Current Concepts of Cerebrovascular Disease and Stroke

Intensive Care for Acute Stroke in the Community Hospital Setting

The First 24 Hours

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Controversy continues over the value of dedicated "stroke units" since improved outcome in patients treated in such units has not been demonstrated.1 For most hospitals a specialized stroke unit would not be feasible even if effective. We have provided neurological consultation for stroke patients at all 16 acute care hospitals in Cincinnati. Only two hospitals have neurology/neurosurgery intensive care units, but the remaining 14 hospitals have fully equipped medical intensive care units with on-site availability of emergency computed tomography and emergency cerebral angiography. At these hospitals and other hospitals nationally, high-quality intensive care can be provided to stroke patients outside of a specialized unit. Standards of care can be developed that offset varying capabilities for highly specialized neurological monitoring.

Respiratory Management

In the first hours after onset of cerebral infarction, acute respiratory failure is exceedingly rare, even in the setting of brainstem infarction (Table 1). Intubation and ventilatory support are more often acutely indicated with intracerebral hemorrhage (Table 1) and, when necessary, should be managed with consultation from a trained intensivist. In the absence of intracranial pressure (ICP) monitoring, the ICP may be assumed to be increased in patients with a Glasgow Coma Score of 5 or less and will probably be increased with a score of 7 or less.2 Where increased ICP is suspected, the Pco2 should be maintained at approximately 30 torr and probably not below 25 torr.3

Supplemental oxygen may be beneficial for the stroke patient. With aging, lung compliance falls, and physiological shunting increases, resulting in a fall of arterial Po2 so that by the age of 70, the Po2 is usually in the 70-80 mm Hg range.4 Peripheral arterial blood remains adequately oxygen saturated at these levels, but the oxygen saturation (and Po2) within the elderly brain may be considerably lower. Direct measurements in humans are not available; in dogs on ambient air, the Po2 within the deep white matter of the brain has been measured as 50% lower than the peripheral arterial Po2.5

The mechanics of ventilation are commonly abnormal in the stroke patient, hypoxemia may be frequent, and inspiratory power may be limited (e.g., in response to an increase in upper airway resistance).6 Sleep may intensify such ventilatory limitations; in normal subjects, alveolar ventilation falls with sleep and respiratory sensitivity to Pco2 declines.7 The risk of supplemental oxygen with a flow rate of 2-4 l/min via nasal cannula is probably very low. In 6 years of routine use at the University of Cincinnati, we have not documented any episodes of carbon dioxide retention or other manifestation of toxicity. Experimental evidence for oxygen toxicity in the setting of acute cerebral ischemia is limited,8 and relative hypoxemia to a degree ordinarily innocuous may be poorly tolerated in areas of focal cerebral ischemia.9 Secondary benefits of supplemental oxygen include elimination of cigarette smoking and demonstrating the reality of stroke as an illness to those patients unable to recognize or appreciate the meaning of their injury.

Pneumonia is usually a later complication of stroke10 except in patients in whom aspiration is suspected. In patients at risk for aspiration, the chest radiograph should be repeated every 24 hours or earlier if signs of infection are detected.

Cardiac Care and Fluid Management

The incidence of significant cardiac arrhythmias in the setting of acute cerebral infarction is approximately 5-10%.11 The likelihood of a life-threatening arrhythmia in the first 24 hours is less (Table 1). Cardiac monitoring during the first 24 hours after stroke onset will allow detection of these arrhythmias and will also facilitate early detection of acute respiratory failure, seizures, acute hypotension, and attempts of some confused patients to climb out of bed.

Myocardial infarction is also unusual, occurring in approximately 2-3% of patients with cerebral infarction.12 Nonetheless, serial electrocardiograms

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and cardiac enzyme measurements are warranted, particularly given the frequency of underlying coronary artery disease in patients with symptomatic cerebrovascular disease. Recently, catecholamine-induced cardiomyopathy has been discussed in relation to stroke and other acute brain injuries. The frequency of this disorder is unknown, but it should be considered in the setting of a massive infarction or hemorrhage associated with declining cardiac function, cardiomegaly, and elevated cardiac enzymes.

Maximizing the cardiac performance of the stroke patient is intuitively appealing and now is coming under systematic study. Experimentally, regional cerebral blood flow (rCBF) to areas of induced focal ischemia increases in response to increases in cardiac output, with the increases independent of alterations in blood pressure. In humans with cerebral infarction, an abnormally low cardiac output has been reported in the few instances in which invasive measurements have been used, but it was largely secondary to inadequate intravascular volume. In such patients volume expansion is appropriate. In other patients cardiac output may be low as a result of primary cardiac disease (e.g., hypertensive cardiomyopathy) or the effects of aging and could be improved by inotropic agents such as dobutamine, which inappropriately raises blood pressure or exacerbates arrhythmias. The role of cardiac output augmentation, if any, awaits further study (which may become feasible with the development of noninvasive hemodynamic measurement techniques).

The early promise for volume expansion and hemodilution has not been sustained in subsequent large, randomized, controlled studies. Therefore, fluid management should proceed with isotonic solutions given intravenously at a rate to eliminate dehydration if present (75–125 ml/hr for most patients). The rate should be lower if severe hypertension without volume contraction is present. For patients with acute ischemic lesions, glucose-containing solutions may be undesirable. If increased ICP is suspected, the diuresis induced by osmotic diuretics should not be replaced, and intravenous fluids, always isotonic, should be minimized. Frequent measurements of serum electrolytes, glucose, and osmolality are necessary in this situation, and appropriate volume management may be facilitated by hemodynamic monitoring with a Swan-Ganz catheter.

### Blood Pressure Management

Despite the common belief that severe hypertension with acute stroke will often result in neurological deterioration, convincing supporting data are lacking. Nonneurological complications such as acute left ventricular failure, acute myocardial infarction, or aortic dissection are likewise uncommon (Table 1).

In contrast, a rapid fall in blood pressure may be particularly hazardous in the stroke patient. Autoregulation is often blunted in the elderly, and rapid lowering of blood pressure may result in a drop in rCBF. Even with preserved autoregulation, cerebral vessels may be maximally dilated in the area of acute ischemia, creating a pressure-passive region where a decrease in systemic pressure may result in a critical decrease in local flow. Normotensive levels in the 120/80 mm Hg range could be disastrous if therapy is begun at initially elevated levels in the 170–190/90–110 mm Hg range, particularly if the lower pressures are attained rapidly within 30–120 minutes.

When should acute hypertension be treated? Some argue against treatment until controlled studies regarding blood pressure management are developed. An alternative approach would be to treat only at a high threshold. The target systolic pressure of 160–170 mm Hg and target diastolic pressure of 95–100 mm Hg, when attained slowly, seem reasonable for the

### Table 1. Frequency of Emergency Complications of Stroke in First 24 Hours

<table>
<thead>
<tr>
<th>Cerebral infarction (n=41)</th>
<th>ICH (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory failure requiring ventilation</td>
<td>1</td>
</tr>
<tr>
<td>Cardiac arrhythmias requiring parenteral therapy or pacemaker</td>
<td>1</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>0</td>
</tr>
<tr>
<td>Left ventricular failure</td>
<td>2</td>
</tr>
<tr>
<td>Blood pressure &gt;239 mm Hg systolic or &gt;120 mm Hg diastolic</td>
<td>6</td>
</tr>
<tr>
<td>Sustained hypertension requiring parenteral therapy</td>
<td>4</td>
</tr>
<tr>
<td>ICP requiring invasive monitoring</td>
<td>1</td>
</tr>
<tr>
<td>Elevated ICP requiring hyperventilation</td>
<td>1</td>
</tr>
<tr>
<td>Elevated ICP requiring osmotic diuretic therapy</td>
<td>1</td>
</tr>
<tr>
<td>Mass or mass effect requiring surgery</td>
<td>0</td>
</tr>
</tbody>
</table>

*Data are from review of all patients with cerebral infarction or spontaneous intracerebral hemorrhage (ICH) admitted to University of Cincinnati Hospital Neurology/Neurosurgery Intensive Care Unit from July 1987 to June 1988. ICP, intracranial pressure.
TABLE 2.  Algorithm for Emergency Antihypertensive Therapy in Acute Stroke

1. If diastolic BP is >140 mm Hg on two readings 5 minutes apart, then start an infusion of sodium nitroprusside (0.5–10 mg/kg/min).

2. If systolic BP is >230 mm Hg and/or diastolic BP is 121–140 mm Hg on two readings 20 minutes apart, then give labetalol 20 mg intravenously over 1–2 minutes. The labetalol dose may be repeated or doubled every 10–20 minutes until a satisfactory BP reduction is achieved or until a cumulative dose of 300 mg has been administered via this minibolus technique.

   After the initial dosing schedule, labetalol doses may be administered every 6–8 hours as needed. Labetalol is preferably avoided in patients with asthma, cardiac failure, or severe cardiac conduction abnormalities.

3. If systolic BP is 180–230 mm Hg and/or diastolic BP is 105–120 mm Hg, emergency therapy should be deferred in the absence of documented intracerebral hemorrhage or left ventricular failure. If the elevation persists, with two readings 60 minutes apart, then use oral labetalol 200–300 mg two or three times daily as needed. Satisfactory alternative treatments to labetalol include oral nifedipine 10 mg every 6 hours or captopril 6.25–25 mg every 8 hours. If oral monotherapy is unsuccessful or if medications cannot be given orally, give labetalol intravenously as outlined above.

4. In acute stroke patients with systolic BP of <180 mm Hg and/or diastolic BP of <105 mm Hg, antihypertensive therapy is usually not indicated. Patients requiring heparin acutely should be anticoagulated after the BP has been cautiously lowered into an acceptable range. We recommend a target BP range of 150/85–95 mm Hg in patients without prior hypertension or 160–170/90–100 mm Hg in patients with a history of hypertension. Since the benefits of heparin are modest even when indicated, rapid lowering of BP cannot be justified.

BP, blood pressure.

patient without preexisting hypertension. For patients with prior hypertension, a conservative target systolic pressure would be 180–185 mm Hg, and a conservative target diastolic pressure would be 105–110 mm Hg. These target pressures are considerably higher than the pressures that frequently result from current emergency blood pressure management.

An algorithm for acute therapy is outlined in Table 2. Sodium nitroprusside is preferable for very severe elevations, which more often complicate intracerebral hemorrhage than cerebral infarction (Table 1). For less severe elevations the combined alpha- and beta-adrenoreceptor antagonist labetalol, which is effective, may be administered intravenously and may be particularly appropriate since stroke-related severe hypertension may largely reflect increased sympathetic tone. Oral or sublingual nifedipine may be substituted when labetalol is contraindicated but should be used with caution because of excessive hypotensive response in some patients.

Treatment of Elevated Intracranial Pressure

In the first 24 hours of stroke, elevated ICP is primarily associated with spontaneous intracerebral hemorrhage (Table 1). Conservative management includes maintaining the head position at an elevation of 30°, minimizing temperature elevations, and treating agitated, mechanically ventilated patients with small doses of sedatives (or, if necessary, neuromuscular blocking agents). Hyperventilation may be used, as discussed above under "Respiratory Management." Mannitol is the preferred osmotic diuretic; an initial dose of 25–50 g in a 20% solution over 30 minutes may be used, with 25-g doses being repeated every 3–12 hours pending clinical response and serum osmolality (aim for 300–315 mosm). Furosemide may be substituted for (or alternated with) mannitol in patients intolerant of the initially increased circulatory load imposed by mannitol. The role of surgical therapy for intracerebral hemorrhage continues to be controversial.

For patients not selected for early surgical evacuation, ICP monitoring may facilitate medical therapy; if the measurement system involves an intraventricular catheter, pressure-controlled ventricular drainage may supplement the effects of medical therapy.

Bedside Patient Assessment

Monitoring of vital signs and neurological function is recommended hourly for the first 4 hours, every 2 hours for the next 8 hours, and every 4 hours thereafter during the intensive care period. A comprehensive neurological scale is impractical at such frequent intervals while a concise, standardized battery may be inappropriate for many patients. An alternative approach is to select that item from the traditional neurological examination that is judged most likely to change first with neurological deterioration. For example, in a patient with cerebral hemispheric infarction and mild hemiparesis, drift of the extended arm may be tested by the physician and nurse together at the time of admission; the intervals for testing and the specific criteria for physician notification may then be discussed (and recorded in the physician’s written orders). Specific and individualized physician notification orders should also be written with regard to vital sign measurements (e.g., blood pressure ranges).

The value of such concentrated nursing care may be limited, pending development of effective specific stroke therapies. Frequent assessments do provide the opportunity to detect neurological or general medical deterioration rapidly and to intervene as discussed above. Concentrated nursing care does not require an intensive care unit. However, the level of care provided is usually not possible on the general medical wards where the patient:nurse ratio is usually 8:1 or higher (particularly at night).

In the general intensive care units found in most hospitals, specialized equipment for EEG monitoring or transcranial Doppler measurement of cerebral blood velocity is not available. However, ICP monitoring is often feasible. Perhaps most important, staffing of nurses within intensive care units is usually sufficient to provide a 2:1 patient:nurse ratio.
Conclusion

Application of these general principles will be governed by the individual patient’s specific diagnosis and particular clinical problems (for example, a patient with a large cerebellar infarction). Developments in the emergency evaluation of the stroke patient have recently been discussed. The advantages of accurate diagnosis and early therapy (within the first hours after stroke onset) are emphasized.

References


**Key Word** cerebrovascular disorders