). In non-pacemaking myocytes, an imbalance between hyperpolarizing and depolarizing diastolic currents can occur under pathological conditions and lead to enhanced automaticity[14]. It is facilitated by partial membrane depolarization (e.g. by ischemia), hypokalemia and sympathetic activation.

Triggered activity is caused by early after-depolarizations or delayed after-depolarizations, ie depolarizing oscillations in membrane voltage that occur during or after complete repolarization of the action potential.[15] ***Early afterdepolarizations*** (ie occurring during phase 3 of the action potential) are voltage oscillations that occur during the repolarization phase of the action potential and are caused by reduced repolarization reserve. They may be due to electrolyte disturbances such as hypokalaemia, hypomagnesaemia, antiarrhythmic drugs (Class Ia and Class III), hypoxia and hypercapnia. Albeit promoted by bradycardia, early after-depolarizations are enhanced by adrenergic stimulation. Typically, they occur in the long QT syndrome. Moderate hypokalaemia is a potent inducer of ventricular arrhythmias mediated by early afterdepolarizations.[16] ***Delayed after-depolarizations*** (ie after complete repolarization of the action potential) are generated by diastolic Ca^{2+} release from the sarcoplasmic reticulum (SR), its intracellular store, to form intracellular "Ca^{2+} waves". [14, 17] They are facilitated by digoxin, artificial pacing, and adrenergic stimulation. Typically, delayed after-depolarizations occur in idiopathic outflow tract tachycardia and catecholaminergic VT. Triggered arrhythmias typically occur in structurally normal hearts but may also be seen in association with congestive heart failure and ischemic heart disease.[15]

Reentry denotes circulation of a wave of depolarization which is initiated when:

- Two functionally distinct pathways are present.
- Unidirectional block is induced in one pathway for example by a premature stimulus or by a physiological tachycardia.
- Sufficient slow conduction exists to allow recovery of excitability in the blocked pathway and permit retrograde conduction over that pathway and completion of the circuit.

The activation front travels around the obstacle (anatomic or functional). For re-entry to continue, the circuit length (set by anatomic obstacle size or tissue functional properties)

must be larger than the "*wavelength*" (WL). The WL concept is based to the mode of re-entry first described and named "*leading circle*".[14]

Reentrant mechanisms can be conventionally classified as:

Anatomical, due to defined loop. There is a large excitable gap between the crest and tail of impulse, ie properly timed stimuli can capture the circuit and reset the tachycardia. Typical examples are the atrioventricular reentrant tachycardias due to accessory atrioventricular connections.

Functional, due to altered cellular electrophysiological properties of myocardial tissue such as functional barriers of conduction block or decremental conduction leading to propagation failure. Functional reentry is exemplified by the "leading circle" hypothesis which may be important in atrial fibrillation.[18] In this model, the reentrant pathway is the shortest possible one and is determined on an instantaneous basis by refractoriness ahead of the activation wave front. The cycle length, therefore, is determined only by refractoriness and the excitable gap is as short as possible, independent of the cycle length. Functional re-entry may also take the form of "*spiral wave*", that have been implicated in both atrial and ventricular fibrillation.[19] [20]

Anisotropic, due to changes in microanatomical structures such as cellular coupling and fibre orientation heterogeneity which lead to anisotropic conduction or spatial inhomogeneity of refractoriness.[21] The length of the circular pathway is determined by electrophysiological-anatomical changes and there may be an excitable gap. This kind of reentry can be seen after myocardial infarctions.

There is evidence that more than 90% of the clinical tachycardias are due to reentrant mechanisms. Typical examples of reentry are tachycardias associated with the Wolff-Parkinson-White syndrome, the AV junctional reentry tachycardias, atrial flutter and most probably atrial fibrillation. In addition, certain forms of sustained monomorphic ventricular tachycardia such as VT in patients with coronary artery disease and bundle branch reentry in cases of dilated cardiomyopathy demonstrate reentrant circuit with fully excitable gap. Anisotropic reentry appears to play an important role in ventricular tachycardias occurring in the setting of chronic healed myocardial infarcts. Torsades de pointes and some forms of ventricular tachycardia complicating coronary artery disease are due to to after-depolarization induced triggered activity.

Reentrant mechanisms are postulated when programmed stimulation demonstrates that the tachycardia behaves in a manner similar to that of reentry. Thus, easy induction and termination by programmed stimulation as well as entrainment of the tachycardia usually indicate a reentrant mechanism. Tachycardias due to triggered activity may also be initiated and terminated by pacing, and may be distinguished from reentrant ones because progressively more premature stimulation may cause progressively faster triggered activity.

Usually distinction from reentry is difficult. Arrhythmias due to abnormal automaticity occur spontaneously or in response to isoprenaline and are not induced by programmed stimulation.

Haemodynamic, metabolic, and hormonal changes, and changes in autonomic function may contribute to increased arrhythmogenesis during **pregnancy** and women with prior history of arrhythmia may experience worsening of symptoms during pregnancy (Figure 3).[22]

Classification of Antiarrhythmic Drugs

The Vaughan-Williams classification, as modified by Singh and Harrison (Table 3) of the antiarrhythmic agents is outdated and over simplistic but, perhaps, more practical than that proposed by the Sicilian Gambit,[23] or other modernized classifications.[24] EHRA has produced a simplified version that, however, considers novel agents (Figure 4).[25] Most drugs have multiple modes of action. Proarrhythmic effects of antiarrhythmic drugs are shown in Table 4. Significant antiarrhythmic drug interactions are presented in Appendix 1.[26]

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