

Dysrhythmias under General Anesthesia and Their Management

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Cardiac arrhythmias during anesthesia are common and almost benign, with the incidence ranging from 60 to 90%. Arrhythmias are one of several significant predictors for severe cardiovascular outcomes. It is essential, therefore, for the anesthetist to evaluate patients at risk preoperatively with a careful history and to have an appropriate knowledge concerning the etiology, electrophysiology, diagnosis, drug effects and treatment of arrhythmias.

Keywords: Anesthesia; Cardiac arrhythmias

Dysrhythmias incidence is an important cause of complications during anesthesia because during this period there are some clinical conditions that may generate changes in cardiac regularity [1]. These rhythm changes might be due to a prime etiology or to reversible reasons that should be fixed. The incidence of cardiac dysrhythmias differs according to the works, kind of surgical technique, and the patient. In a study with 17,201 patients those who had general anesthesia, dysrhythmias (tachy, bradycardia, or else) were detected in 70.2%, of which just 1.6% needed treatment [1]. A large number of patients who had non-cardiac surgeries might have advance dysrhythmias [2]. The occurrence of atrial fibrillation is small after investigative thoracotomy, but in older patients who have lobectomy, pneumectomy, or esophagogastrectomy the occurrence increases from 12% to 33% [3]. Though arrhythmias often occur even in controlled patients, those needing treatment are uncommon [4]. In common, supraventricular rhythms, like atrial rhythm, atrioventricular junctional rhythm, and drifting pacemaker, are minor and not necessitate treatment. Ventricular arrhythmias must be regarded as a sign of real instability [5].

The Holter monitor system was used to correlate the onset of arrhythmias during surgery with the type and length of surgical procedure, anesthetic agent, and technique of anesthesia. The incidence of significant arrhythmias during general anesthesia and surgery was found to be 61.7 per cent. The most common were wandering pacemaker,

isorhythmic, A-V dissociation, nodal rhythm, and premature ventricular systoles. Precipitating factors which cause arrhythmias were the types of anesthetics, intubation, hyperventilation, and duration of surgery. An unexpected finding was the fact that there was no significant difference of arrhythmias occurring in the normal patient compared to those with pre-existent heart disease or arrhythmias [6]. This review was designed to establish the incidence of arrhythmias, the types of arrhythmia happening during surgery, and the relationship of arrhythmias to precipitating factors. Dysrhythmias represent significant reason of perioperative complications because during this period there are several clinical situations that may trigger changes in cardiac rhythm [7].

Mechanisms of Dysrhythmias:

Dysrhythmias are secondary to changes in cardiac ion channels (sodium, calcium, and potassium channels) and adrenergic receptors are the targets. To better understand the mechanism of dysrhythmias and antidysrhythmic agents, one should remember the role of action potential [8]. The initial period of the action potential corresponds and it initiates the conduction in the cardiac tissue. In atria and ventricles the impulse originates in the sodium current. In the sinoatrial (SA) and atrioventricular (AV) nodes depolarization is produced by calcium current, representing repolarization; the plateau (maintained by calcium current) and its end is maintained by potassium current, Nodal cells undergo spontaneous depolarization while atrial and ventricular tissues are hyperpolarized. Dysrhythmias may be due to changes in the formation of the electric impulse (automaticity) or in conduction. Abnormal impulse generation can occur in the sinus node or in ectopic foci. Automaticity refers to abnormal atrial or ventricular depolarization during the repolarization or resting period of the action potential, some molecular substrates, such as prolongation of the QT interval and low potassium (K⁺) concentrations can trigger automaticity. Mutations in ion channels are responsible for repolarization, and that can prolong it, make cardiac cells more sensitive to dysrhythmias [9].

Risk factors for dysrhythmias:

Risk factors for the development of dysrhythmias can be classified as adjustable and non-adjustable. The non-

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modifiable risk factors include dilated cardiac diseases, ischemic cardiomyopathy, autonomic changes of the conduction system, polymorphism of ion channels, or congenital long-QT syndrome (LQTS).

Among modifiable factors there are electrolyte changes: K⁺ changes can generate increases in automaticity and abnormal formation of impulses. Changes in serum K⁺ levels are closely related with the development of dysrhythmias, and abrupt changes are less tolerable than chronic changes. The relationship between preoperative K⁺ and perioperative adverse events was demonstrated by Wahr et al [10].

Differential diagnosis of dysrhythmias:

Supraventricular tachycardia can be defined as sustained, non-sinus related, acceleration of the cardiac rhythm, originating above the AV node. On the other hand, autonomic tachycardia is rare and can be defined as a tachycardia initiated and sustained by an ectopic focus. Automatic atrial tachycardia is a type of automatic tachycardia that involves primarily the atrial tissue. The continuous type is usually symptomatic and often results from dilated cardiomyopathy. The repetitive type is frequently interrupted by periods of sinus rhythm, is less severe, and only becomes symptomatic during periods of very fast heart rate (HR). There is a tendency to distinguish tachycardia as ventricular and supraventricular according to their origin. Patients with electrocardiographic changes compatible with wide-QRS regular tachycardia represent a diagnostic and treatment challenge [11].

Management:

Although severe bradyarrhythmias require treatment they have been reported in only 0.4% of 17,021 patients undergoing general anesthesia. These patients respond well to pharmacological measures or transesophageal atrial pacemaker and rarely require permanent transvenous pacemaker even in the presence of asymptomatic fascicular block or preoperative left branch block [12]. On the other hand, it has been estimated that perioperative tachyarrhythmias affect almost one million elderly Americans, being associated with significant morbidity. A large number of patients undergoing cardiac or non-cardiac surgeries have these dysrhythmias. Because all antiarrhythmics have the potential to cause dysrhythmias, they should be used with caution, and correction of electrolyte imbalances and prevention of bradycardia are required in the management of dysrhythmias [13]. Every patient undergoing oral surgery under general anesthesia requires continuous monitoring of cardiac rhythm on an electrocardiograph so that early detection, diagnosis, and treatment of a dysrhythmia is possible [14]. The necessity to treat a dysrhythmia is for the purpose of hemodynamic and cardiovascular stability. Treatment depends on the cause and effects. In order to find out these factors continuous monitoring of the patient is important. As mentioned, in addition to the cardiac rhythm, observation of the patients color for cyanosis or pallor, of respiration for respiratory obstruction, breath-holding, light or deep anesthesia, plus continuous monitoring of oxygen saturation, end-tidal carbon dioxide, and blood pressure should be stressed. Cardiac dysrhythmias under dental anesthesia usually do not produce hemodynamic and cardiovascular instability and resolve without any intervention [15].

Types of participants:

We searched the published studies from 1970 to 2017 by using these keywords: Cardiac dysrhythmias and Anesthesia; Arrhythmias, Cardiac disease. The publishers chose 95 patients to do the research. The study involved patients aged 6 months to 70 years. This study includes 11 studies which are RCTs. The goal was to review studies: to review the most relevant aspects of cardiac dysrhythmias, as well as establishing the cause-effect relationship between drugs used in the during anesthesia. All the selected reviews were limited to the English language.

Results and Conclusion

About 11 articles are considered in the literature review. Despite the fact that sinus tachycardia was excluded from consideration, 95 patients (61.7 per cent) had one or more abnormal cardiac rhythms, totaling 195 arrhythmias during the surgery). Control electrocardiographic tracings (made before induction of anesthesia) revealed that 12 patients already had an arrhythmia, but only out of three of these arrhythmias changed during the operation. Transient S-T and T wave changes indicative of cardiac ischemia were recorded in 16 patients (10 per cent). In order of frequency the arrhythmias observed were wandering pacemaker (43 patients), isorhythmic A-V dissociation (34 patients), nodal rhythm (29 patients), premature ventricular systoles (28 patients), sinus bradycardia (22 patients), premature supraventricular systoles (19 patients), supraventricular tachycardia (five patients) and ventricular tachycardia (five patients) [16]. After the diagnosis of dysrhythmia the distinction between benign dysrhythmia and those that carry the risk of sudden death is fundamental. The choice of anesthetic agents is important to minimize episodes of tachydysrhythmias. Prevention is as important as treating dysrhythmias. Recognition of risk factors, adequate selection of drugs for each patient and monitoring represent the most important steps in prevention. In patients at risk for prolonged QT, recent baseline ECG is important. Electrolytes should be monitored and corrected whenever necessary. Verification of renal and liver function is important in patients using medications that can cause dysrhythmias. Moreover, doses should be adjusted whenever necessary [17]. In patients at risk for prolonged QT, recent baseline ECG is important. Electrolytes should be monitored and corrected whenever necessary. Verification of renal and liver function is important in patients using medications that can cause dysrhythmias. Moreover, doses should be adjusted whenever necessary. Prolonged QT interval should be closely monitored by the anesthesiologist, as several drugs can potentially prolong it, and the interaction among them can be catastrophic. Supraventricular tachycardia responds to treatment with adenosine, while AF does not, although in both cases the frequency is controlled by beta-blockers or calcium channel blockers. The use of class Ia drugs is gradually declining due to its unfavorable risk/benefit ratio. Class Ib drugs such as lidocaine are still widely used in the operating room to treat ventricular tachydysrhythmias [18].

Discussion

Incidence of Arrhythmias: The incidence of arrhythmias reported by others on patients monitored at consecutive operations ranged between 10 and 29 per cent [19]. In our

series we could find this incidence to be 61.7 per cent. Our distribution of arrhythmias also appears to be different from that found by other investigators [19]. We found the most frequent arrhythmias to be wandering pacemaker, A-V dissociation and nodal rhythm. It is possible that other studies did not reveal this because these arrhythmias are difficult to be observed in a fast moving image on the oscilloscope [19]. They probably escape detection because the QRS configuration is unchanged and the P wave is hard to follow due to its low voltage. Precipitating Factors: It was possible to determine the factors precipitating arrhythmias by correlating the time of their occurrence with the onset of the arrhythmia. The principal factors seemed to be types of anesthetics, duration of surgery, intubation and hyperventilation. Wandering pacemaker, A-V dissociation and nodal rhythm appear to have almost the same precipitating factors: general anesthesia, hyperventilation, and intubation. Numerous reports indicate that various arrhythmias may occur during endotracheal intubation, particularly nodal rhythm and premature ventricular systoles [20]. We must emphasize that even though hyperventilation may produce arrhythmias, we do not suggest hypoventilation as a substitute because of its serious consequences. Premature systoles have been observed with all anesthetic techniques and agents. They are more likely to be observed during halothane and cyclopropane anesthesia, in the presence of acidosis, after catecholamine administration, or in connection with the surgery [21]. Dr. Morrow presented a very concise and informative discussion of the mechanisms of arrhythmias during anesthesia; however, they have been somewhat concerned by his use of lidocaine “to buy time” in the case of persistent ventricular arrhythmias which do not respond to discontinuance of anesthesia and hyperventilation. As Dr. Morrow indicated, ventricular ectopic beats may be a reflection of supraventricular pacemaker failure. Whether this represents simply pacemaker escape or a defense mechanism, it appears that the empiric suppression of this ventricular activity without some assurance of an adequate supraventricular pacemaker is fraught with considerable hazard. We may well be depriving a patient of his only source of rhythmicity, while our therapy might better be directed toward boosting failing pacemaker activity and conduction through the use of a beta-adrenergic drug such as isoproterenol [22].

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