Vasodilator therapy and the anesthetist: A review of nitroprusside, labetalol, hydralazine and nitroglycerin

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Vasodilators are important adjuncts to anesthetic management in a variety of clinically encountered situations. Examples include: congestive heart failure, preeclampsia/eclampsia, management of aortic cross-clamping and treatment of perioperative hypertension.

In this article, the authors review four commonly utilized vasodilating agents: sodium nitroprusside, labetalol, hydralazine and nitroglycerin. Comparisons and contrasts are made among the drugs. Each agent is presented in four sections: (1) mechanism of action, (2) indications, (3) adverse actions, and (4) dosage, administration and precautions.

Sodium Nitroprusside
Mechanism of action. Sodium nitroprusside (SNP) is a direct vasodilator, with equal arterial and venous effects: the results are reduced afterload, preload and blood pressure. SNP dilates all vascular beds, including cerebral and pulmonary. The onset of action is virtually instantaneous, and effects disappear immediately upon discontinuance of the drug. SNP stimulates the synthesis of cyclic guanosine monophosphate (GMP) in smooth muscle which, in turn, leads to the formation of nitric oxide radicals. These radicals initiate a chain of events which lead to dephosphorylation of contractile proteins in smooth muscle, causing relaxation and vasodilation.

The unstable SNP molecule rapidly degrades, releasing free cyanide (CN\(^-\)) into the circulation. Metabolism of the CN\(^-\) involves two processes. Oxidation of hemoglobin to methemoglobin provides binding sites for CN\(^-\), producing cyanmethemoglobin. In addition, endogenous thiosulfate binds CN\(^-\) in the presence of the enzyme rhodanase to yield thiocyanate, which is then excreted in the urine. Conditions associated with abnormalities in the CN\(^-\)-thiocyanate metabolic pathway, such as Leber’s optic atrophy (genetic) or tobacco amblyopia (induced), may lead to early cyanide toxicity. In those situations, SNP may be contraindicated.

Indications. SNP is the standard vasodilator in cardiac and vascular surgery. The combination of afterload and preload reduction enhances SNP myocardial performance by decreasing the risk of ischemia secondary to increased afterload. SNP also controls the hypertensive response to aortic cross-clamping and allows for volume expansion. This minimizes the occurrence of hypotension following cross-clamp release.

Hypertensive emergencies are particularly amenable to SNP. The extremely rapid onset of action and ability to titrate dosage to achieve almost any desired pressure makes SNP a front-line agent in this setting.

SNP is beneficial in the management of pediatric and adult congestive heart failure. Pump function improves secondary to decreased afterload and preload, and in the carefully monitored patient, SNP is described as having minimal side effects.
The use of SNP for controlled hypotension during anesthesia and surgery is controversial. Supporters note that SNP reliably induces easily adjusted hypotension, and does so when other agents have been ineffective. In addition, normotension returns shortly after discontinuance of the infusion. On the other hand, SNP increases the risk of intrapulmonary shunting and hypoxemia, which may decrease critical organ perfusion, including coronary perfusion, and is associated with reflex tachycardia and rebound hypertension. Additional agents such as beta blockers are often required to minimize SNP side effects. For these reasons, some have suggested that SNP not be used for routine establishment of intraoperative hypotension.

Nitroprusside is the most commonly used agent in the management of hypertension associated with excision of pheochromocytoma. Rapidly changing hemodynamic circumstances and the ability to titrate SNP make it useful in the face of fluctuating blood pressure. Excessive catecholamine release during tumor manipulation may require the use of additional agents to augment SNP control of blood pressure. Indications for SNP therapy are summarized in Table 1.

**Table 1**

<table>
<thead>
<tr>
<th>Agent</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium nitroprusside (SNP)</td>
<td>Hypertension secondary to cardiac/vascular surgery, Hypertensive emergencies, Management of congestive heart failure, Controlled intraoperative hypotension, Management of pheochromocytoma, Management of hypertension when other pharmacological agents have failed</td>
</tr>
<tr>
<td>Labetalol</td>
<td>Hypertension secondary to cardiac/vascular surgery, Hypertensive emergencies, Hypertension of pregnancy, Management of pheochromocytoma, Controlled intraoperative hypotension, Management of acute hypertension in patients with: Angina pectoris, Renal insufficiency/failure</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Adjunctive drug in chronic hypertension, Hypertensive emergencies, Management of congestive heart failure, Hypertension of pregnancy</td>
</tr>
<tr>
<td>Nitroglycerin (NTG)</td>
<td>Hypertension secondary to cardiac/vascular surgery, Hypertensive emergencies, Management of congestive heart failure, Controlled intraoperative hypotension, Angina pectoris</td>
</tr>
</tbody>
</table>

Adverse reactions. Excessive hypotension may occur secondary to rapid administration, unintentional bolusing, or when SNP is used in the hypovolemic patient. In the patient with myocardial disease, SNP-induced diastolic hypotension can aggravate myocardial ischemia, increasing the risk of myocardial infarction. Systolic hypotension, in turn, has the potential to exacerbate cerebrovascular disease. SNP-induced hypotension is also associated with tissue hypoxia in liver and skeletal muscle, these effects not being attributed necessarily to hypotension but to altered microvascular hemodynamics.

Hyperdynamic responses to SNP therapy include tachycardia and increased cardiac output, often occurring after major cardiac surgery in patients with hypertrophied left ventricles, or when SNP is the sole agent used for controlled hypotension. The mechanism is thought to be reflex excessive catecholamine activity triggered by sudden afterload decrease. In the patient with ischemic heart disease, the hyperdynamic response can lead to an imbalance in myocardial oxygen supply and demand; the result may lead to myocardial infarction.

SNP tachyphylaxis is characterized by an increasing dose required to sustain a desired blood pressure, the necessity for additional therapy and suggestion of cyanide toxicity. It may be necessary to decrease the rate or discontinue the SNP infusion. Additional therapy may include beta-blocking agents, other vasodilators, or calcium-channel blockers. Sensitivity to SNP with increasing age has been reported. Diminishing baroreceptor function, resistance of cardiac adrenoreceptors to catecholamine stimulation and altered sensitivity to the direct effects of SNP have been suggested as possible mechanisms for the lower dose requirements in the elderly.

Since SNP is a ubiquitous vasodilator, it can be expected that cerebral and pulmonary vascular beds will vasodilate in response to SNP. The patient with increased intracranial pressure (ICP) treated with SNP is at risk for decreased cerebral perfusion pressure secondary to increased cerebral blood volume. It is suggested that SNP infusion not be started until hyperventilation is well established and the dura is opened.

Increased intrapulmonary shunting has been described following SNP infusion in patients with normal lung function secondary to pulmonary hypoperfusion. SNP also inhibits hypoxic pulmonary vasoconstriction. Patients with significant,
chronic obstructive lung disease demonstrate a lower tendency toward intrapulmonary shunting with SNP because of a fixed reduction in blood flow to affected areas.\(^\text{30}\)

Cyanide toxicity occurs when the amount of circulating CN\(^-\) exceeds the ability of metabolic pathways to detoxify the compound. CN\(^-\) binds to cytochrome oxidase and poisons the oxygen transport system, leading to the development of metabolic acidosis.\(^\text{6}\) Other symptoms of cyanide toxicity include tachyphylaxis, mental changes, nausea and vomiting.\(^\text{31,32}\) Previous reports described the phenomenon as occurring in patients on long-term therapy at relatively high infusion rates and total doses (> 8 \(\mu g/kg/min\); total dose > 10 mg/kg).\(^\text{32,33}\) Recent evidence suggests, however, that the occurrence of toxicity is unpredictable, and may appear in as little as 6 hours, and at infusion rates and dosages well below recognized safe levels (4 \(\mu g/kg/min\); total dose> 1.5 mg/kg).\(^\text{8,27,31,32}\) Toxicity is more likely in susceptible patients, including unstable postcoronary bypass patients, the critically ill, patients with compromised renal function, and, possibly, smokers due to chronic exposure to cyanide in cigarette smoke.\(^\text{3,34}\) Mortality is high and may approach 50%.\(^\text{35}\) Therapy is aimed at oxidizing hemoglobin to methemoglobin (amyl nitrate and sodium nitrite) and providing substrate (sodium thiosulfate) for CN\(^-\) detoxification.\(^\text{6}\) Dosages of available antidotes for cyanide toxicity are given in Table II.

### Table II

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium thiosulfate</td>
<td>150 mg/kg in 50 ml IV over 15 minutes</td>
<td>Drug of choice except in patients with diminished renal function</td>
</tr>
<tr>
<td>Amyl nitrite</td>
<td>Inhalation every 2 mins</td>
<td></td>
</tr>
<tr>
<td>Sodium nitrite</td>
<td>5 mg/kg in 20 ml IV over 3-4 minutes</td>
<td></td>
</tr>
<tr>
<td>Hydroxocobalamin</td>
<td>0.1 mg/kg IV</td>
<td>Alternate agent in abnormal renal function</td>
</tr>
</tbody>
</table>

**Dosage, administration and precautions.** SNP dosage information is summarized in Table III. Infusion should begin at lower dose range. If inadequate response is seen at the upper dose range, supplemental agents should be added to control the pressure. The use of an infusion pump is mandated by the drug’s extreme potency. Light decomposes this unstable compound, so aluminum foil shielding is necessary.

Direct arterial pressure monitoring is required with SNP. Arterial blood gases should be checked frequently for the development of intrapulmonary shunting or metabolic acidosis. Adequate intravascular volume is necessary to avoid excessive falls in blood pressure; exaggerated response to SNP should first suggest hypovolemia.\(^\text{6}\)

### Labeltalol

**Mechanism of action.** Labetalol is a unique vasodilator in that it is a competitive inhibitor of both alpha- and beta-adrenergic receptors. It is a nonselective beta blocker and is 1-4 times less potent than propranolol.\(^\text{34}\) Labetalol is a specific alpha\(_1\) receptor antagonist. Having little effect on presynaptic alpha\(_2\) receptors,\(^\text{35}\) it is 6-10 times less potent than phentolamine.\(^\text{34}\) The ratio of beta:alpha blockade produced by labetalol is 4:8:1.\(^\text{41}\)

In contrast to other beta adrenergic antagonists, labetalol reduces blood pressure primarily by decreasing systemic vascular resistance (SVR), secondary to its alpha\(_1\) antagonism.\(^\text{35}\) While the primary effects of propranolol are a reduction in heart rate and cardiac output, the beta-blocking actions of labetalol serve mainly to prevent reflex increases in heart rate in response to decreased SVR.\(^\text{37}\) Labetalol preserves cardiac output while decreasing myocardial oxygen consumption and improving coronary hemodynamics.\(^\text{38,39}\) Important protective effects for the patient with ischemic heart disease.\(^\text{40}\) Labetalol undergoes hepatic metabolism to inactive glucuronides, which are then excreted in the urine; less than 5% of the drug is excreted unchanged.\(^\text{35}\) The dose of labetalol should be reduced in the presence of chronic liver disease.\(^\text{34}\) Renal disease does not appear to affect the elimination of labetalol and no alteration of dosage is necessary in patients with renal insufficiency.\(^\text{35,41}\) Labetalol is removed by peritoneal dialysis, though total body clearance is not enhanced by dialysis.\(^\text{42}\)

**Indications.** The primary indication for the intraoperative use of labetalol is for the management of hypertension. Because of its relatively longer onset and duration of action, when compared to SNP, labetalol is less suited for use when rapid changes in blood pressure are expected, e.g., for the management of pressure changes associated with aortic clamping and declamping.

Labetalol, administered either as a continuous infusion or by bolus injection, has been shown to significantly reduce blood pressure, with minimal change in heart rate or cardiac output, in hyperten-
Table III
Summary of dosage information

<table>
<thead>
<tr>
<th>Agent</th>
<th>Preparation</th>
<th>Dose</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium nitroprusside (SNP)</td>
<td>50 mg/250 ml D5W yields 200 µg/ml</td>
<td>Continuous infusion 0.25-4.0 µg/kg/min max=8.0 µg/kg/min</td>
<td>sec</td>
<td>Effects cease when infusion discontinued</td>
</tr>
<tr>
<td>Labetalol</td>
<td>5 mg/ml</td>
<td>Bolus infusion 0.25 mg/kg in 5-10 mg increments max=300 mg</td>
<td>1-2 min</td>
<td>3-6 hr</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>20 mg/ml</td>
<td>Bolus infusion 5-10 mg increments max=40 mg</td>
<td>15 min</td>
<td>3-6 hr</td>
</tr>
<tr>
<td>Nitroglycerin (NTG)</td>
<td>Dilute in normal saline or D5W to concentration of 100-400 µg/ml</td>
<td>Continuous infusion start at 10 µg/min titrate to effect max=500 µg/min</td>
<td>1 min</td>
<td>5-9 min</td>
</tr>
</tbody>
</table>

tion complicating coronary artery bypass surgery. It is especially beneficial for patients with angina pectoris or ischemic heart disease, increasing exercise tolerance, decreasing the degree of ST segment depression and lowering the frequency of anginal episodes. Postoperative hypertension in association with general anesthesia for routine surgical procedures and for carotid endarterectomy has been effectively managed with labetalol. In all cases, significant blood pressure reductions were achieved with little or no change in other cardiovascular parameters and without evidence of side effects.

Little has been published regarding the use of labetalol for controlled hypotension. A recent report of controlled hypotension for orthognathic surgery concluded that labetalol was an attractive alternative to traditional hypotensive methods because of the effective establishment of hypotension, ease of administration, preservation of arterial oxygen percent saturation (SaO₂), and lack of adverse reactions when compared to other commonly used agents.

The treatment of hypertension complicating pregnancy must take into account placental and fetal effects of the agents being employed. SNP is advocated for use only after delivery because of the potential for fetal or neonatal cyanide toxicity. Labetalol reduces maternal blood pressure without interfering with placental or fetal blood flow. Fetal and neonatal effects of labetalol have been reported to be clinically insignificant, and labetalol is thought to compare favorably with hydralazine in the management of pregnancy-induced hypertension.

Reports concerning the use of labetalol for intraoperative management of pheochromocytoma have been rare. It has been suggested that labetalol is specifically indicated for pheochromocytoma and other conditions characterized by catecholamine excess. In one study, small, repeat bolus injections (total dose = 100 mg) produced stable blood pressure without affecting heart rate or causing excessive hypotension. Indications for labetalol therapy are summarized in Table I.

Adverse reactions. Adverse reactions occurring with single administrations of labetalol are rare. None of the studies examined for this review, in which either single treatment or short-term (less than 48 hours) labetalol use was described, noted any significant side effects as a result of treatment.

One report of apparent anaphylaxis following oral labetalol (100 mg), manifested by urticaria, angioedema and hypotension, has been described. The patient was successfully treated with corticosteroids and antihistamines. Apparent allergy to labetalol was documented by the onset of fever in two patients; discontinuance of labetalol ameliorated the fever. The most common side effects of labetalol therapy for chronic hypertension are: postural hypotension (18%), nausea (15%), fatigue (13%), and skin rash (5%).

Dosage, administration and precautions. Labetalol dosage information is presented in Table III. Bolus doses greater than 1.5 mg/kg have been associated with precipitous falls in blood pressure, nausea, vomiting and evidence of cerebral ischemia. The contraindications to labetalol are generally the same as for other beta-adrenergic blockers. It is contraindicated in patients with asthma, congestive heart failure, sinus bradycardia and atrioventricular block greater than first degree. Of note here are studies which document labetalol's effec-
tiveness and lack of side effects or adverse cardiovascular complications in congestive heart failure and chronic obstructive pulmonary disease and asthma. In such situations, labetalol is recommended only if other antihypertensive therapies are ineffective.

**Hydralazine**

*Mechanism of action.* The hypotensive effect of hydralazine results from its ability to relax smooth muscle in precapillary arterioles. Chelation of certain trace metals which may be required for smooth muscle contraction has been suggested. The resulting dilation reduces vascular resistance and mean arterial blood pressure. There is minimal effect on the venous capacitance vessels. The onset for reduction of mean arterial blood pressure is approximately 15-20 minutes, although peak hemodynamic effects may not occur for 60 minutes. Regional differences exist and vasodilation is more pronounced in carotid, mesenteric, renal and coronary vasculatures.

In both laboratory animals and man, hydralazine increases cardiac output and heart rate. These increases may be explained by two mechanisms: a reflex effect, and a combination of the reflex effect and a direct cardiot stimulatory effect. Hydralazine's reflex effect involves a reduction in mean arterial blood pressure which activates baroreceptors and sympathetic discharge. This occurs in the presence of a relatively normal venous tone resulting in an increased heart rate and cardiac output. The cardiot stimulatory effect demonstrates a direct, positive chronotropic and inotropic response. However, in the patient with congestive heart failure, there is no consistent change in heart rate or blood pressure.

Hydralazine is metabolized in the liver by acetylation. The amount of N-acetyltransferase in the liver is genetically determined. Patients with high levels of this enzyme (fast acetylators) require higher dosages of hydralazine to control pressure, while those with lower levels (slow acetylators) require a lower dose. Hydralazine and its metabolites are mainly excreted in the urine. Patients with severe renal impairment require decreased doses.

*Indications.* Hydralazine is mainly utilized as an adjunctive drug in the progressive treatment of hypertension. Because of its ability to reduce afterload, it is also administered to patients for treatment of congestive heart failure.

In specific situations, hydralazine may be indicated for intraoperative control of blood pressure. Because of its slow onset of action, it should not be utilized in situations where an immediate decrease in blood pressure is desired. Due to the potential for a reflex or direct tachycardia, hydralazine is contraindicated in patients with acute hypertension and preexisting angina. Concurrent administration of beta-adrenergic blocking agents may be warranted to decrease accompanying tachycardia.

Hydralazine increases renal blood flow and therefore may be a useful agent, in decreased doses, for patients with hypertension and coexisting renal impairment. In animal studies, higher doses produced vasodilation in several vascular beds, while lower doses (0.05-0.1 mg/kg) produced dilation only in the renal vasculature.

The effect of any vasodilator upon ICP may be significant, therefore, factors such as the history, CO₂ level, integrity of the dura, EEG and the use of diuretics should be considered.

Hydralazine has demonstrated the ability to inhibit norepinephrine-induced contractions in uterine arteries. In the obstetrical setting, hydralazine may have an unpredictable onset, produce reflex tachycardia, cause abrupt falls in blood pressure and reduce uterine blood flow. Fetal distress has also been reported in conjunction with the use of this drug. Nevertheless, hydralazine is recognized as the drug of choice for acutely lowering blood pressure in pregnant patients and is the most widely used antihypertensive agent in the treatment of preeclampsia and eclampsia. Its use has been well described and is guided by diastolic pressure.

Hydralazine has been effectively utilized in the treatment of chronic congestive heart failure. These studies associate hydralazine with increasing cardiac index and stroke volume while decreasing SVR. No significant differences were noted with respect to heart rate or pulmonary wedge pressures.

Short-term effects of hydralazine in patients with chronic obstructive pulmonary disease, pulmonary hypertension and cor pulmonale have been reported. They include improvement of cardiac index, pulmonary resistance, pulmonary artery pressure, oxygen transport and alveolar ventilation. Indications for hydralazine therapy are summarized in Table I.

*Adverse reactions.* Side effects attributed to hydralazine include: flushing, urticaria, pruritis, nasal congestion, conjunctivitis, headache, dizziness, nausea, vomiting and palpitations. In addition, anginal attacks and changes in the electrocardiogram (ECG), indicative of myocardial ischemia, have been observed. Tachycardia, with its consequences, may be effectively controlled by concurrent administration of beta-adrenergic blocking drugs.
In chronic therapy, hydralazine may lead to a rheumatic state and/or a lupus-like syndrome. These situations, however, are more likely to occur in patients receiving doses greater than 100 mg per day or those patients who are slow acetylators.

Intraoperatively, hydralazine may create an unacceptable level of hypotension with or without an undesirable tachycardia. These events may be avoided by ensuring a normovolemic state, careful titration of the drug and concurrent administration of beta-blocking agents.

**Dosage, administration and precautions.** Hydralazine dosage information is summarized in Table III. It may be administered by continuous infusion or small incremental doses. The authors recommend the latter option with an initial dose of 5-10 mg and a caution that the anticipated effect may be delayed for approximately 15 minutes. Repeat doses may be necessary.

Hydralazine, if taken preoperatively, should be continued up to and including the day of surgery. Anesthetic considerations for these individuals should be no different than those afforded other hypertensive patients. Invasive arterial blood pressure monitoring is not normally indicated when administering intraoperative hydralazine.

### Nitroglycerin

**Mechanism of action.** Nitroglycerin (NTG) is a direct-acting vasodilator producing relaxation of nearly all smooth muscles including veins, arteries and arterioles. In addition, the smooth muscle of the biliary and gastrointestinal tracts, bronchial, uterine and ureteral tissues are affected

Although the mechanism of this biochemical action is not completely understood, NTG may also be involved with the production of nitric oxide, which may stimulate the production of cyclic GMP, leading to relaxation of smooth muscle.

The initial hemodynamic effects of NTG are not uniform with respect to actions on arteries and veins. In lower dosages, capacitance vessels are more sensitive to NTG than conductance or resistance vessels. In higher concentrations, there is an arteriolar vasodilation which accompanies the preexisting venodilation, the latter having the dominant effect.

Due to the above mechanisms, NTG significantly reduces preload and afterload. Consequently, left ventricular filling pressure, right atrial pressure, central venous pressure, mean arterial pressure and SVR are also reduced. The reduction in systolic pressure is greater than the decrease in the diastolic pressure, which may assist in preserving myocardial perfusion. Although increases and decreases in both heart rate and cardiac output have been reported, these parameters are usually unchanged. Due to the decreased workload of the heart, NTG administration results in a reduction of myocardial oxygen demand. Factors which may influence hemodynamic responses to NTG include: quantity of drug, activity of the sympathetic nervous system, status of the left ventricle, baseline hemodynamic values and prior treatment with nitrates.

The onset of NTG is extremely rapid. Increases in venous capacitance and decreases in left ventricular filling pressures and cardiac work occur within one minute of administration. The elimination half-life of NTG has been reported between 1-4 minutes. Return of arterial blood pressure occurs in approximately 9 minutes following cessation of infusion. When compared to SNP, NTG produces a more controllable decrease in blood pressure with less tendency to produce an "overshoot."

NTG is rapidly metabolized in the liver. Reductive hydrolysis, catalyzed by the enzyme glutathione-organic nitrate reductase, is the major metabolic pathway for biotransformation and degradation of NTG. A reduction in liver function may impair clearance of the drug. The resulting metabolites are excreted in the urine. However, the plasma nitrate levels of patients with or without impaired renal function appear to be comparable.

**Indications.** NTG is indicated for the treatment of unstable angina pectoris, congestive heart failure complicating acute myocardial infarction, perioperative control of blood pressure during coronary revascularization and controlled hypotension during noncardiac surgery.

NTG has been effectively utilized in patients undergoing coronary revascularization to offset intraoperative hypertensive responses. In a similar population, NTG significantly improved several hemodynamic parameters to include: mean arterial, right atrial, pulmonary artery, pulmonary capillary wedge pressures, SVR, myocardial oxygen consumption and rate pressure product. In patients undergoing coronary artery bypass procedures, NTG improved myocardial metabolism when compared to SNP.

Advantages of administering NTG for controlled arterial hypotension during total hip replacements have been reported. This report demonstrated benefits of NTG, when compared to SNP, which included: significantly less blood loss and no ECG changes suggestive of myocardial ischemia. NTG was also successfully utilized to control mean arterial blood pressure and decrease blood loss in orthognathic patients. An additional report,
involving controlled hypotension for Harrington rod procedures, demonstrated decreased blood loss when pressure was controlled with NTG and compared to SNP.101

In the obstetrical setting, it is not uncommon to be faced with a patient having an unacceptable level of hypertension about to undergo cesarean section and general anesthesia. Hydralazine, which is frequently utilized in this setting, may be beneficial in certain situations. However, due to its slower onset of action and longer duration, its selection may be precluded. NTG has been effectively administered to severely preeclamptic patients undergoing cesarean section and general anesthesia.102 In this study, NTG successfully blunted the hypertensive response to endotracheal intubation. Furthermore, there were no adverse fetal or maternal side effects noted.

For patients undergoing vascular procedures, NTG has been effective in reducing induction and intraoperative episodes of myocardial and/or hemodynamic complications.103-106 During induction of patients with stable angina undergoing short vascular procedures, NTG has been described as an effective method of preventing hypertensive responses, increases in pulmonary capillary wedge pressures, incidences of ST depression and the need for additional narcotic.103 NTG has been effectively utilized during procedures which require aortic cross-clamping.104, 105 These studies demonstrate the effectiveness of NTG in preventing rises in SVR and falls in cardiac output. During aortic cross-clamping, NTG has also been associated with a restoration of myocardial contractility.104 In patients with angina pectoris undergoing vascular procedures, intraoperative myocardial ischemic events were more effectively reduced when an infusion rate of NTG 1.0 μg/kg/min was compared to a rate of 0.5 μg/kg/min.106

NTG has been recommended for deliberate controlled hypotension in neuroanesthesia.107, 108 Others have recommended its use over SNP.109 However, one must take into consideration the effect of these drugs with respect for their potential of increasing ICP. NTG has been associated with increasing ICP and decreasing intracranial compliance.110 When utilizing NTG for deliberate hypotension in this population, one should be cognizant of concerns previously mentioned in the hydralazine section.

In patients with diminutive peripheral veins and difficult intravenous access, topical NTG ointment has been successfully utilized in decreasing the amount of attempts and trauma required for intravenous cannulation.111 Indications for NTG therapy are summarized in Table I.
fects and potential adverse reactions. In this article, the authors have attempted to provide a basic introduction to these agents, which the nurse anesthetist may use to make safe, effective clinical judgements.

REFERENCES


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This course is of interest to all members of the team caring for the cardiac surgery patient, including physicians and surgeons, perfusionists, critical care nurses and nurse anesthetists, cardiologists, anesthesiologists, intensivists, and biomedical health professionals. Continuing education credit will be awarded to physicians, perfusionists, nurses and nurse anesthetists. To receive a program with registration forms, send a postcard with your name, specialty, and address to: C.R.E.F., P.O. Box 33185, San Diego, CA 92103.

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