surgeons a false sense of security in their ability to repair hernias without the complication of recurrence.

The reported incidence of hernia in the United States is 15 per 1,000. The actual incidence is probably substantially higher because a careful physical examination of asymptomatic persons often reveals small inguinal hernias, especially in men and premature infants. In the United States, indirect inguinal hernia accounts for about 50% of hernias. About 25% to 28% of hernias are direct inguinal, 10% to 12% ventral incisional, 6% femoral, 3% umbilical, and 3% are other hernias. Three basic factors, alone or in combination, are responsible for hernias recurring: patient constitution, such as obesity or constipation; operative technique, such as poor suture placement or a missed indirect sac; and operative complications, such as infection or hematoma. The generally reported incidence of inguinal hernia recurrence at five years is 0% to 20%.

Hernias have both economic and medical importance. They decrease productivity by causing pain, limiting motion, and sometimes causing simple or strangulated intestinal obstruction. The development of a hernia may also be associated with other medical conditions. A continued decrease in the morbidity and mortality of hernia repair is due to improvements in surgical and anesthetic techniques and to the recognition and correction or optimization of coexisting medical conditions, particularly in the elderly. Recognizing and treating factors that produce increased intraabdominal pressure or contribute to infection, before hernia repair, increase the likelihood of a successful repair.

The specific repair used for inguinal hernias varies considerably among surgeons, and each repair has its advocates who have good results with a given technique. In the United States the most commonly used repair for indirect inguinal hernia is the Bassini repair and for direct inguinal hernia are the Bassini, Cooper ligament (McVay), and the Shouldice repairs. While there is considerable variation in the reports of recurrences following the Bassini and Cooper ligament repairs, most surgeons reporting the results of the Shouldice repair have consistently reported five-year recurrence rates—0.7% to 1.3%—equal to and often superior to the best results reported for other repairs. The use of a synthetic mesh in primary inguinal hernia repairs has been recommended for the larger and more complex hernias and has a reported recurrence rate of 0% to 4% in several small series.

Outpatient inguinal hernia repair has been shown to be safe, acceptable to patients, and highly cost-effective for most patients. Patients with significant cardiac and respiratory disease, labile hypertension, and insulin-dependent diabetes mellitus or with large hernias are generally best managed as inpatients. The use of 0.5% bupivacaine hydrochloride as a long-acting local anesthetic even when general anesthesia is used appears to be a major factor in patients' acceptance of outpatient hernia surgery. Early ambulation, the avoidance of bladder distension, and adequate pain control are considered the keys to the success of outpatient hernia repair. Recurrence rates of inguinal hernias done using local anesthesia are equal to those done using general anesthesia.

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Symptomatic Carotid Artery Disease

EXTRACRANIAL CAROTID ARTERY DISEASE is associated with stroke or transient ischemic attacks by three possible mechanisms: emboli, hypoperfusion by flow reduction, and intracerebral propagation of thrombus following occlusion of the internal carotid artery. About 80% of all strokes are thromboembolic in origin. While rare conditions such as fibromuscular disease, aneurysms, and arteritis contribute to this figure, the overwhelming majority of these events are complications of atherosclerosis. A strong correlation between neurologic events and angiographically evident atherosclerotic carotid bifurcation disease has been thoroughly demonstrated. A complex plaque in this region serves as a prime source of cholesterol-fibrin or platelet-aggregate emboli from plaque ulceration. Intraplaque hemorrhage often precedes plaque ulceration. A bifurcation plaque may also cause stenosis of the proximal internal carotid artery; flow limitations will occur if the stenosis is severe (>80% of the area). Symptoms may occur if collateral flow is inadequate. Should total occlusion of the internal carotid occur, propagation of thrombus into the intracranial portion may cause symptoms days later when clot reaches the retinal or middle cerebral vessels.

Hemispheric and retinal symptoms and deficits are classic for carotid disease. Depending on the anatomic situation, however, carotid disease may also contribute to basivertebral or global hypoperfusion symptoms. The pattern of symptoms may be important, as recurrent identical hemispheric symptoms are typical of a carotid embolic source. Chronic ocular ischemia may cause various subtle visual symptoms and signs that should arouse a suspicion of carotid occlusive disease, particularly when not associated with overt retinal disease or hematologic abnormalities. The absence of a cervical bruit in a symptomatic patient should not deter one from looking for carotid occlusive disease.

The possibility of a carotid source of an ischemic event or state should prompt a minimum evaluation with a carotid echo-Doppler scan. In experienced hands, these scans are more than 90% accurate in identifying carotid stenosis and ulceration. They also provide an estimate of plaque "softness," an indicator of increased propensity for embolization. Nonhemispheric symptoms may be further evaluated by physiologic studies such as oculopneumoplethysmography. Isotopic cerebral flow studies also show promise for further clarifying low flow states. Angiography remains the mainstay of the diagnosis of carotid disease and should include intracranial views. Computed tomography (CT) and magnetic resonance imaging can be useful in defining chronic degenerative changes, the presence and location of an infarct, and the age of an infarct by the degree of CT contrast enhancement. This latter study indicates infarct maturation and when operative relief of a tight internal carotid stenosis will be less likely to cause reperfusion injury to the peri-infarct zone.

The treatment of symptomatic carotid disease continues to be controversial. Commonly lost sight of is that the goal of

treating carotid disease, medically or surgically, is to prevent stroke—the rare exceptions being where global ischemia causes disabling symptoms. Studies that focus on late survival as an end point and that do not randomly assign symptomatic patients according to the degree of carotid stenosis or ulceration are numerous. Such studies beg the question of whether there is a subset of patients who have experienced transient ischemic attacks or a limited completed stroke and who have a territorially appropriate carotid lesion and can have their chance of future stroke diminished by a particular form of therapy.

The medical treatment of cerebrovascular disease involves the control of hypertension and antiplatelet therapy. Hypertension remains a critical factor in cerebrovascular morbidity. It has been suggested that improved hypertension treatment is responsible for the decrease in the national stroke rate since 1970, but this remains unproved. Antiplatelet therapy has become a mainstay of the treatment of transient ischemic attacks despite most studies showing no stroke-preventive benefit over placebo, only decreased late mortality; 15% to 20% of patients in the "aspirin trials" have gone on to completed stroke within two years.

Scattered reports have appeared of intraluminal balloon angioplasty of carotid bifurcation atherosclerotic stenoses and internal carotid fibromuscular disease. Close monitoring and careful evaluation will be necessary before welcoming or condemning this technique.

Operative intervention for symptomatic carotid disease has been shown by numerous studies to substantially benefit patients with significant stenotic or ulcerative lesions and ipsilateral ischemic events. The stroke rate following a carotid operation would appear to be 1.0% to 1.8% per year for patients with hemispheric symptoms and lower for patients presenting with amaurosis fugax. The comparative stroke rate for patients with transient ischemia treated medically is at least 5% a year. It should be recalled that this figure includes patients with no or minimal demonstrable carotid disease and almost certainly underestimates the natural history of transient ischemic attacks in that cohort with significant carotid disease. Published perioperative stroke and mortality statistics indicate that a carotid operation can be done safely—combined stroke and death rate less than 4%—by experienced surgeons with the proper patient selection. Some series have shown unacceptable results. It is incumbent on each surgeon who does carotid surgery to approach this procedure only with the appropriate indications, to operate with the utmost respect for the potential morbidity, and to constantly review his or her own statistics.

Carotid bifurcation endarterectomy in well-selected cases will result in the relief of hemispheric symptoms with near certainty, a lower recurrent stroke rate for those patients with limited fixed deficits, and the relief of basivertebral and global symptoms in 50% to 70% of cases. The best results in this last category are obtained in patients with classic posterior circulation symptoms, a highly stenotic internal carotid artery, and angiographic posterior circulation compromise. External carotid reconstruction may help selected patients with internal carotid occlusion and definite hemispheric symptoms, particularly if the middle cerebral artery is supplied from that stenotic artery angiographically. Patients with hemispheric transient ischemic attacks and relatively normal carotid arteriograms appear to be at a lower risk for late stroke, particularly if they have had only a single event. Those with multiple events require more careful follow-up with noninvasive examinations.

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Bleeding, Clotting, and the Use of Blood Products in Trauma Care

IMMEDIATELY AFTER SERIOUS INJURY, the ability to limit bleeding from injured tissues largely determines the outcome. For trauma surgeons, there are few situations more harrowing than dealing with a seriously injured patient who has had a breakdown in hemostatic mechanisms. Various approaches designed to prevent such a loss of hemostatic control have been proposed, and some have gained considerable popularity. Whether they are scientifically correct and clinically beneficial is sometimes doubtful.

Several factors are known or suspected to contribute to a breakdown in hemostasis in an injured patient. Preexisting major abnormalities are fortunately rare and usually related to the use of therapeutic drugs. Anticoagulation with vitamin K antagonists is one of the few legitimate indications for the prophylactic use of fresh frozen plasma. Significant hepatocellular insufficiency is another. Hypothermia is one of the avoidable causes of impaired hemostasis and is probably one of the most underestimated in its importance. Hypothermia impairs various steps in the coagulation sequence and seems to have a remarkably strong effect on platelet function in vivo. Hypoperfusion is emerging as perhaps the key causal element in those few patients who have a true systemic breakdown in hemostasis. This concept has been strongly confirmed by almost all the experimental work in this area and has recently been supported by clinical data. The sooner bleeding is stopped and the blood volume restored, the less probability there is of a coagulopathy developing. This effect of sustained and severe hypoperfusion accounts, at least in part, for the association of impaired hemostasis with a massive transfusion.

Massive transfusion deserves special comment because it has become the focus of prophylactic interventions. Stored blood is variably deficient in some coagulation factors (primarily V and VIII) and is totally devoid of functioning platelets. Therefore, a dilutional or washout effect is anticipated and much feared. Various detailed studies of heavily transfused patients have not shown clinically significant coagulation component deficits, however, except in those with clinically obvious impaired hemostasis in whom the deficits are much greater than can be accounted for by dilution. Factors V and VIII are particularly well maintained because of their rapid rates of synthesis, but these are the two that are depleted in stored blood. Platelets are of a more rational concern, and the platelet count is certainly driven down by dilution in heavily transfused patients. Almost all studies in transfused patients and the pertinent studies in patients un-