Beta adrenergic blockers have been one of the most prescribed category of drugs during the past 15 years. Recently, many new beta blockers have been introduced because they have unique pharmacokinetic and pharmacodynamic properties that benefit patients. The purpose of this article is to review the pharmacological properties and clinical uses of beta blockers. The latter part of this review will discuss the anesthesia implications of this group of drugs.

Since the introduction of propranolol in the early 1970s, beta blockers have become one of the most prescribed drugs in medicine. Propranolol was the only beta blocker available in the U.S. for several years and continues to be popular among many clinicians, even though many additional beta blockers have been introduced into clinical practice since 1980. Each of these more recent drugs offers certain pharmacokinetic and/or pharmacodynamic advantages over propranolol.

In this discussion, the current pharmacology and clinical use of beta blockers will be reviewed. The latter part of the review will deal specifically with the anesthetic implications of these drugs.

Both endogenous catecholamines—epinephrine and norepinephrine—react with beta receptors in the body. The beta receptors that these substance react with can be classified into two groups, beta 1 and beta 2. Table I lists the functions that these receptors perform within the body.

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**Table I**

<table>
<thead>
<tr>
<th>Functions of beta receptors within the body</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta—1</td>
</tr>
<tr>
<td>Increases heart rate</td>
</tr>
<tr>
<td>Increases myocardial contractility</td>
</tr>
<tr>
<td>Increases conduction velocity</td>
</tr>
<tr>
<td>Shortens refractory period at the atrioventricular (AV) node</td>
</tr>
</tbody>
</table>

| Beta—2                                      |
| Dilates blood vessels in skeletal muscle   |
| Dilates bronchiole smooth muscle           |
| Facilitates lipolysis                      |
| Increases glycogenolysis (cardiac and skeletal muscle) |

All of the beta-adrenergic functions in Table I involve postsynaptic effects; however, B-receptors recently have been identified on the presynaptic sympathetic nerve terminal. Stimulation of these receptors facilitates the release of norepinephrine from the postganglionic adrenergic nerve terminal.

Studies have indicated that the population of beta receptors in the body is not constant but varies according to certain controlling influences. It appears that one of the major controlling factors is the magnitude of stimulation of the receptors by sympathetic agonist. That is, when sympathetic activity is high, the density of receptors is low. Conversely, when sympathetic activity is low, the density of receptors is high. This receptor variability will
modulate sympathetic activity. Table II describes some factors that alter the density of sympathetic B-adrenergic receptors.

These findings concerning receptor density may explain certain phenomena observed with this group of drugs, such as:

1. Beta blockers are much more effective in treating young hypertensives than those of increased age.\(^{13}\)
2. Beta blocker “withdrawal” problems can develop secondary to receptor abundance in patients with ischemic heart disease who suddenly have their beta-blocker discontinued.\(^{5}\)
3. Treatment with beta blockers has been shown to be of benefit in patients with acute alcohol and narcotic withdrawal.

**Important pharmacodynamic properties**

All beta blockers are effective in inhibiting catecholamines at the beta-adrenergic receptors; however, the potency of their pharmacological effects varies. The differences in potency are not important pharmacologically, except when considering the proper dose of the drugs needed to produce a specified effect. When considering potency, propranolol is the standard of comparison for all other beta blockers.

Beta-adrenergic blockers can be classified according to beta receptor's selectivity. Most beta receptor antagonists block B\(_1\) and B\(_2\) receptors equally. These drugs are known as non-selective blockers. On the other hand, some beta blockers in lower doses will inhibit B\(_1\) receptors to a greater extent than B\(_2\) receptors. As the dosage of these selective blockers is increased, most of the selectivity is lost.\(^{14}\)

It has been postulated that leaving B\(_2\) receptors unblocked may be beneficial in patients with asthma, hypoglycemia, hypertension or peripheral vascular disease.\(^{15}\)

It also has been found that certain beta blockers have intrinsic sympathetic activity (ISA). When they react with beta receptors, these drugs will cause partial beta receptor activation. This occurs in ad-

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**Table II**

<table>
<thead>
<tr>
<th>Factors that alter sympathetic B-adrenergic binding</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Beta-adrenergic agonist(^4)</td>
</tr>
<tr>
<td>2. Propranolol (antagonist)(^6)</td>
</tr>
<tr>
<td>3. Pindolol (partial agonist)(^6)</td>
</tr>
<tr>
<td>4. Denervation(^7)</td>
</tr>
<tr>
<td>5. Hyperthyroidism(^6)</td>
</tr>
<tr>
<td>6. Hypothyroidism(^9)</td>
</tr>
<tr>
<td>7. Cortisone(^10)</td>
</tr>
<tr>
<td>8. Alcohol withdrawal(^11)</td>
</tr>
<tr>
<td>9. Aging(^12)</td>
</tr>
</tbody>
</table>

---

**Figure 1**

Chemical structure of common beta blockers

- **Atenolol**
- **Labetalol**
- **Nadolol**
- **Propranolol**
- **Esmolol**
- **Metoprolol**
- **Pindolol**
- **Timolol**
dition to preventing access of the natural or synthetic catecholamines to the receptor. The partial agonist effect of these drugs on beta receptors differs from such agonists as epinephrine or norepinephrine in that their maximal pharmacological effect is less, although their affinity for the receptor is high. Figure 1 depicts the chemical structure of common beta blockers.

Table III demonstrates the relative potency, selectivity and intrinsic sympathetic activity of beta blockers that are used clinically.

<table>
<thead>
<tr>
<th>Drug</th>
<th>B-blockade potency ratio (Propranolol = 1.0)</th>
<th>Relative beta selectivity</th>
<th>ISA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acebutolol</td>
<td>0.3</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Atenolol</td>
<td>1.0</td>
<td>++</td>
<td>0</td>
</tr>
<tr>
<td>Labetalol</td>
<td>0.3</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>1.0</td>
<td>++</td>
<td>0</td>
</tr>
<tr>
<td>Nadolol</td>
<td>1.0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pindolol</td>
<td>6.0</td>
<td>0</td>
<td>++</td>
</tr>
<tr>
<td>Propranolol</td>
<td>1.0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Timolol</td>
<td>6.0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

(Modified from Frishman, WH., Clinical Pharmacology of the B-Adrenoceptor Blocking Drugs, 2nd ed., Norwalk, Conn., Appleton-Century-Crofts, 1984, by permission.)

Before concluding discussion of the pharmacodynamic properties of the beta blockers, it must be pointed out that one drug, labetalol, blocks both alpha and beta receptors. This unique property has created much interest in anesthesia, and this drug will be considered in more detail later.

**Pharmacokinetics**

The pharmacokinetic properties that are important with all groups of drugs are gastrointestinal absorption, first-pass hepatic metabolism, lipid solubility, protein binding, volume of distribution, rate of metabolism, activity of metabolites and renal clearance of the drug. The beta blockers exhibit wide variability in these properties.

From a pharmacokinetic standpoint, beta blockers can be divided into two major groups: those metabolized by the liver and those excreted unchanged by the kidneys. The drugs metabolized by the liver tend to have shorter plasma half-lives, greater lipid solubility and highly variable bioavailability. Conversely, drugs that are eliminated unchanged by the kidneys tend to have greater water solubility, longer plasma half-lives and less variable bioavailability in patients with normal renal function. The longer half-life of these drugs allows for better patient compliance, since they can be taken once a day.

Table IV demonstrates the relationship between the route of elimination and the plasma half-life. It should be noted that timolol and acebutolol are exceptions to the previously stated pharmacokinetic concept. These drugs are not lipid soluble and primarily are excreted unchanged by the kidneys. However, they have a relatively short plasma half-life.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Elimination half-life (HR)</th>
<th>Lipid solubility</th>
<th>Route of elimination</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Esmolol</td>
<td>9 min.</td>
<td>High</td>
<td>Hepatic</td>
</tr>
<tr>
<td>2. Labetalol</td>
<td>3 - 4</td>
<td>Moderate</td>
<td>Hepatic</td>
</tr>
<tr>
<td>3. Metoprolol</td>
<td>3 - 4</td>
<td>Moderate</td>
<td>Hepatic</td>
</tr>
<tr>
<td>4. Pindolol</td>
<td>3 - 4</td>
<td>Moderate</td>
<td>Renal-Hepatic</td>
</tr>
<tr>
<td>5. Propranolol</td>
<td>3 - 4</td>
<td>Low</td>
<td>Renal-Hepatic</td>
</tr>
<tr>
<td>6. Timolol</td>
<td>4 - 5</td>
<td>Low</td>
<td>Renal-Hepatic</td>
</tr>
<tr>
<td>7. Acebutolol</td>
<td>3 - 4</td>
<td>Low</td>
<td>Renal</td>
</tr>
<tr>
<td>8. Atenolol</td>
<td>6 - 9</td>
<td>Low</td>
<td>Renal</td>
</tr>
<tr>
<td>9. Nadolol</td>
<td>14 - 24</td>
<td>Low</td>
<td>Renal</td>
</tr>
<tr>
<td>10. Sotalol</td>
<td>8 - 10</td>
<td>Low</td>
<td>Renal</td>
</tr>
</tbody>
</table>

(Modified from Frishman, WH., Clinical Pharmacology of the B-Adrenoceptor Blocking Drugs, 2nd ed., Norwalk, Conn., Appleton-Century-Crofts, 1984, by permission.)

Another important point is that the plasma half life and clinical duration of action in some cases do not correlate. Therefore, beta blockers with short half lives sometimes may only need to be taken once or twice daily. Timolol and acebutolol are again examples of this pharmacokinetic paradox.

Lipid solubility of beta blockers not only influences the route of elimination, but also the drug distribution in the body. This is especially important in determining which drugs go into the central nervous system (CNS).

Beta blockers that are lipid soluble can pass readily across the blood-brain barrier and cause CNS symptoms such as mental depression, lethargy and hallucinations. Drugs that are not lipid soluble, such as acebutolol, timolol, atenolol and nadolol, should have the advantage of not producing adverse CNS effects.
Therapeutic uses of beta blockers

One of the main indications for beta blockers is cardiovascular (CV) disease. All beta blockers appear to have a similar spectrum in treating disorders of the cardiovascular system. That is, if one specific beta blocker does not treat a CV disorder effectively, neither will another. Also, combining one beta blocker with another does not appear to offer any improvement in therapeutic response.17

Hypertension is one of the primary uses for beta blockers. Beta blockers that have been used for this purpose are atenolol, metoprolol, nadolol, pindolol, labetalol and timolol.17 One important advantage of these drugs in treating essential hypertension is the absence of orthostatic hypotension.

Some of the postulated mechanisms by which beta blockers lower blood pressure include:

1. Beta blockers could lower blood pressure by blocking beta receptors in the myocardium and sinoatrial (SA) node. This would result in a reduction in myocardial contractility and heart rate. Both these effects can lower blood pressure by reducing cardiac output.18

2. Non-selective beta blockers could reduce the blood pressure by inhibiting B$_2$-mediated renin release from the renal juxtaglomerular apparatus. This may be true especially in hypertensive patients with a high plasma renin concentration.19

3. Some beta blockers may lower blood pressure by lowering peripheral vascular resistance. This is possible with pindolol, which has partial agonist activity on B$_2$ receptors, and labetalol, which has alpha receptor blocking activity.17

Another important use of beta-adrenergic blockers is in angina pectoris. Certainly, these drugs provide prophylaxis against angina attacks, especially in patients who have a history of chronic stable angina. While beta blockers do not totally relieve the pain of myocardial ischemia, they do reduce the frequency of attacks and improve the patient's exercise tolerance.17

Beta blockers treat angina by reducing myocardial oxygen consumption. They accomplish this by reducing heart rate, depressing myocardial contractility and lowering blood pressure.20 21 Mortality after acute myocardial infarction can be reduced by beta-adrenergic blockade.17 In fact, one of these drugs, metoprolol, has been approved for use in the postmyocardial infarction period in order to limit infarction size.

Beta antagonists have been used in the treatment of cardiac dysrhythmias. These drugs suppress ventricular extrasystoles by decreasing cardiac automaticity. This effect results from reduced beta sympathetic activity to the heart, which causes a depression of spontaneous phase 4 depolarization.22

In addition, beta-adrenergic blockade also reduces sympathetic activity to the SA and atrial ventricular (AV) node, causing bradycardia at the SA node and slowing conduction and increasing the refractory period in the AV node. These effects make these drugs beneficial in the treatment of sinus tachycardia and rapid ventricle response to atrial fibrillation and flutter.17 These drugs must be used with caution in patients with atrial fibrillation who need high levels of sympathetic tone to avoid myocardial failure.

Finally, beta blockers have been used to prevent excess sympathetic nervous system activity. High sympathetic tone can be deleterious in various types of cardiovascular abnormalities, such as hypertrophic obstructive cardiomyopathies,23 mitral prolapse24 and tetralogy of Fallot.25 In these conditions, reducing sympathetic tone can attenuate associated cardiovascular symptoms. Tachycardia and cardiac dysrhythmias caused by sympathetic dominance in pheochromocytoma and hyperthyroidism also can be successfully treated with beta blockers.17

Beta-adrenergic blockers have been used in treating certain non-cardiovascular problems, such as migraine headaches and glaucoma. Documented effectiveness of such treatment has been limited to one drug with each condition. Propranolol has been used for migraine prophylaxis, and timolol has been used in the treatment of glaucoma.17

Perioperative uses of beta blockers

Since both inhaled anesthetics and beta blockers depress myocardial function, an additive effect would be expected to occur. However, clinical experience and controlled studies have revealed that this additive effect is not excessive and does not preclude the cautious simultaneous use of both classes of drug.26 A possible exception to this is topical ophthalmic timolol, which can cause profound bradycardia in the presence of inhaled anesthetic agents.27

It appears that the additive effect between beta blockers and inhaled anesthetics varies according to the anesthetic used. For example, beta antagonist and enflurane appear to have the greatest effect on cardiovascular parameters. Halothane has been shown to have intermediate effects, while isoflurane has been shown to have the fewest cardiovascular effects when administered with a beta blocker.28 29 This sequence is not surprising, because it follows the myocardial depression properties of the inhaled anesthetics.

Beta blockers also could interact with intravenous agents used in anesthesia. It has been demon-
strated, however, that preexisting beta blockade does not alter the cardiovascular response to high doses of opioids, such as fentanyl. On the other hand, beta blockade can cause a reduction in blood pressure and cardiac output when given with an agent that increases sympathetic activity, such as ketamine. In this case, beta blockers unmask the negative inotropic effects of the drug.

**Interaction with anticholinesterase drugs**

Bradygirda can be a problem when anticholinesterase drugs are given to a patient with a significant degree of beta blockade. A patient who is beta-blocked may have a significant bradycardia prior to administering an anticholinesterase. A test dose of an anticholinergic drug (atropine 70µg/kg) should be given to determine if the patient responds with an increase in heart rate. If not, consideration should be given to not administering anticholinesterase. Severe bradycardia that develops secondary to beta blockade and cholinesterase inhibition should be treated with an isoproterenol drip; in some cases, a transvenous artificial pacemaker may be necessary.

**Withdrawal hypersensitivity**

Abrupt discontinuation of beta blockers can cause excess sympathetic activity, which usually begins in 24 to 48 hours. As stated previously, this is thought to be caused by an increase in the number of beta-adrenergic receptors that develop secondary to chronic therapy with beta blockers.

This excess in sympathetic activity can be disastrous in the perioperative period. Patients may develop rebound hypertension, tachycardia or other dysrhythmias. The hypertension and tachycardia that can result from this withdrawal can lead to myocardial ischemia and/or infarction in the patient with a history of coronary artery disease. Ischemia is most likely to occur when sympathetic tone is high, as is seen during endotracheal intubation and emergence from anesthesia.

In order to prevent sympathetic override, the patient on beta blockers should remain blocked during the perioperative period. There have been various protocols, including intravenous infusions, suggested to maintain beta blockade in the perioperative period. No matter what protocol is selected, the anesthetist must understand the pharmacodynamic and pharmacokinetic properties of the beta blocker or blockers used in the care of the patients.

**Interaction with vasodilators used for deliberate hypotension**

When vasodilators are administered for deliberate hypotension, the sympathetic nervous system is activated via baroreceptor mechanisms. It has been demonstrated that the plasma concentration of both norepinephrine and epinephrine is increased when sodium nitroprusside is used to lower the arterial pressure in healthy adults. This can result in significant resistance to sodium nitroprusside. However, Khambatta and coworkers demonstrated that prior treatment of patients with propranolol for one day prior to surgery significantly reduced plasma catecholamine levels. Also, the acute administration of intravenous propranolol in incremental doses was able to decrease heart rate and reduce the amount of sodium nitroprusside necessary to maintain hypotension.

The renin-angiotensin system also is activated when hypotension is induced with sodium nitroprusside. Activation of this system can play an important role in the resistance seen with this hypotensive agent. Studies have shown that prior treatment with propranolol for one day before surgery attenuates the renin-angiotensin response. This effect not only reduces the amount of sodium nitroprusside needed for hypotension, but also abolishes the rebound hypertension seen after the discontinuation of the hypotensive state.

Labetalol and esmolol are two newer and unique beta blockers used in the perioperative period.

**Labetalol**

Labetalol has both non-selective beta blocking and selective alpha blocking properties. Animal studies indicate that labetalol is about one-fourth as potent as propranolol in blocking beta receptors and about one-sixth as potent as phentolamine in blocking alpha receptors. It also is about four to eight times more potent in blocking beta receptors than alpha receptors.

Labetalol has been used in the perioperative period to attenuate the adrenergic response to noxious stimuli, to treat hypertension and to facilitate induced hypotension. It is well known that laryngoscopy and tracheal intubation is a very stressful procedure. Labetalol has been studied to see if it can lessen the stress response associated with this maneuver. Leslie and co-workers gave varying doses of labetalol to patients prior to tracheal intubation and found that this pretreatment reduced both the heart rate and hypertensive response to tracheal intubation in a dose-related manner. The doses of labetalol used in this study ranged from 0.25 mg/kg to 1.0 mg/kg (17.5 to 70 mg/70 kg).

Inada and co-workers also studied the hemodynamic responses to tracheal intubation following labetalol pretreatment. Their study differs from the
study by Leslie and co-workers, because smaller doses of 5 to 10 mg of labetalol were used. In this study, labetalol did not attenuate the arterial pressure response, but did lessen the heart rate response to intubation.

These studies indicate that labetalol is effective in attenuating both the heart rate and arterial pressure response to tracheal intubation; however, the arterial pressure effect is seen only when at least 0.25 mg/kg are administered. It must be pointed out that high doses of 0.5 mg/kg to 0.75 mg/kg may be associated with significant reductions in arterial pressure, especially following tracheal intubation. Therefore, the best technique probably would be to use smaller doses of labetalol (5–10 mg or 0.07 to 0.14 mg/kg) with appropriate doses of sedative/narcotics, which should allow for the safe and effective attenuation of the hemodynamic responses to tracheal intubation. It is hoped that this combination will be studied in the future.

Labetalol also can be used to treat perioperative hypertension, especially that seen in the recovery room. Studies have indicated that labetalol effectively treats patients with hypertensive crises and those who need rapid reductions in their elevated blood pressures. Labetalol causes a fall in blood pressure that is accompanied by a modest reduction in heart rate. This effect on the heart rate is especially beneficial in patients with a history of myocardial ischemia.

Wilson and co-workers attempted to determine the dosage of labetalol needed in order to treat moderate to severe hypertension. They first administered an intravenous bolus of 20 mg labetalol, which reduced the baseline mean blood pressure by 23/14 mmHg. This initial bolus was followed by incremental doses of 20–80 mg at 10-minute intervals until either a therapeutic goal was achieved or a total dosage of 300 mg was administered. The required blood pressure levels were achieved in 53 of 59 patients in this study. The six patients who did not achieve their therapeutic goal did have substantial reductions in blood pressure. The response rate for intravenous labetalol compares favorably with that observed with diazoxide.

Stone and a co-worker studied myocardial ischemia in 128 untreated hypertensive patients who were given a single oral dose of labetalol, atenolol or oxprenolol prior to anesthesia and surgery. When compared with the control group, the patients pretreated with the beta blockers had a much lower incidence of myocardial ischemia. All three beta blockers tested appeared to be equally protective in this study.

Over the last few years, labetalol has been used to supplement other hypotension agents in inducing deliberate hypotension. It has been administered with varying combinations of volatile anesthetics, vasodilators and narcotics-N₂O.

Cope and Crawford studied the use of labetalol with halothane in order to obtain a hypotensive state. They gave 1% halothane to patients and obtained hypotension by administering intravenous labetalol. In this study, the initial dose of labetalol was 20 mg. If the arterial systolic pressure had not decreased in five minutes, increments of 5–10 mg were given until the desired blood pressure was obtained or the heart rate fell below 60 beats per minute. In most cases, this regimen produced an arterial systolic pressure in the range of 60–80 mmHg. The results of this study were satisfactory, because hypotension was attained quickly, maintained without difficulty and reversed readily in all cases. Additionally, in this study the hypotensive effects of labetalol could be reversed significantly by discontinuing the halothane and administering atropine 0.6 mg.

Studies have shown that using labetalol with small to moderate concentrations of volatile anesthetics does not cause a significant reduction in stroke volume or cardiac output. The alpha-blocking properties of labetalol facilitate the hypotension state by reducing peripheral vascular resistance, instead of causing myocardial depression. At the University of Alabama at Birmingham, labetalol is used most often with isoflurane for inducing hypotension. The initial dose of labetalol is 5–10 mg, and the inspired concentration of isoflurane is adjusted in order to maintain an appropriate hypotensive state. With this technique, labetalol significantly reduces the amount of isoflurane that must be administered in order to obtain hypotension. It also attenuates the tachycardia often seen with the administration of isoflurane.

Labetalol has been used to produce hypotension for Harrington rod procedures in which evoked potentials were being monitored. In this situation, volatile anesthetics were avoided, and hypotension was obtained with labetalol and sodium nitroprusside during narcotic-N₂O anesthesia.

With this technique, labetalol significantly reduced the amount of sodium nitroprusside that was needed to lower the arterial pressure. Labetalol also seemed to attenuate the wide fluctuations in arterial pressure that are often seen with sodium nitroprusside administration. With this regimen, the dose of labetalol that was needed to supplement sodium nitroprusside was greater than that needed with the volatile anesthetics.
Esmolol

Esmolol is a cardioselective, ultra-short-acting beta receptor blocking agent that has an elimination half-life of approximately nine minutes. The rapid elimination of this drug is caused by metabolic inactivation by esterases found in the blood and liver. The ultra-short duration of action of esmolol offers a distinct advantage over other beta blockers, because the degree of beta blockade can be controlled and rapidly titrated. During the peri-operative period, the short duration of action can be especially advantageous, considering the rapid and often dramatic changes in hemodynamic status that can occur secondary to the stress of anesthesia and surgery.

Four studies with similar protocols were performed with esmolol in order to ascertain if the drug attenuates the tachycardia and hypertension associated with tracheal intubation. A total of 213 patients participated in these studies. The protocol of the studies included:

1. ASA physical status I-IV.
2. Premedication given 1 to 1 1/2 hour before induction.
3. Sodium thiopental 4-5 mg/kg used for induction.
4. Laryngoscopy and endotracheal intubation took less than 30 seconds.
5. After intubation, anesthesia was maintained with N₂O 60% and oxygen 40%, halothane 0-1.6% or enflurane 0.5-1.2%.

In these studies, 105 patients received a loading dose of esmolol (500 mg/kg/min) for four minutes, followed by a maintenance dose of 300 mg/kg/min during induction and intubation. In the control group of 108 patients, a placebo infusion was used for a corresponding period of time. The infusion regimen was started in both groups four to five minutes prior to induction and was continued for a variable period of time following intubation. In these studies, esmolol was found to cause a significant attenuation in heart rate and systolic blood pressure when the patients were intubated. Similar beneficial effects have been found when esmolol is used in patients who are having coronary artery bypass surgery. Therefore, esmolol appears to be beneficial in certain patients when used during the induction and intubation period of anesthesia.

Conclusion

Beta blockers have become important drugs in the treatment of certain medical conditions. Because of the many beta blockers now available, it has become difficult to stay current on this class of drug. It is hoped that this review will facilitate the understanding of the clinical uses of these drugs.

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Joe R. Williams, CRNA, MS, received his undergraduate education at Baylor University School of Nursing, Houston, and completed his nurse anesthesia education at Duke University Medical Center, Durham, North Carolina, in 1972. He was awarded an MS in pharmacology from the University of North Carolina. At present, Mr. Williams is the Director of the Nurse Anesthesia Program at the University of Alabama in Birmingham.