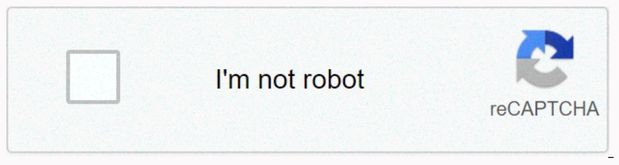

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Antiarritmicos anestesiologia pdf

[36-425-A-10] - Doi : 10.1016/S1280-4703(06)46236-8 M. Cannesson, O. Bastien, J.-J. Lehot ⁎ Service d'anesthésie-réanimation, h'pital cardiovasculaire et pneumologique Louis-Pradel, 28, avenue du Doyen-Lépine, 69500 Source, France *Responsible for correspondence. Welcome to EM consultation, referral of health professionals. Access to the full text of this article requires a subscription. Pages 13 Iconography 10 Videos 0 Other 0 Archived article, published in the treated Anaesthetic-Resuscitation Perioperative heart rhythm disorders are often observed, but rarely serious. Although they may appear in a healthy heart, they are often indicative of underlying heart disease that need to be examined and treated. In the case of a poorly tolerated rhythm disorder, the type of arhythmia should be quickly diagnosed to begin treatment as soon as possible; therefore, you need to know how to interpret an electrocardiogram. Medical care includes the treatment of the cause and flattering factors, as well as specific treatment. It should be remembered that some antiarrhythmic treatments may exacerbate or promote a pre-existing rhythm disorder (proarrrhythmia). In the face of a poor tolerance of a rhythmia with immediate commitment to the important prognosis, the only therapeutic resources are cardiovassion or overstimulation suppression. The full text of this article is available in PDF. Keywords : Arrhythmia, Tachycardia, Bradycardia, Proarritmia, Perioperative, ComplicationEsquema Writers would like to thank Prof. G. Kirkorian for the iconographic material. © 2006 Elsevier SAS. All rights reserved. Welcome to EM consultation, referral of health professionals. Access to the full text of this article requires a subscription. Welcome to EM consultation, referral of health professionals. Purchasing items is not currently available. Already signed to this treaty? Connecting or creating an account Vaughan Williams classification is based on the main mechanism of action of each drug, but, as we'll see later, several antiarthythmics have more than one mechanism of action. Other antiarthythmics such as adenosine, digoxin or magnesium are not found in the classification. Group I: To + channel blockers. They are subdivided into groups a, b, c, based on the changes they cause in the Weidman curve. These differences are due to the rate of dissociation of the channel (Tau, constant association drug channel dissociation), which gives them different electrophysiological characteristics: because the Tau is intermediate, in the Ib is short (less than 1 sec) and the drugs of the Group Ic are the ones that possess the slower dissociation. The antiarthythmics of the Ib group have a greater affinity for Na+ channels when they are in an inactive state, while the other groups do so across the open-state channel. delays phases 0 and 3 of the action potential. They depress the membrane's responsiveness, which reduces the phase 0 slope. They reduce excitement by increased the effective reflection period. Quinidine, procaineamide, disopiramide. Quinidine: Along with Quinine, they were withdrawn alkaloids from the quintine bass. It has antipyretic, antimalarial, and vagolytic effects. It is the patron drug of group Ia, but has fallen into turmoil for its multiple adverse effects and interactions. It blocks Na+ channels, in the open state of the channel, with an intermediate receptor drug dissociation Tau coefficient (1 to 6 sec), reducing the rate of ascent of phase 0. It also blocks potassium channels by prolonging phase 3, and can lead to early post-depolarization formation and tip-turning development. The anticholinergic effect would explain some antiarrhythmic mechanisms, and alpha adrenergic blockage would be responsible for dropping peripheral vascular resistance with hypotension and reflex tachycardia (Luys paradoxical ticardia). Increases basal heart rate and aturiculicular conduction. Stretch DPA and PRE, more intensely at slow frequencies. By reducing excitement and slowing drive it is able to avoid or interrupt re-entry circuits. It has a negative inotropic effect that can decompose the patient with heart failure. The electrocardiogram shows widening the QRS complex and prolonging the QT interval. Ib: They have a virtually zero effect on phase 0. Accelerate the speed of phase 3. Increase the PRE/DPA ratio by shortening the DPA. They depress excitement. To understand the mechanism of action of Ib group drug, their actions must be dynamically interpreted: initially, they join the sodium channel of a cell that has already depolarized, transferring the channel from the open state to the inactive state. At that moment, the antiarthythmic was joined, and dissociated almost immediately. This change causes the channels to be transmitted faster in their closed state, and what is associated with this causes an intense output of phase 3 potassium. With all these effects, for the next cycle, the cell is part of a more electronic membrane potential (since it is hyperpolar), and repolarisation is fast, decreasing the relatively receptive period (the period in which the cell is excited). These drugs are used in ahythmias whose mechanisms include ventricular re-entry. Lidocaine, phenicin (diphenylhydantoin), mexiletine. Lidocaine: This is a local anaesthetic that systematically has antiarrhythmic effects, being the dorga of choice to suppress ventricular arrhythmias in the context of IAM and heart surgery. Block inactive sodium channels with ultra-fast recovery kinetics (tau less than 1 sec). It has fast start-and-end kinetics, and affects the normal automation of the sinus node. It inhibits normal and abnormal forms of automatism, as well as early or late post-depolarisations at the level of Purkinje fiber. Certain factors, such as a reduced ph, an increase in extracellular K+, or reduced membrane potential (all present in iscemia situations), increase the ability of lidocaine to block INa. It can transform a one-way blockage into biiration during iscemia (homogenization effect), thus preventing the development of ventricular reentry mechanisms, such as UIF. It has little or no effect on atrial substance, on automation, and on per-road channels. In high doses it has a myocardial depressive effect. Ic: Delayed phase 0 more intensely than group Ia. They had minimal effect on phase 3 of the action potential. They are powerful depressants of membrane responsiveness, decreasing driving speed, and prolong DPA and PRE. Flecainide, encainide, propafenone, moricizine. Flecainide: This is the standard drug of the group. It has a major depressive effect on the rapid Na+ channel, and the Icc potassium channel reduces the Vmax, in a dose-dependent manner. The dissociation of the canal drug is very slow, with a Tau of 10 to 30 s (climber 4 to 8s, lidocaine 1s). It shortens DPA in Purkinje fiber, but prolongs it into the ventricular muscles, actions that, depending on the circumstances can increase or reduce electric heterogeneity, and cause or suppress arhythmias. Stretch row on all heart rates. It has an important contract point depressive effect, limiting its uses. Encainide and Lorcainida are compounds similar to Flecainide. The encainide does not drain the function of the myocardium. Propafenone: Block the rapid sodium current in Purkinje fiber and to a lesser extent in the myocardium. His constant Tau of dissociation has been slow. Propaphenone reduces excitement, and suppresses spontaneous automatism and enabled activity. It is poor blocker of Icon potassium currents, and has poor adrenergic beta blockage and calcium channel blockage. Expand pr and QRS. The QT interval is extended only depending on the duration of the QRS. It has a negative inotropic effect. Reduces heart output and increases peripheral resistance. Group II: Beta blockers. They inhibit the actions of adrenaline on the myocardial beta 1 receptor, depressing automatism, excitement and management. Slows down the depolarization rate of the automatic cells of the sinus and AV nodes. They also reduce the excitement of ventricular myocardial cells. They are described in detail in the beta-blockers section. Group III: Slow phase 3 of the action potential. As a general characteristic, they share the significant increase of PRE by blocking potassium channels, which reduces excitement. This includes the bretilium, sotalol, as well as more modern molecules such as dofetilia, ibutilide, dronedons, and vernacalant. Group III drugs prolong PRE by blocking potassium channels (Icc, Iks) responsible for repolarising in phase 3 of the action potential. They are useful in reversing reentry arrhythmias such as atrial fibrillation. Sotalol: This is a channel blocker of K+ (d-Sotalol) and also non-selective beta adrenergic blocker without intrinsic sympathomimetic activity or the stabilization of effect of membranes (d,l-Sotalol). Blocking the Icc stream prolongs the DPA and PRE. Delay NS and NAV-level drive. Dofetilide: The unique electrophysiological effect of Dofetilide is the blockage of the Icc potassium channel, which is responsible for rapid late grinding current during the repolarization phase, especially in atria. Prolong refrination without slowing down management. Through this mechanism, dophethomicism prolongs the QT interval, in a dose-dependent manner. No other electrocardiographic changes are observed, as well as any hemodynamic effects. Ibutilide is a derivative of parental use that also blocks an internal slow current sodium. Vernakalant: This is a recently developed drug, which blocks the Icc stream, and to a lesser extent the ITO current and sodium current. It was developed as an alternative to the rapid reversal of atrial fibrillation, parenteral from emergency rooms, achieving a high turnaround rate. Since it does not accumulate, it has no electrophysiological effect on follow-up. Dronedarone: It's a derivative of amiodarone, with certain differences: it's less fat-soluble and has shorter half-life with lower rate of interactions and adverse effects (it also lacks thyroid effects since it has no iodo in its molecule). Its usefulness was evaluated in patients with atrial fibrillation, but in those who left ventricle dysfunction, it had an increase in mortality, so its use was limited to patients without heart failure. Group IV: Calcium blockers. Non-dihydropiridic L-type calcium channel blockers reduce the slow entry of Ca++ responsible for phase 0 of calcium feather, reduce driving speed, increase PRE and reduce automation. Slows down the depolarization rate of the automatic cells of the sinus and AV nodes. The most widely used verapamil and diltiazem. Other antiarthythmics not included in the Vaugham Williams classification: Adenosine: Adenosine is an endogenous core with various effects on the body. By binding to its A1 receptors, it produces potassium channel opening, hyperpolarizing the membrane potential of atrial cells and sinus and AV nodes (similar to Acetylcholien). This mechanism lowers baseline heart rate as well as riding at the to be effective for the treatment of supraventricular arrhythmias (re-entry into the AV node, orthodoromic ticardia of Wolff Parkinson White syndrome). Digoxin: It has an antiarthythmic effect fundamental in reflex form, by activating the parasympathetic system, slowing the discharge rate of the sinus node and av node. As an antiarchymic its main indicator is the reduction of the ventricular response to supraventricular thyrrhythmias. Ivabradine: This is a recently designed drug, which inhibits the ash stream (sodium and potassium conduction, funny) from the sinus node, so it reduces heart rate at the sinus level without affecting hemodynamic effects. It is used ore with an interdose interval of 12 hours, and its usefulness is to reduce heart rate in patients with systolic heart failure, and in patients with stable chronic angina. It can also be useful in patients with inappropriate sinus tachycardia. 2 comments comments