EMERGENT MANAGEMENT OF THE AIRWAY New Pharmacology and the Control of Comorbidities in Cardiac Disease, Ischemia, and Valvular Heart Disease

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Managing the Airway in the Critically Ill Patient

EMERGENT MANAGEMENT OF THE AIRWAY
New Pharmacology and the Control of Comorbidities in Cardiac Disease, Ischemia, and Valvular Heart Disease

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The process of securing the airway by endotracheal intubation is fraught with risk for the patient and requires decisions based on understanding the underlying pathophysiology of the patient in distress and the pharmacology of medications to be given. The severity of the patient's distress and the urgency of securing the airway determine the acceptance of the risk of incurring complications from urgent or emergent intubation. Whereas emergent intubation leaves little time for optimizing conditions, the less urgent and more elective intubation permits a more controlled clinical setting.

Intubations can be characterized into one of two scenarios. The emergent intubation of an airway often results from acute respiratory or cardiac arrest. This patient is often unconscious and hypotensive. The primary goal in this group of patients is to secure the airway as quickly and safely as possible. In such a clinical setting, there is often little need for or availability of sedatives, analgesics, and muscle relaxants. Immediate assurance of adequate ventilation and oxygenation will permit the clinicians to stabilize the patient and address the precipitating factors. The less urgent scenario is that of patients having progressive respiratory or cardiac decompensation. The additional time allows the health care team the luxury of formulating and implementing a safe plan for securing the airway. The elements of a safe plan for securing the airway include:

1. **Review of the patient's chart and clinical situation,**
2. **Assessment of the acuity of establishing endotracheal intubation,**
3. **Consideration of the factors affecting the patient's hemodynamics,**
4. **Assessment of the patient's present and anticipated postintubation volume status,**
5. **Formulation of a plan for induction of anesthesia, including muscle relaxant and sedative use, and**
6. **Confirmation of the availability of emergency drugs based on anticipated liability of the patient's hemodynamics.**

Patients requiring less urgent assistance are usually conscious but either agitated or somewhat obtunded. Respiratory insufficiency is often associated with hypoxia, hypercarbia from hypoventilation or ineffective ventilation, and fatigue from the prolonged period of tachypnea, stress, and lack of sleep. Affected patients demonstrate labile hemodynamics that reflect near-maximal sympathetic outflow. The labile nature of their heart rates and blood pressures also is compounded by relative hypovolemia that may have been induced to improve pulmonary function or resulted from abstinence from oral intake. To minimize hemodynamic perturbation, the clinician must carefully choose the appropriate medications and dosages for the induction of sedation based on the patient's volume status, cardiac function, and anticipation of postintubation hemodynamic changes.

RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION

The patient's protective upper airway reflexes are a potent stimulus during laryngoscopy and endotracheal intubation. The afferent sensory pathway is comprised of the glossopharyngeal nerve, which innervates the pharyngeal structures superior to the anterior surface of the epiglottis, and the vagus, which innervates the posterior epiglottis distally into the trachea. The efferent responses to laryngoscopy and endotracheal intubation are mediated by both the parasympathetic and sympathetic nervous systems. The parasympathetic response, which is mediated by the vagus, can produce sinus arrest or bradycardia. This reflex is more significant in children than in adults. The more dominant response in adults is sympathetic stimulation, which increases heart rate, blood pressure, and pulmonary capillary wedge pressure. [7]

The magnitude of the cardiovascular response reflects a number of factors, including a history of hypertension and valvular disease, volume status, and the extent of the airway manipulation. The hypertensive and tachycardic response to laryngoscopy starts within 5 sec and reaches a plateau after 45 to 60 sec of laryngoscopy and returns to prelaryngoscopy levels 5 min after the procedure. [35] Hemodynamic responses can be minimized by decreasing the duration and force of airway manipulation. [8] [35]

CARDIOVASCULAR EFFECT OF HYPOXEMIA AND HYPERCAPNIA

Most patients who require intubation experience significant hypoxia and hypercarbia. Hypoxemia activates the chemoreceptors in the carotid and aortic bodies, which in turn, activate the respiratory and vasomotor centers in the medulla. The cardiovascular response to peripheral chemoreceptor stimulation consists of peripheral vasoconstriction and bradycardia. In the case of severe hypoxemia (PaO_2 of < 30 mmHg), the sympathetic outflow also may be directly activated centrally. Hypercapnia, another potent stimulus, has a marked vasoconstrictive effect by direct stimulation of the vasomotor center. Profound hypercapnia and hypoxemia can result in acidosis that manifests as bradycardia, hypotension, and decreased cardiac output.

THE POSTINTUBATION PERIOD

As discussed previously, the physiology of hyperdynamic and labile hemodynamics that reflect near-maximal sympathetic outflow and hypovolemia often develop in patients who have respiratory distress. Abrupt abatement of sympathetic tone may lead to profound hemodynamic changes regardless of the technique of induction. Many of the medications that blunt the responses to laryngoscopy and intubation often cause hypotension by decreasing sympathetic outflow. Another consequence of securing the airway and initiating mechanical ventilation is the removal of the stresses of hypoxia and hypercarbia. The resulting decrease in sympathetic vascular tone may compromise hemodynamics by decreasing the cardiac parameters of preload and afterload. The clinical scenario is often further complicated because hypovolemia renders patient more sensitive to the effects of controlled ventilation. Positive pressure ventilation and positive end-expiratory pressure (PEEP) increase intrathoracic pressure, impeding venous return to the right ventricle and increasing right ventricular afterload. This problem may be further exacerbated in patients who have significant obstructive lung disease. [14] Consequently the combined effects from the loss of sympathetic tone after the induction of anesthesia and the decrease in preload from positive pressure ventilation can result in profound hypotension, often with reflex tachycardia.

Another population of patients who are volume sensitive and are at risk of hypotension are those who have aortic or mitral stenosis. In these patients, who have a marked dependence on ventricular preload, one might consider intravascular volume expansion before induction and intubation. This group of patients is discussed in detail later.

PHARMACOLOGY

The choice of pharmacologic agents that are used to induce anesthesia for intubation and to treat the potential perturbations in hemodynamics effectively requires an appreciation of the pharmacology of selected agents and the patient's coexisting pathology. Most induction agents are sedative hypnotics that provide minimal analgesia. Stable hemodynamics during the induction of sedation do not prevent the marked sympathetic discharge associated with laryngoscopy and tracheal intubation. Patients are, therefore, often given a combination of drugs to provide adequate sedation, analgesia, and muscle relaxation. The following section provides a brief review of induction agents, muscle relaxants, and other drugs that are administered to optimize cardiovascular hemodynamics.

Choice of Intravenous Induction Agents *(Table 1)*

TABLE 1 -- INDUCTION AGENTS

Agent	Dosage Range *
Thiopental	3.0-5.0 mg/kg
Methohexital	1.0-2.0 mg/kg
Etomidate	0.2-0.5 mg/kg
Midazolam	0.2-0.4 mg/kg
Diazepam	0.3-0.6 mg/kg
Propofol	1.5-2.0 mg/kg
Ketamine	1.0-2.0 mg/kg

*Dosages should be decreased in patients who are critically ill or who have hypovolemia.

Barbiturates are commonly used induction agents for hemodynamically stable patients. Thiopental, a short-acting barbiturate, and methohexital, a more potent and ultrashort-acting agent, have rapid and highly predictable hypnotic effects and good overall safety profile in hemodynamically stable patients in the operating room. Thiopental produces a dose-related direct negative inotropic effect, [25] a decrease of ventricle filling related to increased venous capacitance, [6] and a transient decrease of sympathetic outflow from the central nervous system. Patients with impaired ventricular function or with hypovolemia experience significant reductions in blood pressure and cardiac output. [24] The degree of cardiovascular depression is equal when these drugs are given in equipotent doses to patients with cardiovascular disease. [20] Barbiturates are not analgesics, and dosages that do not compromise hemodynamics are ineffective in preventing transient hypertension and tachycardia related to tracheal intubation. Several studies have demonstrated attenuation of the sympathetic response by the additional administration of fentanyl. [3] If a barbiturate is administrated for induction, it should be titrated to achieve the appropriate level of sedation before administration of neuromuscular blockers. The clinician should be ready to respond to the immediate (hypertension and tachycardia), and the delayed (hypotension) responses after laryngoscopy. Use of these agents for patients having respiratory or cardiac distress is not recommended, because they require careful titration and their use may result in a high incidence of unacceptable hemodynamic consequences.

Benzodiazepines are sedative-hypnotic agents that have been used for induction of anesthesia because they lack direct cardiovascular effects. [25] The administration of benzodiazepines alone fails to attenuate sufficiently the rise of heart rate and blood pressure associated with laryngoscopy. [25] The combination of benzodiazepines and opiates used to blunt the sympathetic response produced more vasodilatation and hypotension than did either drug alone. [38] An alternative

strategy is to administer a benzodiazepine and ketamine, a sedative-hypnotic agent with analgesic properties. Midazolam, 0.15 mg/kg, and ketamine, 0.75 and 1.5 mg/kg, administered about 90 sec before laryngoscopy and endotracheal intubation in patients undergoing emergency surgery prevented hypotension but failed to attenuate fully the increase in heart rate and blood pressure after intubation. [41] Additionally, administration of benzodiazepines can cause undesirable cognitive and behavioral effects in certain elderly patients.

Etomidate, a carboxylated imidazole, is characterized by a rapid onset and short duration of action. When compared with most other induction agents, etomidate produces the least hemodynamic pertubation and cardiac depression. [17] In patients with compensated ischemic heart disease, administration of 0.15 to 0.30 mg/kg produced no significant hemodynamic changes. [37] In one study of patients with aortic or mitral valvular disease, induction using etomidate (0.3 mg/kg) was associated with a 19% decrease in systemic blood pressure and a slight decrease in cardiac output. [4] As with the barbiturates and benzodiazepines, etomidate does not produce analgesia. Tracheal intubation may be followed by significant increases in heart rate and blood pressure, leading to myocardial ischemia in patients with coronary artery disease. [2] The combination of etomidate, 0.4 mg/kg, and fentanyl, 3 to 4 mcg/kg, for induction has been shown to produce hemodynamic stability during laryngoscopy and endotracheal intubation in New York Heart Association Class III and IV patients. [34] Etomidate is often the induction agent of choice in the intensive care unit because of its minimal cardiovascular effects.

Ketamine, a phencyclidine derivative, produces rapid hypnosis and profound analgesia. It directly stimulates the central nervous system and increases sympathetic outflow, which increases heart rate, blood pressure, pulmonary artery pressures, and cardiac contractility [5] [39] ; however, in vitro ketamine produces direct cardiac depression. [40] The cardiac depressant effects of ketamine may be predominant in severely ill patients whose catecholamine stores are depleted by protracted periods of maximal sympathetic discharge. The variable effect on cardiovascular hemodynamics and a high incidence of emergence delirium limit the clinical usefulness of ketamine as a sole induction agent. Ketamine is often used in combination with other agents, (such as fentanyl or a benzodiazepine) to provide relatively stable conditions during the period of induction and postintubation.

Propofol is an induction agent that is commonly used in the intensive care environment for sedation. Propofol has the advantage of being a readily available, short-acting sedative that permits assessment of the patient's neural status soon after intubation. Enthusiasm for this induction agent in the unstable patient must be tempered by the risks of hypotension and tachycardia. Bolus administration is associated with pain when injected in a peripheral vein and with significant hypotension related to venodilation and myocardial depression. [33] The cardiac depressant effect of propofol may be more pronounced than with an equipotent dose of thiopental. [22] In patients with severe coronary artery disease, aortic stenosis, and hypertension, propofol may induce myocardial ischemia because of reduced coronary perfusion. In patients with mitral regurgitation or aortic insufficiency, propofol may decrease blood pressure but improve cardiac performance because of afterload reduction. Like most other induction agents, it has no analgesic properties, and the patients benefit from the addition of an analgesic agent (i.e., a narcotic). The use of propofol in unstable or hypovolemic patients requires careful titration and anticipation of changes in hemodynamics.

Muscle Relaxants

In the setting of an acute cardiac or respiratory arrest, patients are often unconscious and sufficiently relaxed that laryngoscopy and intubation of the trachea do not require further muscle relaxation. In elective or urgent intubations, however, the process of securing the airway by laryngoscopy is often facilitated by a neuromuscular relaxant. Considerations for the choice of drug are:

1. Acuity,
2. Assessment of the ability to intubate the trachea,
3. Metabolic integrity of liver and kidneys,
4. K+ concentration,
5. Duration of relaxation desired, and
6. Presence of confounding factors (denervation hypersensitivity, increased intracranial pressure).

The optimal drug would be one that quickly achieves maximal effect with a minimal incidence of adverse reactions.

Succinylcholine, the neuromuscular blocker that has the fastest onset, is the classic agent used for rapid sequence induction and intubation (Table 2) . Muscle blockage is produced by depolarization of the neuromuscular motor endplate. Termination of blockade is dependent on diffusion out of the motor end-plate with subsequent hydrolysis by pseudocholinesterase. Although it has the fastest onset, succinylcholine has fallen from use as the first-line agent because of its associated complications. Hyperkalemia resulting in cardiac arrest can occur after its administration to patients with extensive burns, trauma, and denervation hypersensitivity (usually limited to 5 to 60 days after central

nervous system injury). It also has been reported to cause sinus bradycardia and junctional and ventricular arrhythmias. Use of succinylcholine also is controversial in the head-injured patient because of possible increases in intracranial pressure. An absolute contraindication is a history of susceptibility to malignant hyperthermia. Because of these factors, most clinicians now choose an alternate agent.

TABLE 2 -- MUSCLE RELAXANTS

Agent	Bolus Dosage Range	Onset	Duration
Succinylcholine	1-1.5 mg/kg	0.5-1 min	4-8 min
Mivacurium	0.15-0.25 mg/kg	1.5-3 min	12-20 min
Cisatracurium	0.15-0.2 mg/kg	2-3 min	40-60 min
Atracurium	0.4-0.5 mg/kg	2-3 min	40-60 min
Rocuronium	0.6-1.2 mg/kg	1-1.5 min	40-60 min
Vecuronium	0.8-0.1 mg/kg	2-3 min	30-40 min
Pancuronium	0.08-0.1 mg/kg	3-5 min	60-100 min

The short-acting (mivacurium, rapacuronium) and the intermediate-acting (cisatracurium, vecuronium, rocuronium, atracurium) nondepolarizing muscle relaxants are more commonly used because of their relative lack of side effects and contraindications as compared with succinylcholine. The times of onset and dosage guidelines for nondepolarizing muscle relaxants are listed in Table 2 . Some of the muscle relaxants may adversely affect hemodynamics. Atracurium and mivacurium may produce a dose-dependent decrease in blood pressure related to histamine release, an effect minimized by slow (75 to 90 sec) administration. [26] [28] Rocuronium may produce tachycardia by vagal inhibition. [21] Cisatracurium is a common choice for emergent intubations because of its relatively rapid onset, hemodynamic stability, and dependable pharmacodynamics. [18] [27] Both cisatracurium and atracurium depend on hydrolysis in the plasma by Hoffman elimination and so are independent of renal and hepatic function.

Additional Strategies to Modulate Hemodynamic Perturbations from Laryngoscopy

A wide variety of drugs, techniques, and routes of administration are used to counteract hypertensive and tachycardic response to laryngoscopy and endotracheal intubation.

Local Anesthetics

Lidocaine, an amide local anesthetic, has been used as a topical anesthetic for the oropharynx and as an intravenous agent to blunt sympathetic stimulation. Lidocaine has a good safety profile, and adverse effects are dose-related. Increased plasma levels of lidocaine can cause myocardial depression, vascular dilatation, and inhibition of neural activity causing sedation and seizures. The beneficial effects of intravenous lidocaine are inconsistent and therefore its use in these settings is controversial. [12] [30] [36] Usual doses are 0.5 to 1.0 mg/kg given intravenously 2 to 4 min before laryngoscopy.

Narcotic Analgesics ***(Table 3)***

TABLE 3 -- ANALGESICS

Agent	Dose Range *
Fentanyl	1.5-8 mcg/kg
Sufentanil	0.5-1.0 mcg/kg
Alfentanil	10-30 mcg/kg
Remifentanil	1.0 mcg/kg

*Dosages should be decreased in patients who are critically ill or have hypovolemia.

Narcotics are commonly used as adjuvant therapy during induction to attenuate sympathetic stimulation. To reduce the circulatory responses to intubation, narcotics can be administered before laryngoscopy; the timing of administration ranges from 3 to 5 min for fentanyl to about 1 min for alfentanil and remifentanil. [15] [23] They are routinely used intraoperatively during induction in patients who have a history of severe cardiac disease, because they do not directly depress cardiovascular function. Most of the hemodynamic effects of opioids (fentanyl, sufentanil, alfentanil, and remifentanil) can be related to their attenuation on sympathetic outflow and stimulation of central vagal response (bradycardia). The most commonly used drug is fentanyl because of its routine availability in the intensive care environment, wide therapeutic window, and familiarity of practitioners with its pharmacodynamics.

Fentanyl and sufentanil are narcotic anesthetic agents that have been recommended to attenuate the hypertensive response to laryngoscopy and endotracheal intubation in stable patients. [9] [10] Although sufentanil is fivefold more potent than fentanyl, its use is limited by availability and lack of familiarity by most clinicians. In elderly patients (age >65 years), the combination of fentanyl (1.5 and 3 mcg/kg) and thiopental (2.5 mg/kg) attenuated the increase in blood pressure and heart rate from intubation. [32] A 3 mcg/kg dose of fentanyl provided more stable hemodynamics immediately after intubation than 1.5 mcg/kg, but this larger dose was associated with a higher incidence of systolic hypotension. [32] The dosage of fentanyl should be based on the volume status and the severity of the underlying cardiovascular disease.

Alfentanil and remifentanil are potent narcotic analgesics that have the advantages of a short onset of action and a short duration of effect. The use of these agents as adjuncts or as the sole induction agents in unstable patients has not been studied. The following dosing recommendations are for hemodynamically stable patients. Alfentanil (10 to 30 mcg/kg) and remifentanil (1 mcg/k) administered a minimum of 1.5 min before manipulation of the airway in hemodynamically stable patients successfully attenuated the hypertensive and tachycardic responses to intubation. [1] [13] [23] The use of these drugs is limited by the absence of data in unstable patients, routine availability in the intensive care setting, and the incidence of hypotension during the postintubation period.

beta-Antagonists

Esmolol is an ultrashort-acting beta1-blocker with a half-life of 8 min because of rapid metabolism by esterases in the blood. The primary cardiac effect is as a negative chronotropic agent to control heart rate with minimal negative inotropic effect. The negative inotropic effect is more significant in patients with moderate to severe ventricular dysfunction. Use of this beta antagonist also may be contraindicated in patients who already require infusions of vasoactive agents to support the blood pressure or have reactive airway disease. Although it is a relatively specific beta-1 antagonist, the risk of bronchospasm is increased in patients who have reactive airway disease or who are receiving bronchodilator therapy. Administration of esmolol (1 to 2 mcg/kg) about 2 min before laryngoscopy has been used successfully in combination with narcotics to attenuate the increase in heart rate and blood pressure in hemodynamically stable patients. [16] The rationale for administration of esmolol to control heart rate in patients who require urgent or elective intubation should be on the basis of such risk factors as coronary artery disease, hypertension, aortic stenosis, and history of arrhythmia.

Vasodilators (Table 4)

TABLE 4 -- VASODILATORS

Agent	**Dose Range** *
Nicardipine	15-30 mcg/kg
Diltiazem	200-300 mcg/kg
Verapamil	50-100 mcg/kg
Nitroglycerine	1.5-2.5 mcg/kg
Nitroprusside	1-2 mcg/kg

*Dosages should be decreased in patients who are critically ill or who have hypovolemia.

Vasodilators are used to attenuate the hypertensive response to laryngoscopy and intubation. The choice of drug is related to availability, familiarity with single bolus administration, effect on contractility, and preferential effect on venous as opposed to arterial vessels. These drugs lack analgesic properties and, therefore, do not blunt the sympathetic response to laryngoscopy. The strategy of preemptive vasodilator therapy should be reserved for patients who require tight control of blood pressure or ventricular afterload, for example, individual with severe aortic insufficiency or aortic dissection.

Calcium channel antagonists block L-type channels, which are located in cardiac and smooth muscles. They have an element of negative inotropy (verapamil > diltiazem > nicardipine), vasodilation (nicardipine > diltiazem > verapamil), and negative chronotropy (verapamil = diltiazem > nicardipine). Because Ca^{++} channel blockers affect cardiac conduction and prolong refractory nodal tissue, their use with beta-antagonists can lead to atrioventricular block and severe myocardial depression. These agents are generally not administered as adjuncts for intubation because of their incidence of negative cardiac effects and lack of immediate availability for bolus injection.

Nitroglycerine and nitroprusside are vasodilators that have rapid onset and short duration of action. At small doses, nitroglycerine decreases ventricular preload because of prominent venodilation in the splanchnic capacitance vessels. At higher doses, nitroglycerine dilates smaller arterioles and resistance vessels, thus reducing afterload and blood pressure. Nitroprusside is a potent arterial and venous dilator. Patients who require emergent intubation often have hypovolemia with increased sympathetic tone, so the magnitude of the vasodilator response can be exaggerated and produce profound hypotension.

COEXISTING CARDIOVASCULAR PATHOLOGY

The following sections are a brief review of potentially lethal conditions that would markedly complicate the choice of pharmacologic agents and strategies to treat hemodynamic lability during and after intubation (Tables 5 and 6) .

TABLE 5 -- MANAGEMENT STRATEGIES TO TREAT HYPOTENSION

Causes of Hypotension	Strategies
Aortic stenosis	Administer vasopressor (i.e., phenylephrine), volume administration, restore sinus rhythm
Aortic insufficiency	Maintain slightly elevated heart rate, administer positive inotropic agent, and check volume status (consider volume administration)
Mitral stenosis	Administer positive inotropic agent (i.e., epinephrine, dobutamine)
Mitral insufficiency	Maintain slightly elevated heart rate, administer positive inotropic agent, and check volume status
Sepsis	Vasopressor and volume administration
Cardiac ischemia	Slow heart rate, vasopressor for coronary perfusion, intraaortic balloon counterpulsation
Cardiogenic shock	Vasopressor (i.e., phenylephrine), inotropic agent (i.e., epinephrine), intraaortic balloon counterpulsation
Bradycardia	Atropine, positive chronotrope (i.e., epinephrine, isoproterenol), transvenous or transcutaneous pacing

TABLE 6 -- THE HEMODYNAMIC GOALS FOR VALVULAR LESIONS *

Valvular Lesion	Inotropy	Heart Rate (bpm)	Preload	Afterload
Aortic stenosis, hypertension	↔	60-70	↑	↔ ↑
Aortic regurgitation	↔ ↑	80-90	↔	↔ ↓
Mitral stenosis	↔	60-70	↑	↔
Mitral regurgitation	↔ ↑	80-90	↔	↔ ↓

↑, increase; ↓, decrease; ↔, no change.

*No studies have been performed in unstable, respiratory compromised patients.

Myocardial Ischemia

Myocardial ischemia may be either the precipitating cause of respiratory distress, the result of hypoxemia, or the result of laryngoscopy for endotracheal intubation. Most elderly patients, regardless of a documented history of coronary disease, should be considered at risk for myocardial ischemia. Simply stated, myocardial ischemia results from the imbalance between myocardial oxygen demand and supply. The general strategy is to maximize the delivery of oxygen while minimizing metabolic demand.

Under normal conditions coronary blood flow is determined by perfusion pressure and coronary arterial resistance. The primary mechanism by which coronary flow adjusts to metabolic demand is by the autoregulation of blood flow, which can increase coronary flow by a factor of 4 to 6. Significant coronary arteriosclerosis exhausts this reserve capacity to increase flow and causes the coronary blood supply to be solely dependent on the pressure gradient (usually diastolic blood pressure, left ventricular end-diastolic pressure). Discrete coronary lesions are not the only pathology that can jeopardize coronary blood flow. Myocardial hypertrophy associated with aortic stenosis and hypertension not only increases demand, but also increases compressive resistance during systole to impair perfusion of subendocardium.

The major determinants of myocardial oxygen demand are heart rate, afterload, and contractility. Tachycardia (heart rate > 90 beats per min) has a greater impact on occurrence of myocardial ischemia than hypertension or transient hypotension. [19] [31] The management strategies to address the risk of myocardial ischemia are to minimize myocardial oxygen consumption by preventing the increase in heart rate and avoiding an extreme increase in wall stress (increased inotropy and afterload) while maintaining coronary perfusion. These goals are best addressed by use of an appropriate combination of adjunct therapies and induction agents. The administration of a narcotic and a small dose of esmolol and the induction agent should prevent tachycardia, the major determinant of oxygen use.

Left Ventricular Hypertrophy: Aortic Stenosis

and Hypertension

The intracavitary systolic pressure that is generated to overcome resistance to left ventricular ejection increases myocardial wall tension (Laplace's law) and leads to myocardial hypertrophy. In the case of aortic stenosis, the increase in afterload occurs at the level of the aortic valve. In the case of hypertension, the increase in afterload occurs at the level of the system circulation. Although the severity of aortic stenosis may not be considered clinically important until the valve area is less than 1.2 cm^2, the compensatory effect of ventricular hypertrophy renders patients with aortic stenosis at risk for complications. [11]

The physiologic consequences of left ventricular hypertrophy include alterations in diastolic compliance and imbalances in the relationship of myocardial oxygen supply and demand. In addition, epicardial vessels and capillary density do not

increase proportionally to compensate for the hypertrophy. The decreased compliance of the left ventricle increases the importance of the atrial systole to ventricular filling. The increased wall stress and left ventricle hypertrophy render the subendocardium at risk of myocardial ischemia, even in the absence of discrete coronary stenosis.

Hemodynamic management of patients with hypertrophic left ventricle is based on the avoidance of systemic hypotension, maintenance of an adequate intravascular volume, prevention of atrial dysrhythmia, and awareness of the potential for myocardial ischemia. Hypotension, regardless of primary cause, should be treated immediately to restore coronary perfusion pressure by administration of an alpha-adrenergic agonist (for example, phenylephrine) and assurance of adequate preload and the presence of sinus rhythm. If clinical assessment supports a diagnosis of hypovolemia, one might consider fluid administration before the procedure to avoid postintubation hypotension and compensatory tachycardia. Because a higher mean left atrial pressure is necessary to distend a hypertrophic left ventricle, the loss of sinus rhythm may, in part, be compensated by volume infusion. The best therapy, however, is restoration of sinus rhythm. To maintain sinus rhythm at a controlled rate, pretreatment with esmolol may reduce the risk of tachycardia and supraventricular arrhythmia that would compromise diastolic filling.

Aortic Regurgitation

Congestive heart failure and pulmonary edema are clinical manifestations of severe aortic insufficiency (AI) that may precipitate respiratory embarrassment requiring intubation. Patients who have chronic AI are generally better compensated than those with acute AI (i.e., Type A aortic dissection). As compensation for AI, the left ventricle increases size, systolic wall tension, muscle mass, and heart rate, all of which contribute to an increase in oxygen demand. Another complicating factor is that patients with AI often have coincident aortic stenosis. Strategies to manage hemodynamic perturbations depend on the dominant lesion (aortic stenosis versus insufficiency) and status of ventricular function.

The key goals of management of patients with aortic regurgitation are to maintain ventricular preload, preserve low afterload, keep the heart rate slightly elevated, and support contractility. Patients with aortic insufficiency differ widely in the severity of concomitant ventricular dysfunction; the management strategies must be individualized. The choice and dose of induction agent should focus on the severity of myocardial depression. Patients with severe ventricular dysfunction should receive drugs that have minimal cardiac depression (such as etomidate and narcotics); a decrease in sympathetic vascular tone will decrease afterload and improve forward flow. Hypertension should be treated with short-acting vasodilators (such as sodium nitroprusside, nitroglycerine, and hydralazine) or narcotic analgesics; drugs causing significant cardiac depression (such as esmolol, verapamil) should be avoided. Postintubation hypotension may require administration of intravenous fluids to maintain ventricular preload or a sympathomimetic to improve contractility (i.e., dobutamine, epinephrine, and dopamine).

Mitral Stenosis

Mitral stenosis is characterized by a significant pressure gradient between the left atrium and ventricle that results in an underfilled left ventricle. Any intervention that increases cardiac output (i.e., sympathetic stimulation or volume loading) or decreases diastolic time (i.e., tachycardia) will increase the impedance of flow across the stenotic mitral valve. The impedance to blood flow at the level of the mitral valve causes an increase in left atrial pressure, which is associated with atrial fibrillation, pulmonary hypertension, and right ventricular dysfunction.

Patients with mitral stenosis can be some of the most challenging to manage, because they are sensitive to changes in volume, inotropic state, and $PaCO_2$. Less-than-optimal management can precipitate an acute right ventricular failure and pulmonary edema. In the setting of urgent intubation, sedation should be titrated carefully to prevent further anxiety while the clinician begins to control respiration. Taking control of ventilation will reverse hypercapnia, allow for hyperventilation to decrease pulmonary artery pressures, and assure adequate oxygenation. One should avoid agents that produce marked afterload reduction of the left ventricle, because vasodilatation aggravates the diastolic underfilling of the left ventricle, an unfavorable location on the Starling pressure-volume curve. Some of the chief hemodynamic goals in the management of patients with mitral stenosis are to prevent any increases in pulmonary artery pressure, avoid tachycardia, and select drugs that cause minimal cardiac depression. Appropriate dosing with analgesics and beta-blockers (i.e., esmolol) may be particularly useful in controlling the heart rate. Systemic hypotension raises concerns about the adequacy of both right and left coronary perfusion. alpha-Adrenergic agonists should be used with caution, because acute drug-induced increases in pulmonary vascular resistance can precipitate right ventricular failure. Often the choice of a positive inotropic agent is a more suitable strategy to increase the blood pressure.

Mitral Regurgitation

Most patients with chronic mitral regurgitation experience few symptoms until late in the course of their disease,

whereas patients with acute, severe mitral regurgitation (i.e., ruptured chordae or papillary muscles) often manifest symptoms of acute pulmonary edema, cardiovascular collapse, or both. The main goals of hemodynamic management in patients who have mitral regurgitation are to decrease afterload, maintain a slightly elevated heart rate, maintain preload, and support impaired contractility. Patients under stressful situations maintain high sympathetic tone with elevated heart rates. This relative tachycardia promotes forward flow by decreasing the time of systole, and therefore the time of retrograde flow into the atrium.

The pathophysiology of mitral regurgitation provides the left ventricle with optimal conditions for a high ejection fraction. There is abundant preload and minimal afterload, making it difficult to estimate the severity of ventricular dysfunction. Only a fraction of ventricular ejection is antegrade; a low or even low-normal ejection fraction should be a warning of depressed ventricular contractility. Induction can be performed safely by the titration of a low-dose narcotic and etomidate. The reader is cautioned about giving drugs (such as verapamil, barbiturates, and nonspecific beta-blockers) that have significant negative inotropic effect. Cardiac performance can be increased by administration of positive inotropic drugs (i.e., dobutamine) to enhance contractility and vasodilators to promote forward flow, thus reducing the regurgitant volume.

SUMMARY

Once it is decided that the patient in distress requires tracheal intubation, the primary goal is to secure the airway as quickly and safely as possible to assure adequate oxygenation and ventilation. The clinician should quickly review the patient's history, physical examination findings, and laboratory data to determine the presence of cardiovascular disease, assess intravascular volume status, and formulate a plan for induction of anesthesia. The stresses of hypoxia, hypercarbia, acidosis, and extreme fatigue result in near-maximal sympathetic outflow that is manifest as tachycardia, labile blood pressure, and increased myocardial contractility. The astute clinician should anticipate that the tachycardia and hypertension associated with laryngoscopy and tracheal intubation is followed by a period of hypotension. This postintubation hypotension results from the acute marked attenuation of the sympathetic tone associated with resolution of hypoxia and hypercarbia, direct drug-induced negative inotropic effect, and vasodilation. The decrease in sympathetic vascular tone may result in hypotension by exacerbating the decrease in cardiac preload and afterload from hypovolemia. In addition, the use of positive pressure ventilation and positive end-expiratory pressure (PEEP) in these hypovolemic patients will further decrease ventricular preload by impeding venous return, leading to profound hypotension.

Several pharmacologic agents are required to treat effectively the hemodynamic perturbations associated with induction, laryngoscopy, and tracheal intubation. Most sedative hypnotic agents that are administered for induction provide minimal to no analgesia. Patients are most often given a combination of drugs to provide adequate sedation, analgesia to blunt the noxious stimuli, and muscle relaxation to facilitate the laryngoscopy. The major challenge is to choose a combination of drugs that at the appropriate doses, effectively blunt the responses to intubation without contributing to postlaryngoscopy hypotension. One can use several strategies to accomplish these goals; administration of a narcotic analgesic before induction decreases the dose of induction agent and can attenuate the sympathetic response to intubation. Because of the prevalence of cardiovascular disease and hypovolemia in this population of patients, all chosen drugs should have minimal negative effect on cardiac function and patients with hypovolemia should be hydrated. Most clinical studies have been performed in hemodynamically stable patients, so the routine dosages of sedative hypnotics should be reduced substantially and titrated to effect. An additional strategy is to treat significant hemodynamic perturbations with vasopressors, vasodilators, short-acting selective beta-1 blockers, and inotropic agents. The choice of vasoactive agent depends on the magnitude of the hemodynamic response and the presence of specific underlying cardiovascular pathology.

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