Esmolol and beta-adrenergic blockade

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Tachycardia often presents difficult management problems in anesthesia. Because it increases myocardial oxygen demand so sharply, tachycardia can quickly place patients at risk of myocardial ischemia. It can occur for any number of reasons. Deepening the anesthetic, either with inhalation agent or opioids, will ablate increases in heart rate, but changes in heart rate are often transient and changes in anesthetic depth are often not.

Esmolol (Brevibloc®) is a unique, short-acting beta blocker that is strongly beta1 selective at usual clinical doses. As with other beta blockers, esmolol becomes less selective for the beta1 receptor as its dose is increased. It is metabolized by red blood cell esterases resulting in a half-life of 9 minutes. Fifteen minutes after a bolus dose, esmolol is difficult to detect in the plasma. Its metabolites have clinically undetectable activity and are eliminated renally.

Esmolol may be administered by intermittent, intravenous bolus doses or by continuous infusion. Infusions should be preceded by loading doses. Dose range varies with the patient's status, clinical situation, concomitant medications, and desired result. Patients receiving esmolol should be monitored because of its bradycardic and hypotensive effects.

Key words: Beta-adrenergic blockade, esmolol, hypertension, pharmacology, tachycardia.

Introduction

Changes in heart rate and arterial blood pressure, reflecting alterations in cardiovascular homeostasis, commonly occur in the perioperative setting. Perioperative tachycardia and hypertension may be caused by many factors, one being an increase in sympathetic or adrenergic activity secondary to noxious stimuli. These hemodynamic changes may stress the cardiovascular integrity of the patient, and no group of patients is more at risk than those with coronary artery disease (CAD).1,4 In patients with CAD undergoing myocardial revascularization, the reported incidence of prebypass myocardial ischemia is 37-50%, and the incidence of perioperative myocardial infarction is almost three times higher in the presence of ischemia.1,2

Tachycardia has been shown to be more dangerous than hypertension to patients with CAD. When patients with fixed coronary lesions are stressed to similar increases in myocardial oxygen consumption, the stress of tachycardia results in a statistically significant higher incidence of ischemia than that caused by hypertension.5 In the Slogoff and Keats study, the incidence of ischemia was significantly more frequent in patients who were tachyycardic (heart rate greater than 100 beats per
minute), before or during anesthesia, than in those
who developed hypertension or hypotension. A
more recent paper by the same authors has demonstrat-
ed a doubling in the incidence of ischemia
when the heart rate was greater than or equal to 110
beats per minute. The tachycardia is dangerous because it disrupts
the delicate equilibrium between myocardial oxygen
supply and demand. Tachycardia decreases
myocardial oxygen supply and increases myocar-
dial oxygen demand.

- **Decreased supply.** The left ventricular myo-
cardium is perfused during diastole, the period
following contraction of the ventricles, when the
ventricles fill with blood. When the heart rate in-
creases, both the systolic and diastolic time inter-
vals decrease. However, the diastolic time (coro-
nary perfusion time) decreases more than systolic
time, and hence the systolic/diastolic time ratio
increases. This results in less blood perfusing the
myocardium. Moreover, when the heart rate in-
creases beyond a certain point, there is insufficient
filling of the ventricles, insufficient blood pumped
into the aorta, and, therefore, insufficient coronary
diastolic pressure. The heart rate where this occurs varies
from patient to patient. In patients with CAD it is
generally recommended that the heart rate be kept
less than 110 beats per minute. The situation is
worsened in cases of increased left ventricular end
diastolic pressure (decreased coronary perfusion
pressure), seen in the ischemic heart, where the
flow of blood may be impeded from the epicardium
to the endocardium.

- **Increased demand.** Increases in heart rate in-
crease the work of the heart and therefore increase
myocardial oxygen consumption. This results in a
greater demand for oxygen by the heart.

- **Result.** The resulting imbalance between
myocardial oxygen supply and demand may be toler-
ated for a time in the normal heart with its
greater reserve. However, in cases of CAD, the coro-
nary blood flow is limited by fixed lesions and the
vessels are unable to dilate and effectively provide
an adequate supply of oxygen to already stressed
myocardial cells. Thus, in the patient with ischemic
heart disease, tachycardia may result in decompensa-
tion and thus ischemia at lower heart rates than
in patients with normal coronary arteries.

**Tachycardia and anesthesia**

Tachycardia is a common event in the periop-
ervative period. It is frequently present prior to and
during induction, during laryngoscopy and intu-
bation, and with the onset of noxious stimulation
(skin incision, sternotomy, and dissection). Tachy-

Tachycardia may also result from intense sympathetic
stimulation such as with electroconvulsive therapy
for depression. In many cases the tachycardia could
have been prevented by deepening the anesthetic.
However, the stimulation may be of a short dura-
tion, and deepening the anesthetic would result in
either a prolonged anesthetic time or overwhelm
the short-lived stimulus and result in hypotension.

When one is presented with a tachycardic pa-
ient, one needs to understand the other causes of
elevated heart rate. For supraventricular tachycar-
dias these include: catecholamine releasing states
(light anesthesia, inadequate analgesia, and anxie-
try), reflex tachycardia secondary to hypotension
or hypovolemia, adrenocortical insufficiency, ana-
phylaxis, hypercarbia, hypoxia, fever, malignant
hyperthermia, hyperthyroidism or thyroid storm,
pheochromocytoma, sepsis, congestive heart fail-
ure, pulmonary embolism, drug or alcohol with-
drawal states, electrolyte disturbances, chronic dys-
rhythmias secondary to conduction anomalies or
abnormalities, and administration of vagolytic or
beta-adrenergic agonists. These causes need to be
understood before embarking on a treatment plan.

**Beta receptors and beta-adrenergic blockade**

Beta-adrenergic receptors are part of the sym-
pathetic nervous system and exist throughout the
body on the postsynaptic membranes of effector
cells. There are two types of beta receptors, beta_1 (\(\beta_1\))
and beta_2 (\(\beta_2\)). \(\beta_1\) receptors are found primarily in
the heart and are responsible for increases in heart
rate, conduction (automaticity), and contractility.
They may also be responsible in other parts of the
body for lipolysis and insulin release but this is
controversial. \(\beta_2\) receptors are found in blood ves-
els, lungs, smooth muscle, heart, and other parts of
the body. Stimulation of \(\beta_2\) receptors results in vaso-
dilation; bronchodilation; gastrointestinal, uterine
and bladder relaxation; glycogenolysis; and lipoly-
sis. The function of the cardiac \(\beta_2\) receptors is not
known at this time. It is also not clear which beta
receptor is responsible for ADH and renin release,
although there is evidence that \(\beta_2\) blockade will
decrease plasma renin levels that are usually in-
creased with sodium nitroprusside use. The natu-
ral catecholamines, epinephrine, norepinephrine
and dopamine all have beta receptor agonist activ-
ity, but norepinephrine has almost no \(\beta_2\) and dopa-
mine has only slight \(\beta_2\) activity.

Beta-adrenergic blockade involves competitive
antagonism or competition with beta agonists for
beta-adrenergic receptors. \(\beta_1\) selective blockers are
cardioselective beta-adrenergic inhibitors and com-
pete preferentially for \(\beta_1\) receptor sites found pri-
arily in the heart. Nonselective β-adrenergic blockers compete for cardiac (β₁) and β₂ (bronchial, peripheral vascular, etc.) receptors. Oral and parenteral beta-adrenergic blockade has been utilized for many years for the management of supraventricular tachycardias, hypertension, and acute ischemic syndromes. Perioperatively, they have been used in the prevention and treatment of tachycardia and hypertension, especially that secondary to intubation. In CAD patients undergoing coronary revascularization, propranolol, a nonselective beta-blocker, has been shown to successfully attenuate the stress induced changes in heart rate and, to a lesser extent, changes in mean arterial pressure, pulmonary artery wedge pressure and cardiac index in proportion to a function of its plasma concentration. The properties of commonly used beta blockers are shown in Table I.

**Calcium channel blockers**

With the advent of calcium channel blocking agents it was felt that they may present an alternative to beta blockade. Calcium channel blocking drugs inhibit the influx of calcium ions through the slow calcium channel into cardiac and smooth muscle cells. The different agents possess antiarrhythmic and vasodilatory properties to varying degrees. They are widely used in cardiology and intensive care medicine. The only currently available intravenous, antiarrhythmic calcium channel blocker is verapamil. Verapamil reduces muscle contraction and prolongs the conduction through, and refractoriness of, the A-V node. It is useful in the treatment of supraventricular tachycardias. The overall effects of this calcium channel blockade are a decrease in contractility, systemic vascular resistance and the ventricular response to atrial fibrillation and flutter. Verapamil is relatively long acting with an elimination half-life of 5-7 hours. Other calcium channel blockers are used for their coronary vasodilatory properties.

**Efficacy of conventional beta and calcium channel blockade**

Studies into the effectiveness of conventional beta and calcium channel blockers have failed to document the efficacy of calcium channel blockade when compared to beta blockade. In the intensive care unit setting, propranolol and other beta-adrenergic blockers were shown to decrease the immediate postinfarct death rate by 15%, while calcium channel blockade had no effect. In patients with CAD undergoing coronary revascularization, Slogoff and Keats found that preoperative beta blockade resulted in a statistically significant decrease in perioperative ischemia when compared to groups of patients receiving either no blockade or calcium channel blockade alone (no statistical difference in ischemia between the latter two groups). This was due to the effectiveness of preoperative beta blockade in keeping the heart rate less than 110 beats per minute. Preoperative calcium channel blockade offered no benefit in controlling or preventing perioperative ischemia in this study.

Despite the effectiveness of conventional intravenous beta blockers, both they and the calcium channel blockers have several disadvantages in their perioperative use. They all have long elimination

<table>
<thead>
<tr>
<th>Name</th>
<th>Beta₁ selectivity¹</th>
<th>ISA²</th>
<th>MSA³</th>
<th>Half-life (hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acebutolol</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>8</td>
</tr>
<tr>
<td>Atenolol</td>
<td>++</td>
<td>-</td>
<td>-</td>
<td>6 to 9</td>
</tr>
<tr>
<td>Esmolol</td>
<td>++</td>
<td>-</td>
<td>-</td>
<td>0.15 (9 minutes)</td>
</tr>
<tr>
<td>Labetalol⁴</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>5.5</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>++</td>
<td>-</td>
<td>-</td>
<td>2.5 to 4.5</td>
</tr>
<tr>
<td>Nadolol</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>16 to 24</td>
</tr>
<tr>
<td>Oxprenolol</td>
<td>-</td>
<td>++</td>
<td>+</td>
<td>2 to 3</td>
</tr>
<tr>
<td>Pindolol</td>
<td>-</td>
<td>+++</td>
<td>+</td>
<td>3 to 4</td>
</tr>
<tr>
<td>Propranolol</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>2 to 4</td>
</tr>
<tr>
<td>Timolol</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>4 to 6</td>
</tr>
</tbody>
</table>

1. May show beta₂ effects at higher doses.  
2. Intrinsic sympathomimetic activity.  
3. Membrane stabilizing activity.  
4. Labetalol also has alpha-blocking activity.

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1. = None
2. + = Mild
3. ++ = Moderate
4. +++ = Most

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half-lives (2 to 7 hours). This prolongs their effects often longer than desired or needed. Even though the effects of beta blockade may be reversed with administration of sufficient beta agonists, this is often undesirable. Adverse effects that may occur, such as cardiac failure, hypotension, bradycardia, atrioventricular block, bronchoconstriction or bronchospasm (nonselective beta blocker) may be difficult to reverse. Finally, these agents are difficult to titrate to the desired level of blockade. Once administered, there is no way to make downward adjustments in the degree of beta or calcium blockade, should this be necessary.

**Esmolol**

Esmolol was developed to meet the need for a highly titratable, cardioselective, intravenous beta blocker. It has a rapid onset and ultrashort duration of action. Steady state blood levels are achieved within 5 minutes after administration of a loading infusion (500 μg/kg) and within 30 minutes if administered without a loading infusion. The distribution and elimination half-lives are 2 and 9 minutes respectively. This is contrasted to elimination half-lives of 2 to 5.5 hours for commonly utilized beta blockers (propranolol, labetalol, and metoprolol).

After discontinuing administration of esmolol, recovery from beta blockade begins within 1 to 2 minutes, substantial reversal of effect is seen within 10 to 12 minutes, and complete reversal of beta blockade is seen within 20 minutes. This rapid diminution of effect after discontinuing administration is due to rapid metabolism of esmolol. Esmolol undergoes rapid hydrolysis of its ester linkage by esterases in the cytosol of red blood cells with the formation of an acid metabolite and methanol. The acid metabolite has 1/1500th the beta-blocking potency of esmolol, undergoes renal elimination with a half-life of 3.7 hours, and blood levels of this metabolite do not correlate with beta blockade following esmolol administration. Methanol blood levels, measured during esmolol infusions, were similar to endogenous levels and were less than 2% of levels associated with methanol toxicity. Dosage adjustment is not necessary in patients with hepatic or renal disease; however, caution is urged with end-stage renal failure patients where the acid metabolite may accumulate.

There is a direct correlation between the blood levels of esmolol and degree of beta blockade and a rapid reversal of effect after discontinuation of the drug. These pharmacokinetic properties of esmolol have resulted in a drug that is highly titratable and may be used in the acute care setting where a high degree of control is necessary.

The final criterion in the search for a new beta-adrenergic blocker was cardioselectivity. In clinically useful dose ranges, esmolol has shown relative β₁ selective blockade and is therefore useful in the treatment of patients with mild chronic obstructive pulmonary disease. Similar to the other β₁ selective blockers, at higher doses, esmolol may lose its β₁ selectivity, resulting in some β₂ blockade. The unique kinetics of esmolol with rapid diminution of effect after discontinuation of the drug allow for titration to the lowest possible effective dose, minimizing β₂ effects. Rapid recovery is beneficial should undesirable side effects or overdose occur. Esmolol has no clinically significant intrinsic sympathomimetic activity or membrane-stabilizing cardiodepressant effects at usual clinical doses. Esmolol has no alpha-blocking activity. It is recommended that the concentration of esmolol administered be no greater than 10 mg/mL due to venous irritation or phlebitis.

Esmolol has been indicated for use in control of supraventricular tachyarrhythmias and hypertension in the acute care setting and in perioperative situations. In clinical studies of supraventricular tachycardia, esmolol produced dose-related reductions in heart rate, systolic and diastolic blood pressure, and rate-pressure product. Esmolol has shown to be more beneficial than placebo; equal in efficacy to propranolol but, perhaps, the agent of choice due to its short duration of effect. It has also compared favorably to verapamil in the treatment of supraventricular tachycardia.

Esmolol is useful in the treatment of tachycardia and hypertension in patients with CAD. This use is based on successful animal studies where esmolol reduced myocardial infarct size following induced coronary artery occlusion and prevented early functional deterioration of the myocardium following reperfusion. In dogs with acutely induced coronary artery occlusion, esmolol infusion preserved coronary perfusion pressure and certain left ventricular hemodynamic variables, preserved endocardial to epicardial blood flow ratios, and decreased the magnitude of lactate production when compared with placebo.

In patients with stable CAD, both esmolol and propranolol lowered heart rate, systolic blood pressure, rate-pressure product, left ventricular ejection fraction and cardiac index. During exercise, significant decreases in heart rate, systolic blood pressure, rate-pressure product, cardiac index, and right ventricular ejection fraction were noted with a greater magnitude of drug effect than at rest. However, these parameters quickly reversed when the esmolol infusion was discontinued but persisted after propranolol.
In patients with acute ischemic heart disease and elevated ventricular heart rate, esmolol infusion produced statistically significant decreases in ventricular rate, systolic blood pressure, rate-pressure product, and cardiac index without changes in wedge pressure. These changes reverted to baseline with discontinuation of esmolol treatment. Finally, esmolol may be useful in the control of heart rate in patients with left ventricular dysfunction. Although esmolol depressed left ventricular ejection fraction in these patients, it could be titrated to minimize this effect. Hence, esmolol may be safely used to control heart rate and systolic blood pressure in patients with acute, unstable, and severe ischemic heart disease with a greater degree of control than that which can be gained with currently available, longer acting, intravenous beta blockers.

**Recommended dosage guidelines for supraventricular tachycardia**

Doses of esmolol for the treatment of supraventricular tachycardia range from 50 to 200 μg/kg/min, although doses as low as 25 and as high as 300 μg/kg/min have been used. A dose of 300 μg/kg/min may be associated with an increase in adverse effects such as hypotension. A suggested regimen for infusion titration is as follows:

1. Begin a loading infusion of 500 μg/kg/min for 1 minute. Follow with a maintenance infusion of 50 μg/kg/min for 4 minutes and assess the patient’s response. If a sufficient response is obtained, continue the maintenance infusion.

2. If a sufficient response is not obtained or maintained, repeat the loading infusion for 1 minute and begin a maintenance infusion of 100 μg/kg/min for 4 minutes.

3. Repeat evaluation as above. Repeat loading infusion and increase maintenance infusion in increments of 50 μg/kg/min until either sufficient response is obtained, or 300 μg/kg/min is reached.

4. As the end point for heart rate is reached or an unwanted decrease in blood pressure occurs, the increase in maintenance infusion may be lowered from 50 to 25 μg/kg/min and the loading infusion may be omitted.

**Adverse effects of esmolol**

In a series of clinical studies utilizing esmolol for the treatment of supraventricular tachycardia, the incidence of hypotension was 13% to 39%, symptomatic with diaphoresis and dizziness in 12% and asymptomatic in 25%. Hypotension resolved during infusion in 63% and after discontinuation of the infusion in 80%. The treatment of hypotension is cessation of the infusion until blood pressure returns to normal and then resuming the infusion (without a bolus) at a lower rate. In these studies, hypotension occurred more frequently with infusions greater than 200 μg/kg/min. Evidence from animal studies suggests that hypotension may be caused by a negative inotropic effect of moderate to large doses of esmolol (≥300 μg/kg/min) that is not due to β1 blockade. Other adverse effects include: diaphoresis (10%); peripheral ischemia (1%); pallor, flushing, bradycardia, chest pain, heart block, and pulmonary edema (<1%); dizziness and somnolence (3%); bronchoconstrictive symptoms (<1%); nausea (7%); vomiting (1%); and skin inflammation (8%).

The β1 selectivity of esmolol is not absolute and high therapeutic doses may result in some β2 blockade. Hence, esmolol must be used with caution in patients with bronchospastic or peripheral vascular disease where β2 blockade may result in bronchospasm or peripheral vasoconstriction. The advantage of esmolol compared to other β1 selective or nonselective beta blockers is its short duration of action should these unwanted effects occur.

In diabetics, β-blockade will decrease insulin release. Also, esmolol may mask hypoglycemic-induced tachycardia. Esmolol is relatively contraindicated in most compensatory tachycardias unless transient treatment of the tachycardia is necessary until definitive treatment of the underlying cause is effective. Esmolol is contraindicated in sinus bradycardia, second- or third-degree heart block, cardiogenic shock, and overt cardiac failure. In patients with congestive heart failure, beta blockade of sympathetic function may result in a worsening of the myocardial function and failure.

**Esmolol and anesthesia**

Perhaps the greatest use for esmolol is in anesthesia where there is a current need for short-acting, titratable, cardioselective, beta-adrenergic blocker. Previous attempts at beta blockade were successful in attenuating the tachycardic and hypertensive responses to noxious stimuli. However, this blockade frequently lasted longer than the stimulus it was administered to treat. The efficacy of esmolol infusions in attenuation of these short-lived adrenergic responses are well reported in the literature. Esmolol was shown to have normal pharmacokinetics under anesthesia; however, hypothermia in vitro was found to decrease esmolol hydrolysis. Animal studies suggested that the effect of esmolol was more pronounced under anesthesia.

In a nonrandomized, controlled study in pa-
patients with ischemic heart disease and normal left ventricular function, Menkhaus showed that esmolol significantly attenuated the heart rate response to intubation, resulted in significantly lower rate-pressure products, antagonized the beta-adrenergic effects of norepinephrine, had a rapid onset of action and short duration of effect under anesthesia, and, therefore, was useful. In a double-blind, randomized study, Girard found that a loading dose of 500 \( \mu \)g/kg/min for 1.5 minutes followed by an infusion of 200 \( \mu \)g/kg/min significantly prevented tachycardia in response to intubation in patients undergoing coronary revascularization. Other studies in open heart surgery patients confirmed this dose. Cucchiara used a slightly higher infusion rate (300 \( \mu \)g/kg/min) in a double-blind, randomized study of patients undergoing carotid endarterectomy and found that esmolol significantly blunted the maximal increase in heart rate and blood pressure in response to intubation. Harrison utilized the same infusion rate (300 \( \mu \)g/kg/min) in a double-blind, randomized study of patients undergoing coronary revascularization and showed a decrease in prebypass ischemia from 20% in the control group to 6.7% in the esmolol group.

Despite agreement on the efficacy of esmolol in attenuating the response to adrenergic stimulation, controversy continues into the proper dosage and method of administration. Gold found the optimal dosing regimen to prevent tachycardia and attenuate the hypertension associated with ketamine induction and subsequent endotracheal intubation to be a 500 \( \mu \)g/kg/min loading dose over 4 minutes, followed by a 300 \( \mu \)g/kg/min infusion. However, other studies showed that there was a ceiling effect of esmolol at a 100 \( \mu \)g/kg/min infusion rate for attenuating the increases in heart rate and systolic blood pressure following sodium thiopental induction and intubation.

Finally, there is controversy regarding whether to bolus esmolol or infuse it. Studies have shown that bolus doses of 100 to 200 mg are sufficient to blunt increases in heart rate and blood pressure in response to laryngoscopy and intubation, especially in patients with CAD. Another study showed that a 200-mg bolus will decrease or prevent both hypertension and tachycardia while a 100-mg bolus will only attenuate the tachycardia. This latter paper confirms our experience in obstetrics. On a milligram per kilogram basis, some have shown that it takes 1.0 to 1.5 mg/kg to attenuate the tachycardic and hypertensive responses to intraoperative stimuli, rather than the 0.5-mg/kg bolus doses advocated by others. Bolus doses of 1.5 mg/kg were shown to blunt changes in heart rate without changes in mean arterial pressure or cardiac index. However, three mg/kg bolus doses were found to decrease mean arterial pressure and cardiac index.

Similar controversy occurs in regard to beta blockade for electroconvulsive therapy. Some advocate a 500-\( \mu \)g/kg loading infusion over 1 minute followed by 100 to 300 \( \mu \)g/kg/min infusions and have noted no change in seizure duration. Others have claimed that 100-mg boluses are as adequate. Still others claim that you need greater than or equal to 2 mg/kg to blunt the adrenergic stimulus, and that this bolus dose, as well as 200-mg boluses, decreases seizure time. No matter which regimen you choose, experience using this drug leads one to consider its use not only in preventing the effects of beta-adrenergic stimulation, but as a rapid treatment for tachycardia and hypertension when they occur.

Another controversy concerns the use of esmolol in obstetrics where we have found esmolol to be effective in attenuating the tachycardic and hypertensive response to intubation. Ostman et al. documented a rapid but relatively small transplacental passage in the gravid ewe with rapid elimination from both maternal and fetal plasma. However, a recent study by Eisenach and Castro showed that in ewes, maternally administered esmolol may result in prolonged fetal beta blockade and fetal hypoxemia.

Finally, two points need to be made regarding esmolol and control of hypertension. In post-aortocoronary bypass patients, esmolol has been shown superior to sodium nitroprusside (SNP) and nifedipine as it prevents ischemia by decreasing myocardial work. Esmolol potentiates SNP-induced hypotension, decreases the SNP-associated increase in plasma renin activity, decreases plasma norepinephrine levels, and decreases heart rate and rebound hypertension. Esmolol also improves oxygenation by attenuating the SNP-induced inhibition of hypoxic pulmonary vasconstriction. Esmolol has also been effective in the perioperative management of pheochromocytoma.

**Conclusion**

Esmolol is a rapidly acting, short duration, titratable, \( \beta_1 \) (cardioselective) blocker that is useful in the attenuation and/or treatment of the tachycardic and hypertensive adrenergic responses to noxious stimuli and supraventricular tachycardias. It is also useful, either alone or in combination with other agents, for induced hypotension. Esmolol's cardioselectivity at therapeutic doses affords us the ability to provide short-acting beta blockade to a
whole population of patients to whom it was previously denied. The ability to titrate the drug to effect and its short elimination half-life, make it an ideal agent for use in anesthesia to combat tachycardia-induced myocardial ischemia and perhaps useful in myocardial protection.

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