Pharmacologic and mechanical support of the circulation

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The main sources of arrhythmias and rate changes are sympathetic overstimulation, electrolyte disturbances, and cellular damage. Cellular damage, reversible or not, may result from the added actions on the basic disease of ischemia, edema, and hypoxia induced by causes such as direct manipulation of the heart, ischemic injury during cross-clamping of the aorta, and electrolyte disturbances.

The alert anesthesiologist can prevent, correct, or at least minimize the consequences of these rate and rhythm changes by proper anesthetic management and specific drug therapy. The most common situations in which the anesthesiologist can intervene in the general setting of heart surgery are discussed.

Bradycardia

In principle, bradycardia is safe, especially for the coronary patient. The rate is mainly under the influence of the sympathetic tone and the pharmacologic effects of drugs such as beta-blockers, fentanyl, halothane, and digoxin.

Before treating bradycardia, its general impact on circulation must be assessed for signs of danger, premature ventricular contractions, signs of reduced cardiac output, atrioventricular block, and imminence of complete block.

In managing bradycardia, treatment is not necessary in all cases, but it is important not to exacerbate it. Treatment consists of atropine, isoproterenol (Isuprel), and the use of pacemakers. As a word of caution, beta-blockers may prevent expected increase in rate in presence of hypovolemia.

Tachycardia

Before treating tachycardia, remember that it may be a defense mechanism and can be a normal response to hypovolemia, hypoxemia, and tamponade. These above conditions must be ruled out before a reduction in the rate is attempted. Also, tachycardia may be a normal response to what could be called "iatrogenic sympathetic overstimulation" such as stress of entering the operating room, stress of intubation and operation. The latter is essentially preventable by (1) good mental preparation of the cardiac patient (importance of the preoperative visit), (2) heavy premedication, (3) maintenance of beta-blocking therapy, (4) preference of fentanyl over morphine for induction of anesthesia, (5) preventive management (supply increased analgesia ahead of time), (6) good constant monitoring, (7) liberal use of nitroglycerin, and (8) use of propranolol if the patient is not on beta-blocker therapy.

Arrhythmias

There is general agreement that most arrhythmias result from disturbances in impulse generation, impulse conduction, and a combination of both.

In impulse generation (ectopic tachyarrhythmias), latent pacemakers may accelerate in frequency or nonpacemaking cells may develop phase IV spontaneous depolarization as a result of anoxia, fiber stretch, edema, or sympathetic overstimulation.

Impulse conduction (reentrant arrhythmias) can occur when the following conditions are present: (1) asymmetrical conduction: an influx (from sinus or ectopic origin) meets an area of blocked conduction, (2) unidirectional block; this area blocks the influx from one direction but not from the opposite direction, and (3) slow conduction: conduction must be slow enough for the returning influx to find the area that conducted normally completely repolarized and capable of conduction.

With a combination of both mechanisms, the mechanism that maintains the arrhythmia may not be the same that triggered it; thus, extrasystoles from enhanced automaticity may trigger tachyarrhythmias of the reentrant type.