HEMANGIOMAS are the most common soft-tissue tumors of infancy, occurring in approximately 5 to 10 percent of one-year-old children. Despite the frequency of these tumors, their pathogenesis is not completely understood, and the best approach to their management remains controversial. Research on angiogenesis, an improved knowledge of the classification of the vascular anomalies of infancy, advances in imaging techniques, recently identified complications, and new therapeutic approaches, such as treatment with interferon and lasers, have altered our understanding of this common childhood problem and the way we approach it.

The term hemangioma has traditionally been applied to a variety of vascular lesions, ranging from lifelong conditions to self-limited, benign tumors of infancy. The term “cavernous hemangioma” is particularly confusing, because it has been used to describe hemangiomas with subcutaneous components as well as structural anomalies of venous origin — two types of lesions that may appear similar in infancy but that differ in their growth patterns, response to treatment, and prognosis.

In 1982, Mulliken and Glowacki proposed a biologic classification of vascular birthmarks on the basis of their clinical manifestations, histopathological features, and natural history. They defined hemangiomas as vascular tumors with a growth phase, marked by endothelial proliferation and hypercellularity, and an involutional phase. They recognized that many entities referred to as hemangiomas are actually structural malformations of the vasculature, derived from capillaries, veins, lymph vessels, or arteries or from a combination of these sources. Although hemangiomas and vascular malformations do not usually occur in the same setting, they do occasionally coexist. A recent revision of Mulliken and Glowacki’s classification has broadened the category of vascular tumors of infancy to include hemangio- pericytoma, pyogenic granuloma, tufted angioma, and kaposiform hemangioendothelioma.

PATHOGENESIS

Although the precise mechanisms controlling the growth and involution of hemangiomas are not clearly understood, recent advances in our knowledge of normal vascular development and angiogenesis do provide some clues. Vasculogenesis refers to the processes by which precursors of endothelial cells give rise to blood vessels, whereas angiogenesis refers to the development of new vessels from the existing vasculature. During the proliferative phase, hemangiomas are composed of densely packed endothelial cells that form small capillaries. Cellular markers of angiogenesis, including proliferating-cell nuclear antigen, type IV collagenase, basic fibroblastic growth factor, vascular endothelial growth factor, urokinase, and E-selectin, can be identified by immunohistochemical analysis. Urinary levels of basic fibroblastic growth factor are elevated considerably in infants with hemangiomas and offer a potential means of monitoring the efficacy of treatment.

The role of vasculogenesis is less clear, but the presence of anomalous arteries in some patients with extensive hemangiomas has been attributed to developmental-field defects that occur at approximately 8 to 10 weeks of gestational age. The risk of hemangioma is 10 times as high in the children of women who underwent chorionic-villus sampling as in the children of women who did not undergo the procedure. The increased incidence of hemangiomas associated with this procedure, which on rare occasions disrupts vascular structures, suggests that errors in vasculogenesis may provide a fertile field in which hemangiomas later proliferate. Incomplete maturation of the vasculature in the embryo may also result in vascular endothelial rests that proliferate after birth more rapidly than normal blood vessels. Although this explanation is speculative, it could account for the increased incidence of hemangiomas in premature infants, since the likelihood of immature autonomous rests would be greater in these infants than in full-term infants.

The life cycle of a hemangioma differs from that of most tumors in that a hemangioma has a phase of rapid proliferation that is followed by spontaneous involution. The mechanisms that control the involution of hemangiomas are also poorly understood. Genetic techniques have been useful in showing that the regulation of angiogenesis and vasculogenesis is defective in rare hereditary vascular disorders. Moreover, there are no applicable animal models of involuting hemangiomas. In rare cases, hemangiomas are familial, and several kindreds with a presumably autosomal dominant pattern of inheritance have recently been described. The analysis of mutations in such kindreds, the development of a rep-
representative animal model, and further advances in our knowledge of angiogenesis and vasculogenesis may improve our understanding of the pathogenesis of hemangiomas.

**CLINICAL MANIFESTATIONS**

Hemangiomas are clinically heterogeneous, with their appearance dictated by the depth, location, and stage of evolution. In the newborn, hemangiomas may originate as a pale macule with thread-like telangiectases. As the tumor proliferates, it assumes its most recognizable form: a bright red, slightly elevated, noncompressible plaque. Hemangiomas that lie deeper in the skin are soft, warm masses with a slightly bluish color (Fig. 1). Frequently, hemangiomas have both superficial and deep components. Hemangiomas range from a few millimeters to several centimeters in diameter. They are usually solitary, but as many as 20 percent of affected infants have multiple lesions.

Female infants are three times as likely to have hemangiomas as male infants, and there is an increased incidence in premature infants. Approximately 55 percent of these tumors are present at birth, and the remainder develop in the first weeks of life. Classically, hemangiomas exhibit an early proliferative phase, slow involution, and, in most cases, complete resolution. In rare cases, they may appear as fully grown tumors at birth, and these congenital hemangiomas resolve rapidly, often leaving pronounced atrophic skin changes. Although the sequential behavior of hemangiomas is well known, it is difficult to predict the duration of the growth and involutional phases for an individual lesion. Superficial hemangiomas usually reach their maximal size by 6 to 8 months, but deep hemangiomas may proliferate for 12 to 14 months or, in rare cases, for as long as 2 years. The onset of involution is even more difficult to predict but is usually heralded by a change in color from bright red to purple or gray. Approximately 20 to 40 percent of patients have residual changes of the skin; hemangiomas of the tip of the nose, lip, and parotid area are particularly slow to involute, and very large, superficial hemangiomas of the face often leave disfiguring scars.

The diagnosis of hemangioma may occasionally be difficult to establish, particularly in patients with large congenital lesions and hepatic lesions. Imaging studies help distinguish vascular malformations from more aggressive neoplastic processes. Ultrasonography with Doppler studies is a cost-effective, noninvasive technique that demonstrates the high flow pattern that is characteristic of hemangiomas, thus differentiating them from solid tumors and malformations of the veins, lymph vessels, and capillaries. On a computed tomographic scan, a hemangioma appears as a homogeneous mass with large feeding vessels, with intense and persistent enhancement after the administration of contrast material. Magnetic resonance imaging of hemangiomas demonstrates well-circumscribed, densely lobulated masses, with an intermediate signal intensity on $T_1$-weighted images and a moderately hyperintense signal on $T_2$-weighted images.

**COMPLICATIONS**

Despite the benign and trivial nature of most cutaneous hemangiomas, a considerable number cause functional compromise or permanent disfigurement (Table 1). Visceral hemangiomas, particularly lesions of the liver, are associated with a much higher mortality rate than hemangiomas in other locations and therefore require meticulous monitoring.

**Ulceration**

Ulceration, the most frequent complication, can be excruciatingly painful and carries the risk of infection, hemorrhage, and scarring. Ulceration results from necrosis and typically occurs in deep, rapidly
enlarging hemangiomas but occasionally may be present at birth. Infants with ulcerated hemangiomas are often irritable, feed poorly, and are unable to sleep. Pain may precede ulceration and is probably the result of ischemia and necrosis within the hemangioma. Hemorrhage, although rare and usually inconsequential, can be very alarming to parents. In most cases, blood loss is minimal and can be controlled with direct pressure. Hemangiomas of the anogenital region are at high risk for ulceration and infection and may cause severe pain on urination or defecation (Fig. 2A and 2B). Superinfection may lead to cellulitis, osteomyelitis, or septicemia and in some cases has been lethal. Application of topical antibiotics and thin hydrocolloid dressings may be sufficient to manage small ulcers; larger ulcers may require compresses, pulsed-dye laser therapy, oral antibiotics, oral corticosteroids, or a combination of these approaches (see the section on management), as well as oral analgesics to control pain. Excisional surgery is occasionally warranted.

**Kasabach–Merritt Phenomenon**

The Kasabach–Merritt phenomenon, a complication of rapidly enlarging vascular lesions, is characterized by hemolytic anemia, thrombocytopenia, and coagulopathy. The differences between the vascular lesions that induce this disorder and classic infantile hemangiomas have been recently emphasized. The massive tumors that cause the Kasabach–Merritt phenomenon are usually deep red-blue and firm; they grow rapidly and affect male and female infants in equal numbers. These tumors tend to proliferate for a longer period (two to five years) than classic infantile hemangiomas and have a different histologic pattern. It is now clear that most patients with the Kasabach–Merritt phenomenon do not have typical hemangiomas but instead have kaposiform hemangioendotheliomas or tufted angiomas. The syndrome requires aggressive treatment (often with multiple therapies) and is associated with a high mortality rate.

**Regionally Important Lesions**

Location has a crucial role in determining the seriousness of hemangiomas. Small, slowly proliferating lesions may be problematic or even life threatening if they compromise the function of a vital structure (Table 1). Hemangiomas of the periorbital region pose considerable risk to vision and should be carefully monitored. Amblyopia may result from obstruction of the visual axis. The most common complication, astigmatism, is caused by insidious compression of the globe (Fig. 3) or extension of the tumor into the retrobulbar space (Fig. 4B). All patients with periorbicular hemangiomas should be evaluated by an ophthalmologist. Hemangiomas involving the ear may obstruct the external auditory canal, resulting in otitis or a decrease in auditory conduction, which ultimately may delay speech development.

Multiple cutaneous hemangiomas (diffuse hemangiomatosis) and large facial hemangiomas may be associated with visceral hemangiomas. Infants with numerous cutaneous hemangiomas should be observed carefully for signs and symptoms of visceral lesions, and screening abdominal ultrasonography with Doppler studies should be considered. Visceral hemangiomas are associated with much higher morbidity and mortality rates (40 to 80 percent), because lesions with a high flow pattern, such as those in the liver, may cause high-output cardiac failure and anemia. Hemangiomas of the liver that are not associated with cutaneous lesions often pose a diagnostic problem. They must be differentiated from arterial or venous malformations, because they differ with respect to both prognosis and treatment. Visceral hemangiomas, like their cutaneous counterparts, grow rapidly during infancy, and if the child survives they regress spontaneously during early childhood, whereas vascular malformations progress slowly and do not resolve. Hemangiomas, the most common hepatic vascular lesions in children, are usually multiple and involve both lobes of the liver.

---

**Table 1. Dysmorphic Features of Hemangiomas, Complications, and Regionally Important Lesions.**

<table>
<thead>
<tr>
<th>Dysmorphic features</th>
<th>Complications</th>
<th>Regionally important lesions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large facial hemangiomas*</td>
<td>Ulceration</td>
<td>Multiple cutaneous lesions — markers of visceral hemangiomas</td>
</tr>
<tr>
<td>Posterior fossa malformations</td>
<td>Infecion</td>
<td>Lesions in a beard distribution — markers of airway hemangiomas†</td>
</tr>
<tr>
<td>Hemangiomas of the cervicofacial region</td>
<td>Hemorrhage</td>
<td>Lesions that occlude vital structures</td>
</tr>
<tr>
<td>Arterial anomalies</td>
<td>High-output cardiac failure</td>
<td>Lesions in a beard distribution — markers of airway hemangiomas†</td>
</tr>
<tr>
<td>Cardiac anomalies</td>
<td></td>
<td>Regionally Important Lesions</td>
</tr>
<tr>
<td>Eye anomalies</td>
<td></td>
<td>High-output cardiac failure</td>
</tr>
<tr>
<td>Sternal or abdominal clefting or ectopia cordis</td>
<td></td>
<td>Ulceration</td>
</tr>
<tr>
<td>Lumbosacral hemangiomas</td>
<td></td>
<td>Infection</td>
</tr>
<tr>
<td>Spinal dysraphism</td>
<td></td>
<td>Hemorrhage</td>
</tr>
<tr>
<td>Anogenital anomalies</td>
<td></td>
<td>Lesions associated with slow involution or scarring</td>
</tr>
<tr>
<td>Urogenital anomalies</td>
<td></td>
<td>Parotid lesions</td>
</tr>
<tr>
<td>Lip lesions</td>
<td></td>
<td>Lesions on the tip of the nose</td>
</tr>
<tr>
<td>Lesions on the tip of the nose</td>
<td></td>
<td>Lip lesions</td>
</tr>
<tr>
<td>Anogenital lesions</td>
<td></td>
<td>Lesions on the tip of the nose</td>
</tr>
</tbody>
</table>

*We refer to the abnormalities as the PHACES syndrome.
†The beard distribution refers to the chin, lips, mandibular region, and neck.

---

The differences between the vascular lesions that induce this disorder and classic infantile hemangiomas have been recently emphasized. The massive tumors that cause the Kasabach–Merritt phenomenon are usually deep red-blue and firm; they grow rapidly and affect male and female infants in equal numbers. These tumors tend to proliferate for a longer period (two to five years) than classic infantile hemangiomas and have a different histologic pattern. It is now clear that most patients with the Kasabach–Merritt phenomenon do not have typical hemangiomas but instead have kaposiform hemangioendotheliomas or tufted angiomas. The syndrome requires aggressive treatment (often with multiple therapies) and is associated with a high mortality rate.

**Regionally Important Lesions**

Location has a crucial role in determining the seriousness of hemangiomas. Small, slowly proliferating lesions may be problematic or even life threatening if they compromise the function of a vital structure (Table 1). Hemangiomas of the periorbital region pose considerable risk to vision and should be carefully monitored. Amblyopia may result from obstruction of the visual axis. The most common complication, astigmatism, is caused by insidious compression of the globe (Fig. 3) or extension of the tumor into the retrobulbar space (Fig. 4B). All patients with periorbicular hemangiomas should be evaluated by an ophthalmologist. Hemangiomas involving the ear may obstruct the external auditory canal, resulting in otitis or a decrease in auditory conduction, which ultimately may delay speech development.

Multiple cutaneous hemangiomas (diffuse hemangiomatosis) and large facial hemangiomas may be associated with visceral hemangiomas. Infants with numerous cutaneous hemangiomas should be observed carefully for signs and symptoms of visceral lesions, and screening abdominal ultrasonography with Doppler studies should be considered. Visceral hemangiomas are associated with much higher morbidity and mortality rates (40 to 80 percent), because lesions with a high flow pattern, such as those in the liver, may cause high-output cardiac failure and anemia. Hemangiomas of the liver that are not associated with cutaneous lesions often pose a diagnostic problem. They must be differentiated from arterial or venous malformations, because they differ with respect to both prognosis and treatment. Visceral hemangiomas, like their cutaneous counterparts, grow rapidly during infancy, and if the child survives they regress spontaneously during early childhood, whereas vascular malformations progress slowly and do not resolve. Hemangiomas, the most common hepatic vascular lesions in children, are usually multiple and involve both lobes of the liver.
apeutic approaches are often required for the successful treatment of such hemangiomas.

Hemangiomas may occur in any organ system, and a variety of complications requiring aggressive intervention has been reported. Hemangiomas of the airway may be life threatening, given their potential for proliferation with subsequent obstruction. Subglottic hemangiomas are manifested by hoarseness and stridor. These lesions may progress rapidly to respiratory failure, often occurring when the infant is 6 to 12 weeks old. Approximately 50 percent of such infants have associated cutaneous hemangiomas; thus, noisy breathing in an infant with a cutaneous hemangioma warrants direct visualization of the airway. Infants with cutaneous hemangiomas involving the chin, lips, mandibular region, and neck (Fig. 4B and 4C) are at greatest risk for airway involvement. Asymptomatic infants with extensive hemangiomas in this region of the face and neck (“beard” distribution) should be carefully monitored, since symp-

Figure 2. Anogenital Hemangioma with Painful, Superinfected Ulceration.
The hemangioma (Panel A) began to heal after treatment with a pulsed-dye laser (Panel B). There will be marked residual scarring from the ulceration.
Figure 3. Small Periocular Hemangioma in an Infant That Resulted in Astigmatism.

Figure 4. Large, Superficial Hemangioma of the Face, Parotid Area, and Anterior Neck. Panel A shows the infant at four weeks of age. A tracheostomy was subsequently required because of occlusion of the airway due to a subglottic hemangioma. A T₂-weighted magnetic resonance image (Panel B) shows expansion of the hemangioma into the left retrobulbar space. By eight months of age, the infant had considerable regression of the cutaneous and ocular hemangioma but still required a tracheostomy because of subglottic involvement (Panel C).
tomatic hemangiomas of the airways will develop in 60 percent of these patients.\textsuperscript{32,33}

ASSOCIATION WITH DYSMORPHIC FEATURES

Historically, certain dysmorphic conditions have been associated with vascular lesions; according to the diagnostic criteria of Mulliken and Glowacki, the majority of these conditions should be classified as vascular malformations (port-wine stains and venous or lymphatic malformations) rather than as hemangiomas.\textsuperscript{34} There are at least two instances, however, in which true hemangiomas appear to be an integral component of abnormal morphogenesis (Table 1).

Extensive hemangiomas of the neck and face (Fig. 4A) may be associated with multiple anomalies, including what we refer to as the PHACES syndrome (posterior fossa malformations, hemangiomas of the cervicofacial region, arterial anomalies, cardiac anomalies, eye anomalies, and sternal or abdominal clefting or ectopia cordis) (Table 1).\textsuperscript{6,8,35-38} This syndrome has a marked predominance among female infants (ratio of affected girls to affected boys, 9:1) and is thought to represent a developmental-field defect that occurs between 8 and 10 weeks of gestation.\textsuperscript{6,8,39} Recent observations suggest that infants with these associated arterial anomalies are at risk for cerebrovascular occlusive disease.\textsuperscript{8,40} Burrows et al. described eight infants with hemangiomas of the neck and face and congenital arterial anomalies: seven had neurologic abnormalities, and four had radiologic evidence and neurologic sequelae of acute cerebrovascular infarction due to acquired occlusion of the anomalous arteries.\textsuperscript{40}

Hemangiomas of the lumbosacral region may be markers for occult spinal malformations and anomalies of the anorectal and urogenital regions.\textsuperscript{41,42} These hemangiomas are superficial, span the midline, and often have a central membranous defect (Fig. 5A). Imaging of the spine is indicated for all patients with midline hemangiomas in this region (Fig. 5B).

MANAGEMENT

The management of hemangiomas continues to be a subject of considerable controversy. Irradiation was used in the 1940s and 1950s, and many experts subsequently decried the use of aggressive treatment for these self-limiting tumors. Studies demonstrated that the results of irradiation or excisional surgery were worse than the consequences of leaving the tumors untreated. Nevertheless, the development of newer therapeutic options, including lasers, corticosteroids administered systemically and directly to the lesion, and interferon alfa, and the prospect of new inhibitors of angiogenesis have led many investigators to question the wisdom of a uniformly hands-off approach. Complications of hemangiomas of the periocular region, airway, and liver can impair function and even be life threatening. Although the choice of treatment may vary, the decision whether to treat in these cases is usually straightforward.

The management of hemangiomas that may cause permanent disfigurement is more problematic. Accurate prediction of which hemangiomas are likely to cause permanent damage is a formidable task, and the selection of appropriate treatment (if any) depends on several factors. These factors include the location of the hemangioma, the depth of the lesion within the skin, the age of the patient, the presence or likelihood of complications, the availability of certain treatments (such as laser therapy), the expertise of the treating physician, and parental preference. The opinions of 21 physicians, including pediatricians, dermatologists, and surgeons with special expertise in managing hemangiomas, were recently published as a symposium.\textsuperscript{43} Although there was some diversity in the views expressed, most of these physicians agreed that large hemangiomas of the face with prominent dermal components, particularly those involving the lips, nose, ears, and glabella, pose the greatest risk of scarring.\textsuperscript{17,43} Decisions about treatment must take into account the age of the patient at the time of the evaluation. A faint, minimally elevated hemangioma of the face in a 2-week-old infant may have a completely different course from an identical lesion in a 12-month-old child, since the potential for growth is much greater in the very young infant. Because it is difficult (if not impossible) to predict the ultimate size, duration of growth, and rate of involution of such tumors, very young infants need to be monitored frequently.

Treatment Options

The effect of a hemangioma, particularly a facial hemangioma, on the parents of an affected child should not be underestimated.\textsuperscript{44,45} A recent study of patients with facial hemangiomas larger than 1 cm in diameter found that parental reactions of disbelief, fear, and mourning were common and were similar in intensity to parental reactions to permanent malformations.\textsuperscript{46} Parents reported being barraged with comments by strangers, including accusations of child abuse, and more than half the parents expressed dissatisfaction with aspects of their children’s medical care. Even (perhaps especially) in cases in which no specific treatment is undertaken, active emotional support of the parents is important. Showing parents photographs of hemangiomas before and after spontaneous involution can be immensely reassuring. Photographing the hemangioma and comparing the photographs on subsequent visits can also be helpful in demonstrating the gradual involution of the lesion.

Systemic corticosteroids have become a mainstay in the treatment of hemangiomas, yet their mechanism of action is not well understood.\textsuperscript{43,45,47-49} Daily
doses of 2 to 3 mg of prednisolone or prednisone per kilogram of body weight are usually given, and some investigators have recommended even higher doses (5 mg per kilogram daily). This treatment results in dramatic shrinkage of the hemangioma, usually within days, in approximately one third of infants, stabilization of growth without measurable shrinkage in another third, and a minimal response or none in one third. Despite many potential side effects, including irritability, gastrointestinal upset, immunosuppression, hypertension, and growth retardation, most treated infants do well, and catch-up growth occurs after the cessation of therapy. The duration of treatment ranges from a few weeks to many months, depending on the age of the child, the indications for treatment, and the growth characteristics of the hemangioma, since rapid tapering of the dose of corticosteroids while the tumor is still in the proliferative phase may result in rebound growth. High doses of intravenous methylprednisolone have been used to treat life-threatening hemangiomas and the Kasabach–Merritt phenomenon.

Corticosteroids administered directly to the lesion can also be effective in cases of small, localized, cutaneous hemangiomas. One or two additional injections are often necessary after the initial injection. Doses of triamcinolone should not exceed 3 to 5 mg per kilogram per treatment session. Treatment with potent topical corticosteroids resulted in improvement in one small series of patients, but to our knowledge, there are no other data on the efficacy of this treatment. Treatment in the periocular region is contraindicated, since it is fraught with complications. Atrophy of the skin, necrosis, and occlusion of the central retinal artery, with resultant blindness, have been reported in patients treated with intralesional corticosteroids.

Recombinant interferon alfa, an inhibitor of angiogenesis, has been used successfully in the treatment of life-threatening hemangiomas that failed to respond to corticosteroid therapy. Both interferon alfa-2a and interferon alfa-2b have been used, usually administered as a subcutaneous injection of 3 million units per square meter of body-surface area per day. The interval between the administration of interferon alfa and the response to treatment ranges from a few weeks to several months. Common side effects include irritability, neutropenia, and abnormalities of liver enzymes. A particularly worrisome side effect, spastic diplegia, has recently been reported in as many as 20 percent of patients. This potentially irreversible side effect can occur with both types of interferon alfa, and its mechanism is unknown. Interferon alfa should therefore be reserved for infants with life-threatening hemangiomas in whom high-dose corticosteroid therapy has failed, and when it is administered, neurologic status should be monitored closely.

**Figure 5.** Large Lumbosacral Hemangioma with Ulceration. The hemangioma is shown in Panel A. Magnetic resonance imaging of the spine revealed an occult lipomeningocele and a tethered spinal cord (Panel B).
Several laser systems have been used to treat hemangiomas. The flash-lamp–pumped pulsed-dye laser, though extremely effective in treating port-wine stains, is less effective for hemangiomas. Because of the limited depth of penetration (approximately 1 mm), this laser works better for thin superficial hemangiomas than for those that are destined to be both superficial and deep. It can improve residual telangiectases after involution and is effective in treating ulcerated hemangiomas, resulting in decreased pain and prompt recanalization. Continuous wave lasers, such as argon, neodymium:yttrium–aluminum–garnet, and potassium–titanyl–phosphate lasers, have also been used, but their efficacy is more dependent on the ability of the operator and they are associated with a higher risk of scarring. Optimal laser-delivery systems and methods specific for the treatment of hemangiomas have yet to be defined.

The management of subglottic hemangiomas deserves particular consideration, since these lesions can progress rapidly to respiratory failure and the optimal therapeutic approach remains controversial. Systemic corticosteroids continue to be the first line of therapy for subglottic hemangiomas; however, tracheotomy is often required to maintain the airway. Some institutions advocate tracheotomy without pharmacologic intervention, because subglottic hemangiomas, like their cutaneous counterparts, regress spontaneously. Interferon alfa has a limited role in the treatment of subglottic hemangiomas, since the response time is slow. Ablation with a carbon dioxide laser, although useful for small, solitary mucosal lesions, may result in scarring and laryngeal stenosis, particularly in cases of large, circumferential lesions of the subglottic space.

Cryotherapy, which is popular in some countries in Europe and South America, has been reported to have favorable results with superficial hemangiomas, but concern about potential scarring has limited its use in North America. In rare cases, embolization has been used to treat cutaneous hemangiomas that have not responded to medical therapy. Bleomycin injected directly into the lesion has been reported to be effective, but its role in the treatment of hemangiomas is not yet established. Surgical excision is used most frequently to repair residual cosmetic deformities, but early excisional surgery is a reasonable option in some cases, such as for pedunculated hemangiomas that are almost certain to result in residual abnormalities and hemangiomas that are life-threatening or impair function and for which pharmacologic therapy is not effective or well tolerated. In cases without medical complications, but for which there is uncertainty about the outcome, the benefits and risks of a surgical approach must be weighed carefully, since the scar may be worse than the results of spontaneous regression. We generally recommend a reevaluation when the child is about four years old to assess the extent of residual hemangioma, and we consider surgery at that point if the hemangioma is involving very slowly and if surgery is likely to have a good result.

CONCLUSIONS

Hemangiomas are common, yet many questions about their pathogenesis and optimal management remain unanswered. Better recognition and treatment of complications and newer treatments, such as laser therapy and the use of pharmacologic inhibitors of angiogenesis, may help in cases in which the results of spontaneous resolution are poor. Although most hemangiomas are best left untreated, the large number of exceptions dictates that clinicians observe infants with hemangiomas carefully and tailor their approach to the specific characteristics of each case.

REFERENCES