Management of Cardiac Dysfunction in ICU

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The abnormalities of cardiac function are often seen during operation or postoperatively in ICU. These abnormalities include hypotension, low output syndrome, perioperative myocardial infarction, hypertension and dysrhythmias. Analysis of cardiac function requires evaluation of major determinants of cardiac output e.g, cardiac rate and rhythm, preload (the degree to which the myocardium is stretched before the onset of contraction), after load (the sum of external factors that oppose the shortening of myocardial fibres), and contractility.[1]

1. **PRELOAD ABNORMALITIES.** Perload abnormalities are common after major surgery. The cause of hypotention is usually hypovolaemia. Use of central venous and pulmonary artery catheters will facilitate diagnosis of preload abnormalities.

   Volume replacement should rapidly reverse low preload associated with hypotension. Fluid selection (blood, FFP, hydroxy ethyl starch, or crystalloid) is based on hematocrit value and urine output. A high preload (CVP greater than 20 mm Hg) should suggest overtransfusion and impaired myocardial compliance.

2. **AFTERLOAD ABNORMALITIES.** Increased afterload levels are commonly associated with hypertension, hypothermia and vasoconstriction.

VASODILATORS

Vasodilators may enhance cardiac output significantly. Generally their site of action i.e venous bed, arterial bed, or both classify vasodilators. Vasodilators that affect venous capacitance bed primarily (e.g Nitrates) reduce preload and myocardial oxygen consumption but may influence cardiac output slightly. The drugs acting mainly on arterial bed, (e.g, hydralazine, phentolamine) will decrease systemic venous resistance and increase cardiac output without affecting left ventricular end diastolic volume (LVEDV). The drugs with mixed action (e.g, nitroprusside, prazosin, captopril) have arterial and venous dilating properties decreasing LVEDV, myocardial oxygen consumption, and systemic vascular resistance while raising cardiac output. Vasodilator use should always follow the principle of minimizing afterload and maximizing preload:

(1) **SODIUM NITROPRUSSIDE:-** It acts directly on smooth muscle fibres in the vascular wall, and its effects are noted in both arterial and venous circulation. It can decease systemic vascular resistance, pulmonary vascular resistance, pulmonary artery pressure, pulmonary capillary wedge pressure, and CVP, and in this fashion may cause significant increase in cardiac output. It has no effect on myocardium and no effect on alpha or beta receptors.[3]

(2) **NITRATES:-** Predominant vasodilator effect of nitrates is on the large veins, with less marked effect on the arteries. Flaherty and colleagues.[4] compared the effects of nitroglycerine in patients developing hypertension following coronary bypass surgery. Moderately high infusion rates of nitroglycerine were required than nitroprusside. Haemodynamic responses to the two vasodilators were similar, except that increase in cardiac output was greater with nitroglycerine.

(3) **HYDRALAZINE:-** It is a direct vasodilator of vascular smooth muscle. It has little effect on venous capacitance vessels.

ABNORMALITIES OF CARDIAC CONTRACTILITY.

A variety of factors are responsible for depressed ventricular function e.g, preexisting left or right ventricular dysfunction, acute MI, hypoxaemia, acid base imbalance, allergic reaction etc. A cardiac index below 2L/min/m^2 in the presence of adequate rate, ryhthm, preload, and afterload will generally require use of an inotropic agent. The agents commonly used to treat reduced cardiac output are:-

CATECHOLAMINES

(1) **DOPAMINE.** Dopamine is a naturally occurring catecholamine that is both a chemical transmitter and an intermediate in synthesis of adrenaline and noradrenaline. Dopamine specific receptors have been identified in renal, mesenteric, coronary, and cerebral vessels, as was published by Goldberg and Rafter.[7] One of the inotropic effects of dopamine is the release of endogenous noradrenaline, once it is depleted, the inotropic

effect is no longer present. In addition, administration of dopamine over 24 hours may result in a loss of its inotropy, while alpha adrenergic and dopaminergic stimulation may persist. Dopamine specific receptor effects are noted at very low infusion rates of 0.5 to 1 microgram per kg per min, becoming maximal at 2 to 3 microgram per kg per min. Dopamine can cause tachycardia and dysrhythmias. In fact dopamine is not the drug of choice in the treatment of severe cardiac dysfunction.

(2) **DOBUTAMINE:-** It is a synthetic inotropic catecholamine that can selectively increase cardiac contractility without causing positive chronotropic effects. It acts on B-adrenergic receptors in myocardium. It does not stimulate renal dopamine receptors to cause vasodilatation, however, it increase renal blood flow by increasing cardiac output. Sethna et al[8] studied the effects of dobutamine (5.1 ± 2.5 ug/kg/min) in patients with low-output syndrome. Cardiac index increased 40% with slight increase in heart rate and decrease in systemic vascular resistance. Myocardial oxygen consumption was increased by 29% with a corresponding 35% increase in myocardial blood flow. No change in coronary sinus oxygen or lactate extraction occured?

(3) **ADRENALINE:-** It has prominent actions on myocardium and smooth muscle, stimulating both alpha and beta adrenergic receptors. The result is increased heart rate and markedly increased cardiac work and oxygen consumption[9]. Adrenaline may precipitate premature ventricular contractions (PVC's) and ventricular fibrillation(VF). Adrenaline at a dose of 1-2 ug/kg/min primarily causes B_1 and B_2 adrenergic stimulation. At doses of 2-10 ug/kg/min the alpha adrenergic stimulating effects become more prominent. At high doses (10-20 ug/kg/min) the intense alpha mediated vasoconstriction masks the Beta cardiac stimulating effects. Adrenaline is very useful in low-output syndrome, particularly in patients not responding to moderate doses of dopamine and dobutamine, but it's s usefulness can be limited by development of tachycardia and dysrhythmias.

(4) **ISOPRENALINE:-** Its $beta_1$ and $beta_2$ actions are unopposed by alpha stimulation, thus it causes marked dilation of vascular smooth muscle bed. It is useful in stimulating cardiac pacemaker cells in patients with bradydysrhythmias and A-V blocks.

(5) **COMBINED INOTROPIC AND VASODILATOR THERAPY.** Combination of dopamine, dobutamine or adrenaline with nitroprusside or nitroglycerin have been the most popular methods. With mild postoperative dysfunction dobutamine combined with nitroglycerin may be adequate. With more profound depression of cardiac function, adrenaline with nitroglycerine is or nitroprusside may be required.

(6) **XANTHINE AND PHOSPHODIESTERASES:-** Aminophylline, Amrinone, Milrinone have been developed, and used as "Inodilators" (Inotrope and Vasodilator). Their role in cardiac dysfunction still evolving.

(7) **INTRA AORTIC BALLOON COUNTERPULSATION (IABP).** Counterpulsation is a technique in which aortic pressure is reduced in systole thereby reducing after load and thus facilitating LV ejection and increased diastole, raising mean arterial pressure, and improving diastolic coronary flow. IABP is indicated in the treatment of low output syndrome not responsive to pharmacological manipulations.

CONCLUSION

A high index of suspicion is necessary to reveal the early cardiac dysfunction perioperatively. An aggressive intervention following determined diagnostic efforts will stand the best chance of altering patient outcome positively.

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