

Managing Hypertension in Patients With Stroke

Are You Prepared for Labetalol Infusion?

Cindy Harrington, RN, CCRN

The statistics on high blood pressure and stroke are sobering: every year, about 600 000 persons in the United States have a stroke (83% ischemic, 17% hemorrhagic).¹ Roughly half of those persons have hypertension,¹ and hypertension develops after stroke in many patients who were previously normotensive.² In the landmark 1996 National Institute of Neurological Disorders and Stroke (NINDS) trial of recombinant tissue plasminogen

activator (rtPA) in patients with stroke, 19% of the subjects were hypertensive at the time of their acute ischemic stroke; within 24 hours, that rate had tripled.³ For these reasons, critical care nurses need expertise in managing hypertension in patients who have had an acute ischemic stroke.

Comprehensive stroke protocols include administration of alteplase (rtPA) for acute ischemic stroke and provide guidelines for management of hypertension; intravenous labetalol (Normodyne, Trandate) is often recommended.⁴⁻⁸ Although intravenous labetalol is often used in critical care units and medical units, its administration as an intravenous infusion may not be common, and many nurses may not be knowledgeable about blood pressure issues related to patients who have had an acute ischemic stroke.

In this article, I focus on the nursing considerations for management of hypertension, including administration of labetalol, in patients with acute ischemic stroke treated with rtPA. In addition, I describe the relationships among cerebral blood flow, blood pressure, and stroke: concepts that direct guidelines for care. This overview is appropriate for staff nurses who work in emergency, intensive care, and medical units. Nurse educators and managers involved in the development of stroke protocols and educational programs also will find the information useful.

Meet Ruth
11:30 PM

Your night shift is just getting started when the telephone rings. The emergency department has admitted 68-year-old Ruth Casey. Diagnosis? Acute ischemic stroke. Treatment? Thrombolytic therapy with alteplase.

You listen to the report, assign a bed, and check the equipment in the room. If you are like many nurses, you also pull out the policy and procedure book because your commu-

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nity hospital has not been using this treatment for very long; you want to review the details of the new pathway.

12:15 AM

Ruth arrives awake and alert with slight residual hemiparesis on the right side, a right-sided facial droop, and dysarthria. The rtPA was administered in the emergency department, and now an isotonic sodium chloride solution is being infused intravenously at a rate of 50 mL/h. Neurological findings and vital signs are unchanged from those in the report, except for the blood pressure, which has increased to 220/100 mm Hg. Heart and respiratory rates are stable. Oxygen saturation is fine at 98%. Ruth answers, “No,” when you ask if she is in pain.

1:30 AM

You set the blood pressure auto-cuff, perform another neurological assessment, and notify the physician. Ten minutes pass quickly; the blood pressure is unchanged. The physician determines that the pressure must be treated and prescribes labetalol, 10 mg administered intravenously over 2 minutes, followed by an infusion if necessary.

1:45 AM

You have the labetalol vials in front of you. For years you have given labetalol to your patients with myocardial infarction; you are familiar with its cardioprotective effects. But have you ever administered it as an infusion? Are you aware of the mixing guidelines? The precautions? The concerns about patients’ safety? Can you explain why treatment of hypertension in this patient must be approached delicately?

Normal Physiology of Cerebral Blood Flow

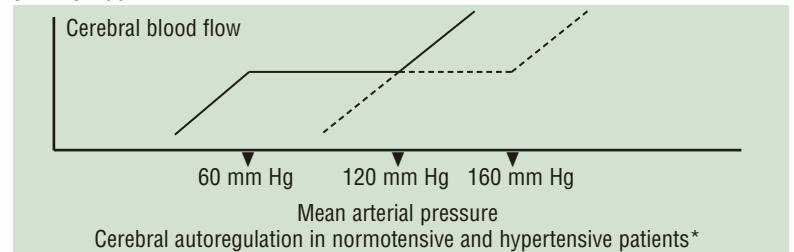
Complex autoregulatory mechanisms seek to maintain cerebral blood flow at a constant rate of about 750 mL/min.⁹ The 2 main regulatory factors are cerebral perfusion pressure and cerebral vascular resistance. Cerebral perfusion pressure, the blood pressure gradient across the brain, is in turn influenced by mean arterial pressure and intracranial pressure. Like vascular resistance elsewhere in the body, cerebral vas-

cular resistance is determined by the diameter of the vessels. In response to elevated blood pressure (from any cause), cerebral vessels constrict, restricting cerebral blood flow to a homeostatic rate. Thus, “myogenic autoregulation”⁹ protects the brain from increasing intracranial pressure by limiting cerebral intravascular volume (see Figure).

Cerebral myogenic autoregulation can usually accommodate a wide range of fluctuations in blood pressure, from a mean arterial pressure

Cerebral perfusion pressure (CPP) is the pressure gradient across the brain. It is the difference between mean arterial pressure (MAP) and intracranial pressure (ICP): CPP = MAP – ICP.

Cerebral blood flow (CBF) is regulated by CPP and cerebral vascular resistance (CVR): CBF = CPP/CVR.



Intracranial pressure is determined by the volumes of cerebral blood, cerebrospinal fluid, and brain matter within the fixed cranial vault. An increase in one component, without a compensatory decrease in another, will result in an elevated ICP (Monroe-Kellie doctrine).

Myogenic autoregulation maintains a constant CBF despite changes in MAP:

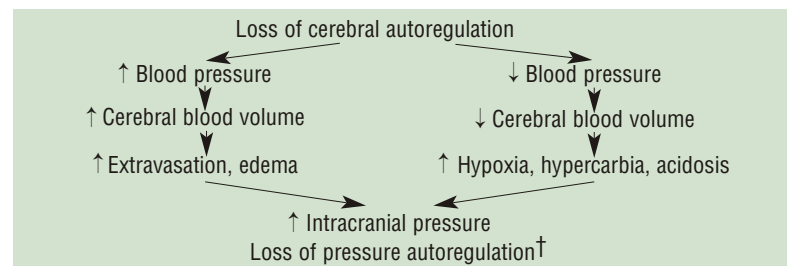
↑ MAP → cerebral vasoconstriction (↑CVR) → stable CPP and normal CBF.
(Without autoregulation:

↑ MAP → ↑CPP → ↑CBF → ↑cerebral blood volume → ↑ICP.)

Hypertension after a stroke may compensate for increased ICP:

↑ ICP → ↓ CPP (remember: CPP = MAP – ICP). Compensatory ↑ MAP → ↑ CPP.

Autoregulation and compensatory mechanisms will fail when brain tissue is injured or when the ICP or MAP falls far outside the normal range.



Cerebral blood flow: key concepts.

*Reprinted from Varon and Marik,^{10(p216)} with permission.

†Reprinted from Thelan et al,^{11(p830)} with permission.

as low as 60 mm Hg^{2,9,10,12} to a high of 120 mm Hg¹⁰ or even 150 mm Hg.^{2,9,12} In chronic hypertension, however, both limits are shifted upward. This shift in limits must be considered when treating stroke patients who have preexisting chronic hypertension. Although autoregulation adjusts to broad variations in blood pressure, it will fail when brain tissue is injured, when intracranial pressure becomes extremely high (>40 mm Hg⁹), or when mean arterial pressure is outside the patient's usual range. Once autoregulation fails, cerebral blood flow becomes passively regulated by the systemic mean arterial pressure.

Finally, metabolic factors that act independently of the perfusion pressure affect cerebral blood flow. Increased metabolic rate (fever), hypercapnia (in both local brain tissue and the blood), acidosis, hypoglycemia (<70 mg/100 mL),² and hypoxia all stimulate cerebral vasodilation and a subsequent increase in cerebral blood flow. Conversely, hypocapnia and alkalosis cause vasoconstriction.

Mechanisms for Hypertension in Acute Ischemic Stroke

After a stroke, hypertension can develop in previously normotensive patients for a variety of reasons. Sudden and severe hypertension, particularly after administration of alteplase, is alarming because it may indicate—or cause—intracerebral hemorrhage. In addition to hypertension, signs and symptoms of intracerebral hemorrhage include neurological decline, new headache, nausea, and/or vomiting. Any indication that a patient may have intracerebral hemorrhage demands immediate discontinuation of the

rtPA, reassessment of neurological function, and emergent computed tomography.^{4,7,8,13,14}

Conversely, hypertension may be a vital compensatory mechanism, promoting perfusion to threatened brain tissue. Cerebral ischemia, the consequence of occlusion of the cerebral artery by an embolus or a thrombus, damages the cellular sodium-potassium pump. This damage permits, among other things, an influx of sodium and water; the cells swell, cerebral edema ensues, and intracranial pressure becomes elevated. Without a corresponding increase in blood pressure, cerebral perfusion pressure would be impaired (see Figure).

Furthermore, higher than normal blood pressure may be of particular importance in the protection of the ischemic penumbra, a zone of injured, but still viable, brain tissue that surrounds the central core of infarction. Although the ischemic penumbra is temporarily perfused by collateral circulation, as increasing intracranial pressure impairs brain perfusion, the collateral vessels dilate.¹² Sustaining cerebral perfusion pressure through the dilated vessels requires a greater blood pressure.

In addition to stroke, hypertension may be associated with other acute conditions such as aortic dissection, myocardial infarction, unstable angina, acute renal failure, pulmonary edema with respiratory failure, eclampsia, pheochromocytoma, and microangiopathic hemolytic anemia. Finally, emotional stress, bladder distention, pain, and hypoxia can cause elevations in blood pressure after a stroke.¹⁵ Searching for the cause of hypertension is vital to prescribing the most appropriate therapy.

Treatment of Hypertension in Patients With Ischemic Stroke Treated With Alteplase

In the NINDS trials, treatment of hypertension correlated with less favorable outcomes than did untreated hypertension. This unexpected finding led the researchers to advocate “careful attention to” but “gentle management” of blood pressure,³ a position that is echoed throughout the literature.^{10,11,15-17} Although reluctant to define optimal blood pressure for patients after acute ischemic stroke, experts in the management of stroke do agree about when high blood pressure should be treated.

The American Heart Association defines high blood pressure as either a systolic blood pressure greater than 140 mm Hg or a diastolic blood pressure less than 90 mm Hg.¹⁸ As a result of the NINDS trials, however, parameters for stroke treated with rtPA are considerably higher. During and/or after administration of rtPA, 2 successive measurements, taken 5 to 10 minutes apart, should meet 1 or both of the following criteria before antihypertensive agents are given^{3,4,6-8}: systolic blood pressure greater than 180 mm Hg or diastolic blood pressure greater than 105 mm Hg. (Note: rtPA is contraindicated in stroke patients with sustained systolic blood pressures >185 mm Hg or diastolic blood pressure >110 mm Hg who are unresponsive to either nitroglycerin paste or a maximum of two 10- to 20-mg doses of intravenous labetalol.⁴⁻⁸)

High blood pressure in patients who have had an ischemic stroke is managed with restraint; the risk of intracerebral hemorrhage must be

weighed against the need to preserve perfusion to the ischemic penumbra. When treatment is indicated, the drug used should have a rapid onset, a predictable effect, a low risk for causing precipitous decreases in blood pressure, a minimal effect on cerebral blood flow, and a relatively short half-life; it should also be easy to titrate. Because it meets most of these standards, labetalol is recommended for these patients.^{3-10,14,15,19,20}

Actions and Effects of Labetalol

Labetalol is a complex medication. It acts on α - and β -receptor sites in distinct locations in the heart, lungs, and vasculature. Specifically, labetalol is a selective, postsynaptic α_1 -adrenergic blocking agent and a nonselective, β -adrenergic blocking agent (Table 1). Because of its unique combination of actions, labetalol decreases systemic blood pressure and vascular resistance without markedly affecting cardiac output, renal hemodynamics, or cerebral blood flow.

Labetalol is metabolized in the liver and excreted in the urine. After

intravenous injection, its effects occur within 5 minutes, peak within 15 minutes, and persist at least 2 to 4 hours (effect lasting 24 hours has been reported). The half-life is 5.5 hours.²¹⁻²³

Contraindications and Precautions

Contraindications (Table 2) and precautions (Table 3) for administration of labetalol are related to the effects of the drug on the sympathetic nervous system. Although it may not produce the same degree of sympathetic blockade as pure α - or β -adrenergic blockers, restrictions for each of these categories are generally applied to labetalol.²¹⁻²⁶ The manufacturers also urge caution in administering labetalol to patients with ischemic heart disease (myocardial infarction and exacerbation of angina have been documented), vasospastic angina or pheochromocyt-

Table 2 Contraindications to the use of labetalol²¹⁻²⁶

Contraindication	Reason
Bronchial asthma	Drug may seriously increase airway resistance
Cardiogenic shock Overt heart failure	Negative inotropic effect can decrease cardiac output and increase pump failure
Severe bradycardia	Drug may prolong atrioventricular nodal conduction, reduce heart rate, and impair cardiac output
Greater than first-degree heart block (Note: prescribing information does not define severe bradycardia.)	Reduced automaticity may further decrease heart rate and reduce cardiac output
Significant hypotension	
Sensitivity to the drug (or to its components)	

Table 1 Actions and effects of labetalol

Specific receptor sites of labetalol action ²¹	Effect of adrenergic blockade
α_1 : Vascular smooth muscle	Vasodilatation
β_1 : Myocardium	Decreased automaticity Prolonged conduction Decreased contractility
β_2 : Bronchial and vascular smooth muscle	Bronchial constriction Vasoconstriction

Table 3 Precautions in the use of intravenous labetalol²¹⁻²⁶

Precaution	Reason
Major surgery	Drug may suppress protective tachycardia in response to bleeding
Any degree of heart failure	Negative inotropic effect may decrease cardiac output and increase pump failure
Diabetes or hypoglycemia	Beta-blockade can have unpredictable results with these patients because it may: Mask the signs and symptoms of acute hypoglycemia (tachycardia, tremors, palpitations) Cause hypoglycemia by interfering with glycogenolysis Promote hyperglycemia by inhibiting insulin secretion from the pancreatic islet cells and interfering with the effects of sulfonylureas
Emphysema Chronic obstructive pulmonary disease Chronic bronchitis	Drug can increase airway resistance; it also competes for receptor sites with exogenous catecholamines, interfering with the therapeutic effects of β -agonist bronchodilators (intravenous use has not been studied in patients with these conditions; the manufacturers state that labetalol should not be administered to these patients)
Hyperthyroidism Thyrotoxicosis	Drug may blunt tachycardia, an important assessment parameter

toma (paradoxical hypertensive crisis has been reported for both), and severe hepatic dysfunction (labetalol metabolism may be impaired; the drug can also further injure hepatocytes). Pregnant women (pregnancy category C: may be harmful to the fetus) and nursing women (small amounts excreted in breast milk) should receive this medication only when the benefit outweighs the risks and when alternatives are not available. Finally, safety and efficacy of labetalol in children have not

been established for intravenous administration.^{22,23}

Drug Interactions

Standard nursing practice calls for obtaining a thorough and accurate medication history before a new drug is administered. Today, this history must include information on use of over-the-counter medicines and herbal remedies, some of which have long-acting effects. Because patients may not consider over-the-counter drugs and herbal remedies

when reporting their medication lists, nurses must specifically ask about these preparations (Table 4).

Adverse Effects and Reactions

Most of the undesirable effects of labetalol appear to be dose related, are both mild and transient, and occur early in treatment. Because labetalol blocks the ability of the heart to pump faster in response to changes in body position, dramatic orthostatic hypotension can occur. Symptomatic postural hypotension

Table 4 Drug interactions with labetalol

Substance	Interaction
β -Adrenergic agonist bronchodilators ²¹⁻²⁶ : albuterol (Proventil, Ventolin, Volmax), bitolterol mesylate (Tornalate), levalbuterol hydrochloride (Xopenex), metaproterenol sulfate (Alupent), pirbuterol acetate (Maxair), terbutaline sulfate (Brethine), salmeterol xinafoate (Serevent)	Labetalol may blunt bronchodilatory effects in patients with bronchospasm; a greater than normal dose of the bronchodilator may be required
Calcium channel blockers: verapamil, diltiazem ²¹⁻²⁴	Significant atrioventricular block may occur
Cevimeline hydrochloride (Evoxac) ²⁴	Cardiac conduction disturbances may occur
Oral cimetidine ²¹⁻²⁶ (Tagamet)	Cimetidine increases the bioavailability of oral labetalol, an important consideration when a patient is switched from intravenous to oral labetalol; reduced doses of labetalol may be required
Halothane anesthesia ²¹⁻²⁶ as well as isoflurane, ether, and cyclopropane ²⁴	Myocardial depression (severe with halothane) may occur
Liothyronine (Cytomel) ²⁴	Effectiveness of labetalol is inhibited
Mefloquine hydrochloride (Lariam) ²⁴	Electrocardiographic abnormalities, cardiac arrest may occur
Monoamine oxidase inhibitors ^{21,24}	Increased bradycardia may occur
Nitroglycerin and other antihypertensives ²¹⁻²⁶	Hypotension may be increased because labetalol may block reflexive tachycardia
Sulfonylureas (Amaryl, DiaBeta, Diabinese, Glucotrol, Glynase, Micronase)	Competition for β_2 -receptor sites may antagonize the effects of these antidiabetic agents, promoting hyperglycemia ²⁴
Tricyclic antidepressants ^{21-24, 26}	Tremors may occur (reported with oral labetalol)
Cold and allergy over-the-counter drugs (as well as prescription medications) that contain sympathomimetics (ephedrine and pseudoephedrine) ²⁴ : Primatene, Triaminic, Sudafed, Actifed, Neo-Synephrine Khat, ma huang ^{27,28} and other sympathomimetic herbs Licorice and yohimbe ²⁹ and herbs with vasoconstrictive or hypertensive effects	Effectiveness of labetalol may be inhibited, with potential for increased pressor response
Hawthorn ²⁴ (<i>Crataegus laevigata</i>) and other herbal remedies used for cardiovascular disease and/or diuretic therapy	Hypotension may be increased

is the most common adverse effect,^{21,25} and patients may experience dizziness, light-headedness, and/or syncope.

Other serious adverse effects include increased atrioventricular block, bradycardia, worsening of heart failure, ventricular arrhythmia, dyspnea, wheezing, and bronchospasm. In addition, after intravenous administration, at least 5% of patients experience 1 or more of the following: scalp tingling, nausea, and transient elevated serum levels of urea nitrogen and creatinine (in patients with preexisting renal insufficiency).^{22,23} Management of overdose is aimed at countering the effects of the sympathetic blockade (Table 5).

Guidelines for Administration of Labetalol in Patients With Acute Ischemic Stroke Treated With rtPA

Mixing Guidelines

Labetalol must be diluted for intravenous infusion and can be mixed in most common intravenous solutions such as isotonic sodium chloride solution and 5% dextrose in water. Both commercial preparations of labetalol are supplied in vials that contain 5 mg/mL. Three specific solutions, 1.25 mg/mL, 2.5 mg/mL, and 3.75 mg/mL, are stable for at least 24 hours.^{21,30,31} In addition, more dilute preparations of 1 mg/mL or 2 mg/3 mL may be prepared.^{21-23,25,26,30,31} Table 6 offers mixing guidelines for each of these 5 concentrations.

Table 5 Management of labetalol overdose²¹⁻²⁶

Sign or symptom	Treatment*
Hypotension	Reduction in or cessation of labetalol therapy Intravenous fluids Vasopressors (norepinephrine or dopamine)
Bradycardia	Atropine and epinephrine
Refractory hypotension and bradycardia	Glucagon
Cardiac failure	Digitalis, diuretics, and dopamine or dobutamine
Bronchospasm	Epinephrine and/or an aerosolized β_2 -agonist
Seizures	Diazepam

*See each drug's prescribing information for dosage and administration.

Table 6 Labetalol mixing guidelines*

For this concentration	→	2 mg:3 mL	1 mg:1 mL	1.25 mg:1 mL	2.5 mg:1 mL	3.75 mg:1 mL
Add this amount of labetalol	→	200 mg (40 mL)	100 mg (20 mL)	125 mg (25 mL)	250 mg (50 mL)	375 mg (75 mL)
To this volume of isotonic sodium chloride solution	→	250 mL	80 mL	75 mL	50 mL	25 mL
Yielding this final solution	→	200 mg/290 mL [†]	100 mg/100 mL	125 mg/100 mL	250 mg/100 mL	375 mg/100 mL

Titration chart

Ordered dose, mg/min	Resulting hourly dose, mg/h	2 mg/3 mL solution	1 mg/1 mL solution	1.25 mg/mL solution	2.5 mg/mL solution	3.75 mg/mL solution
1	60	90	60	48	24	16
2	120	180	120	96	48	32
3	180	270	180	144	72	48
4	240	360	240	192	96	64
5	300	450	300	240	120	80
6	360	540	360	288	144	96
7	420	630	420	336	168	112
8	480	720	480	384	192	128

*Labetalol can be mixed in many intravenous fluids: isotonic sodium chloride solution, 5% dextrose in water, 5% dextrose in water mixed with isotonic sodium chloride solution, Ringer's solution, lactated Ringer's solution, 5% dextrose in water mixed with lactated Ringer's solution.^{21-23,30,31}
[†]200 mg/290 mL is considered equivalent to a 2:3 concentration.^{22,23,31}

Drug Compatibilities and Incompatibilities

When mixed with 5% dextrose in water in a 1:1 solution, labetalol is compatible for 24 hours with the medications listed in Table 7. It is incompatible with alkaline solutions.

Recommended Dose and Administration

Labetalol is administered by repeated bolus doses given by slow intravenous injection or by continuous infusion until the desired blood pressure is achieved. The benefits of continuous infusion may include greater control of antihypertensive action as well as milder and fewer adverse effects (including hypotension).³² Once the target blood pressure is reached, the infusion can be stopped,²¹⁻²⁵ although it is sometimes continued for 24 hours (A. M. Pancioli, MD, University of Cincinnati Medical Center, e-mail, August 21, 2001); in either instance, once the infusion is discontinued, an oral regimen is considered²¹⁻²⁵ (A. M. Pancioli, MD, e-mail, August 21, 2001). An infusion pump is necessary for safe administration. Refer to Table 6 for a titration chart and to Table 8 for a specific dosing regimen. Although the manufacturers recommend a maximum cumulative bolus dose of 300 mg,^{22,23} a maximum of 150 mg is recommended for patients with ischemic stroke treated with rtPA.^{3,4,6-8,14,20}

Promoting Patients' Safety

The goal of hypertension management in acute ischemic stroke is gradual and controlled reduction in blood pressure to a prescribed value.¹⁷ Avoiding precipitous decreases in blood pressure reduces the likelihood of hypoperfusion

Table 7 Drugs compatible and incompatible with labetalol^{30,31}

Compatible*	Incompatible
Aminophylline	Sodium bicarbonate
Calcium gluconate	Furosemide
Dopamine hydrochloride	Heparin
Esmolol hydrochloride	Amphotericin B
Fentanyl citrate	Cefoperazone
Lidocaine hydrochloride	Ceftriaxone
Magnesium sulfate	Nafcillin
Norepinephrine bitartrate	Thiopental
Potassium chloride	Warfarin
Sodium nitroprusside	

*For 24 hours when mixed with 5% dextrose in water in a 1:1 solution.

injury to the brain, myocardium, kidneys,^{10,22,23} and optic nerve.^{22,23} Nursing interventions are outlined in Table 9.

And Ruth?

By 2 AM, you had administered a bolus dose of 10 mg of labetalol intravenously. Ruth's blood pressure changed but remained elevated. The physician ordered a labetalol infusion with a target systolic blood pressure of 185 mm Hg. To limit the intravenous fluid intake, you mixed a solution of 3.75 mg/mL and titrated the infusion accordingly.

Ruth's systolic blood pressure gradually improved, and as it neared the target value, you discussed further treatment with the physician.

5:30 AM

Throughout the past few hours, you have continued to assess Ruth's vital and neurological signs. You have also monitored for postthrombolytic complications. Finally, you have created a restful environment for her and have offered ongoing support and information to Ruth and her family. It has been a busy night!

Table 8 Labetalol regimen for hypertension during and after administration of recombinant tissue plasminogen activator for acute ischemic stroke

Blood pressure, mm Hg	Recommended dose of labetalol* ^{6,8,20}
Systolic 180-230 or diastolic 105-120	10 mg intravenously over 2 minutes Can be repeated or doubled every 10 minutes Maximum dose = 150 mg After the initial bolus, an infusion may be initiated at 2 mg/min, titrated up to 8 mg/min as needed
Systolic >230 or diastolic 121-140	20 mg intravenously over 2 minutes Can be repeated or doubled every 10 minutes Maximum dose = 150 mg After the initial bolus, an infusion may be initiated at 2 mg/min, titrated up to 8 mg/min as needed Consider sodium nitroprusside if no satisfactory response
Diastolic >140	Consider nitroprusside at 0.5 µg/kg per minute

*Labetalol infusions as low as 0.5 mg/min may be effective in lowering blood pressure,²¹ although this rate is not mentioned in stroke protocols.

Table 9 Nursing interventions with labetalol infusion

Intervention	Rationale
Obtain a thorough medical history including prescribed medications, over-the-counter drugs, and herbal remedies	Detect sources of possible drug interactions
Assess for pain, anxiety, bladder distention, and hypoxia before initiating treatment	Identify treatable causes of hypertension
Identify target blood pressure levels Assess blood pressure with patient supine every 5-10 minutes during infusion ²⁶	Lowering the blood pressure in stroke patients can increase cerebral ischemia and has been associated with adverse outcomes for patients
Keep the patient supine during infusion ²¹⁻²⁶	Minimize risk of postural hypotension
Once labetalol is discontinued, monitor the blood pressure every 5 minutes for 30 minutes, every 30 minutes for the next 2 hours, then hourly for at least 6 hours ²¹ After discontinuation, continue to assess for postural hypotension. As blood pressure allows, gradually permit supervised changes in position, starting with raising the head of the bed	Effects may last hours after the drug has been stopped
Instruct the patient to report dizziness, lightheadedness, syncope, dyspnea, wheezing, or nausea	Recognize possible signs and symptoms of serious adverse effects such as cardiovascular compromise and bronchospasm
Assess skin for bruising underneath the blood pressure cuff	Monitor for superficial bleeding related to administration of thrombolytics
Collaborate with the physician if maximum labetalol dose is being approached or if blood pressure increases during infusion	Other medication, such as sodium nitroprusside, may be required. In addition, hypertension may be a symptom of intracranial hemorrhage or another acute condition.
Institute precautions against risk of falling according to hospital policy Explain to the patient and the family why the nurse must be called before the patient may raise the head of the bed, dangle legs at the bedside, or stand	Postural hypotension carries a risk for injury of patient from a fall
Assess patient education needs related to hypertension management and modification of stroke risk factors; include in discharge preparation as appropriate	Achieving target blood pressure reduces the risk of stroke ^{8,33} and stroke recurrence ³⁴ ; hypertension is the most significant risk factor for stroke but only 27% of people with high blood pressure have it under control. ¹ Patient education is crucial to compliance with the medical regimen. ¹⁷

Summary

As community hospitals adopt comprehensive stroke pathways, nurses in critical care areas must prepare for each possible complication. Although the thrombolytic component of a new stroke protocol may receive the most attention, the various details of the pathway—in this case, management of blood pressure—should not be overlooked. Critical care nurses who appreciate the considerations for treatment of hypertension and who can correctly and efficiently administer intravenous labetalol may save precious time and enhance outcomes for patients with stroke who have hypertension.

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