After exclusion of pseudoresistant hypertension and secondary hypertension, some patients have severe drug-resistant primary hypertension. Fourth- and fifth-line therapies include a vasodilating β-blocker and spironolactone (even in the absence of primary aldosteronism). Percutaneous catheter-based renal denervation is being investigated as a novel interventional approach to treat drug-resistant hypertension.

Acute Severe Hypertension

Of all the patients in the emergency department, 25% have an elevated BP. *Hypertensive emergencies* are acute, often severe, elevations in BP that are accompanied by acute or rapidly progressive target-organ dysfunction such as myocardial or cerebral ischemia or infarction, pulmonary edema, or renal failure. *Hypertensive urgencies* are severe elevations in BP without severe symptoms and without evidence of acute or progressive target-organ dysfunction. The key distinction and approach to the patient depends on the state of the patient and the assessment of target-organ damage, not simply the absolute level of BP. The full-blown clinical picture of a hypertensive emergency is a critically ill patient with a BP greater than 220/140 mm Hg, headaches, confusion, blurred vision, nausea and vomiting, seizures, heart failure, oliguria, and grade III or IV hypertensive retinopathy (Fig. 12-14).

Hypertensive emergencies require immediate admission in an intensive care unit for intravenous therapy and continuous BP monitoring, whereas hypertensive urgencies can often be managed with oral medications and appropriate outpatient follow-up in 24 to 72 hours. The most common hypertensive cardiac emergencies are acute aortic dissection, hypertension after coronary artery bypass grafting, acute myocardial infarction, and unstable angina. Other hypertensive emergencies include eclampsia, head trauma, severe body burns, postoperative bleeding from vascular suture lines, and epistaxis that cannot be controlled with anterior and posterior nasal packing. Neurologic emergencies, which include acute ischemic stroke, hemorrhagic stroke, subarachnoid hemorrhage, and hypertensive encephalopathy, can be difficult to distinguish from one another. Hypertensive encephalopathy is characterized by severe hypertensive retinopathy (i.e., retinal hemorrhages and exudates with or without papilledema) and a posterior leukoencephalopathy affecting mainly the white matter of the parieto-occipital regions as seen on cerebral MR imaging or CT scanning. A new focal neurologic deficit suggests a stroke-in-evolution, which demands a much more conservative approach to correcting the elevated BP.

In most other hypertensive emergencies, the goal of parenteral therapy is to achieve a controlled and gradual lowering of BP. A good rule of thumb is to lower the initially elevated arterial pressure by 10% in the first hour and by an additional 15% over the next 3 to 12 hours to a BP of no less than 160/110 mm Hg. BP can be reduced further over the next 48 hours. Unnecessarily rapid correction of the elevated BP to completely normal values places the patient at high risk for worsening cerebral, cardiac, and renal ischemia. In chronic hypertension, cerebral autoregulation is reset to higher-than-normal BPs. This compensatory adjustment prevents tissue overperfusion (i.e., increased intracranial pressure) at very high BPs, but it also predisposes the patient to tissue underperfusion (i.e., cerebral ischemia) when an elevated BP is lowered too quickly. In certain clinical settings, such as aortic dissection or acute pulmonary edema, more rapid reduction in BP may be required to avoid further propagation of dissection or to minimize myocardial oxygen demand and increase oxygenation. Secondary causes of hypertension should be considered in every patient who is admitted to the intensive care unit with hypertensive crisis.

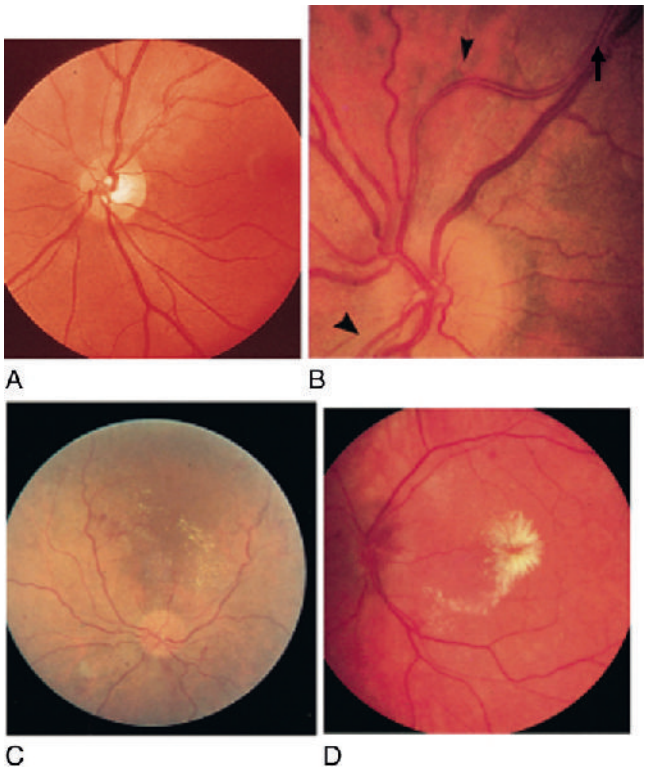

FIGURE 12-14 Hypertensive retinopathy is traditionally divided into four grades. **A,** Grade 1 shows very early and minor changes in a young patient; increased tortuosity of a retinal vessel and increased reflectiveness (silver wiring) of a retinal artery are seen at the one o'clock position in this view. Otherwise, the fundus is completely normal. **B,** Grade 2 also shows increased tortuosity and silver wiring *(arrowheads)*. In addition, nipping of the venules at arteriovenous crossings is visible *(arrow)*. **C,** Grade 3 shows the same changes as grade 2 plus flame-shaped retinal hemorrhages and soft cotton-wool exudates. **D,** In grade 4, swelling of the optic disc (papilledema) is observed, retinal edema is present, and hard exudates may collect around the fovea, producing a typical macular star. (From Forbes CD, Jackson WF: Color atlas and text of clinical medicine, ed 3, London, 2003, Mosby.)

Parenteral agents for the treatment of hypertensive emergency are summarized in Table 12-8. Intravenous labetalol (a combined α- and β-blocker) is an effective first-line drug for many hypertensive crises, particularly myocardial ischemia with preserved ventricular function. Sodium nitroprusside, a nitric oxide donor, is popular because it can be titrated rapidly to control BP. Intravenous nitroglycerin, another nitric oxide donor, is indicated mainly for hypertension in the setting of acute coronary syndrome or decompensated heart failure. Nicardipine is a parenteral dihydropyridine CCB that is particularly useful in the postoperative cardiac patient and in patients with renal failure to avoid the thiocyanate toxicity with nitroprusside. Urapadil is a new central sympatholytic agent.