

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<sup>8</sup> 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 occurred?
- (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<sup>9</sup>. 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<sub>1</sub> and B<sub>2</sub> 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 usefulness can be limited by development of tachycardia and dysrhythmias.
- (4) **ISOPRENALINE:-** Its beta<sub>1</sub> and beta<sub>2</sub> 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 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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# *Mechanical ventilation*

## *An Overview*

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Absolute indications for initiation of mechanical ventilation include apnea and the need for administration of paralyzing agents. Mechanical ventilation is also usually required in patients who exhibit ineffectual respiratory efforts, inspiratory muscle fatigue, refractory hypoxemia, or progressive hypercapnea with acidosis. More specific indications are advocated by some physicians, and these include a vital capacity less than 15 ml per kg, FEV<sub>1</sub> less than 10 ml per kg, PaO<sub>2</sub> less than 60 mmHg on supplemental oxygen, and PCO<sub>2</sub> more than 55 mmHg.

Although a number of types of different ventilators exist, volume-cycled ventilators (i.e. ventilators that deliver a preset tidal volume) are by far the most common. When mechanical ventilation is instituted with such a ventilator, tidal volumes in the range of 10 to 15 ml per kg are reasonable. Tidal volumes of this size minimize atelectasis but at the upper end of this range may generate high peak airway pressures. In general, if peak pressures exceed 40 cmH<sub>2</sub>O at higher tidal volumes, the tidal volume should be lowered to the range of 10 to 12 ml per kg to minimize the risk of barotrauma.

When the respiratory rate of a patient who is being mechanically ventilated is to be determined, a crucial decision concerns which ventilator "mode" is most appropriate. In the assist-control mode, the ventilator delivers a full preset tidal volume each time the patient initiates an inspiration. This mode is preferred for most conscious patients and should be used in patients with cardiogenic or other forms of shock. This method decreases the blood flow to the diaphragm and allows blood to be diverted to more critical organs. In addition, patients with diffuse lung disease and metabolic acidosis should be placed on assist-control to allow them to reduce their level of carbon dioxide to compensate for the acidosis. However, problems may arise with patients who "fight the ventilator" and with patients with hyperventilation. The former situation arises when the patient perceives that the ventilator settings as determined by the physician are inadequate and breathes out of synchronization with the ventilator. This problem can be corrected by

changing the settings, by sedating the patient, or by switching to another mode. Hyperventilation results when the patient's drive to breathe is not diminished by correction of hypoxemia or the provision of large tidal volumes, as often occurs with diffuse pulmonary processes and in patients with altered sensorium and increased central drive. This condition can most readily be corrected by changing modes to intermittent mandatory ventilation (IMV).

IMV is a mode of ventilation in which the patient draws spontaneous tidal volumes from a reservoir and receives intermittent breaths from the ventilator at mandatory intervals. Thus, IMV combines assisted and unassisted ventilation. IMV should always be used in patients who are intubated for chronic carbon dioxide retention. Rapid reduction of carbon dioxide leads to CNS alkalosis and results in seizures. IMV may be useful in patients with central hyperventilation by avoiding the delivery of large tidal volumes each time the patient inspires. Also, IMV may be useful in weaning patients from mechanical ventilation because it provides a method for gradually reducing the amount of mechanical assistance to ventilation. IMV is obviously not well suited to the patient with respiratory muscle fatigue in whom rest is required; indeed, if the spontaneous circuit is not properly adjusted, the work of breathing with IMV may be substantially greater than with unassisted breathing.

One other newer mode is available on some ventilators, namely "pressure support". A preset inspiratory pressure is maintained throughout inspiration, whereas tidal volume depends on patient effort. This mode helps to overcome the systemic resistance during spontaneous ventilation, thereby decreasing the work of breathing. Pressure support may prove to be helpful in weaning patients from the ventilator.

To select the initial FiO<sub>2</sub> when instituting mechanical ventilation, there are two reasonable approaches. With many patients, previous blood gas determinations will be of help in knowing what oxygen concentrations were previously inadequate, and a