SUMMARY

Many complex causes are involved in the process of cardiovascular failure after cardiac surgery, and there is also a common possibility to have several factors interacting with each other in the same patient. Sometimes, causes interact at the same time and often they follow each other in a sequence of events. The most common causes include: right-sided failure, low cardiac output syndrome, cardiac arrhythmias, recurrent ischemia, hypertension, delayed tamponade, constrictive pericarditis, post-pericardiotomy syndrome, chylopericardium ...

Deterioration in cardiac function after coronary surgery is documented in several studies. Although surgical techniques and operative care should result in better postoperative function, early biventricular dysfunction is a common finding. Significant decline in either left ventricular or biventricular in the first post-operative hours is hazardous, with gradual return to preoperative values within 8-24 hours. Protection of the myocardium during surgery is central to the prevention of postoperative dysfunction. Although few surgeons continue to employ intermittent ischemic aortic cross-clamping (no cardioplegia or hypothermia), most still use some combination of hypothermia and crystalloid or blood cardioplegia to arrest the heart and reduce its metabolism.

Support of the failing heart can be implemented, either by physiological support or, in the worst scenario (when the heart fails to compensate itself), pharmacological and mechanical support can be introduced. Pharmacological support includes all

drugs that maintain "acceptable" hemodynamics. The role of the anesthetist/intensivist is to continuously assess the status of the patient so as to mix appropriately inotropic drugs and vasodilators/ vasoconstrictors without harming the fragile physiology of the recovering patient.

Main inotropes available to the intensivist are: epinephrine, norepinephrine, isoproterenol, phenylephrine, dopamine, dobutamine and the newest agents - phosphodiesterase inhibitors (amrinone, milrinone, enoximone) and dopexamine.

Levosimendan is a member of a new class of drugs that are known as calcium sensitizing drugs. It is a revolutionary drug that exclusively increases sensitivity of cardiac myofilaments to any given concentration of intracellular calcium, thus increasing inotropy dramatically.

Cations (calcium, magnesium) can be independently used to potentiate the action or limit potentially hazardous side effects of other cardioactive drugs.

Thyroid hormone and arginine vasopression (AVP) are two experimental agents that have doubtful promising potentials in the rapid and safe recovery of the coronary patient in the acute postoperative period.

Several vasodilators are also used either independently or in conjunction to control episodes of hypertension and reduce the afterload in the immediate postoperative period and include: sodium nitroprusside, nitroglycerine, angiotensin-converting enzyme inhibitors, hydralazine, calcium-channel blockers and the latest addition to this group - fenoldopam - which is a dopaminergic agonist (DA_1) with selective coronary, renal, mesenteric and peripheral arterial vasodilator action. It is

particularly valuable as it does not affect renal blood flow despite an effective reduction in systemic afterload.

Mechanical support involves the use of the intra-aortic balloon and the circulatory assisting devices. The intra-aortic balloon is the most popular as it provides hemodynamic support and/or control of ischemia both before and after surgery. In contrast to most inotropic agents, it provides physiologic assistance to the failing heart by decreasing myocardial oxygen demand and improving coronary perfusion. Circulatory assisting devices include: right, left and bi-ventricular assisting devices. These devices provide flow to support the systemic or pulmonary circulation while awaiting recovery of myocardial function.