Acute and chronic ischemia of the hand: Pathophysiology, treatment, and prognosis

Fifty consecutive patients with acute and chronic ischemia of the hand were investigated by Allen testing, Doppler ultrasound, digital plethysmography, and angiography over a 4-year period. The pathophysiologic mechanism responsible for the ischemia was determined to be emboli in 6%, vasospasm in 10%, thrombosis or “sludging” in 28%, occlusive disease in 26%, and occlusive disease associated with either vasospasm or external compression in 30%. Ten patients required emergency medical treatment with intraarterial streptokinase, intravenous heparin, or dextran 40 and continuous stellate ganglion blocks, and three patients required emergency microsurgical revascularization because of radial artery thrombosis. Patients with chronic ischemia of the hand were maintained on nifedipine, 30 to 60 mg daily, and pentoxifylline, 1200 mg daily. Seven patients underwent digital sympathectomy and 14 patients underwent microsurgical revascularization as prophylactic procedures for chronic digital ischemia. Amputations were required in 18 patients because of end-stage gangrene. Long-term follow-up revealed a 20% incidence of recurrent digital ulcerations. There have been six deaths during follow-up, five of them due to myocardial infarction; this reflects the underlying systemic arteriopathy in many of these patients. (J HAND SURG 1991:16A:1074-83.)

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Patients with symptoms and signs of ischemia of the hand may be treated by general physicians, rheumatologists, general surgeons, and vascular surgeons as well as by specialist hand surgeons. This may explain why the subject is addressed relatively infrequently in the hand surgery literature. Ischemia due to occlusive disease of the proximal subclavian and axillary arteries or to emboli lodging at the bifurcation of the brachial artery is traditionally treated by general surgeons and vascular surgeons. Raynaud’s phenomenon and digital ulceration associated with the connective tissue disorders are usually managed by rheumatologists and general physicians. In addition to the investigation and diagnosis of these patients, hand surgeons may also be involved in the treatment of emboli and occlusive disease of the distal radial and ulnar arteries and incipient and frank gangrene of the digits.

Long-term follow-up of patients with ischemia of the hand is also poorly documented in the literature. Although amputation remains the standard surgical treatment for patients with end-stage gangrene, the prognosis for patients with severe Raynaud’s phenomenon, digital ulceration, or incipient gangrene may potentially be improved by prophylactic surgical intervention in the form of digital sympathectomy or microsurgical revascularization. The indications for such techniques remain imprecise, however, and no long-term follow-up of patients undergoing such surgery has been reported.

Materials and methods

Fifty consecutive patients with acute and chronic ischemia of the hand were evaluated over a 4-year period. Forty-four patients were followed up prospectively after systematic investigation by a standardized protocol, and 6 patients were reviewed retrospectively. Patients were referred either directly or secondarily after initial diagnostic work-up had been initiated by a general physician, a rheumatologist, or a vascular surgeon.
Table I. Pulse volume recordings suggestive of occlusion of both the radial and the ulnar arteries in a patient with connective tissue disease and progressive gangrene of the left index and long fingers.

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<tr>
<th>Finger</th>
<th>Pulse volume</th>
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<td>Left (mm Hg)</td>
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<tr>
<td>Thumb</td>
<td>34</td>
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<tr>
<td>Index</td>
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<td>Long</td>
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There were 27 female and 23 male patients, with an age range from 9 years to 74 years. Ischemia occurred unilaterally in 32 patients and bilaterally in 18 patients. Symptoms of ischemia were acute in 20 patients but were chronic in 30 patients. Characteristic symptoms of ischemia, including cold intolerance, Raynaud’s phenomenon, and rest pain, were elicited from the patient’s history, together with any associated factors such as coronary artery or peripheral vascular disease, renal disease, diabetes, connective tissue disorders, and smoking.

Systematic examination of the vascular system of the entire involved upper extremity included palpation of the brachial, radial, and ulnar arterial pulses and auscultation of the supraclavicular fossa for bruits in the subclavian artery. Thoracic outlet syndrome was tested by Adson’s and Wright’s maneuvers. Allen testing, which involved comparison with the uninvolved hand, allowed rapid determination of the patency of arterial inflow into the hand through the radial and ulnar arteries. The pencil Doppler probe was used to assess the patency of the radial and ulnar arteries at the wrist, the superficial palmar arch, the dorsal branch of the radial artery in the first web space, and the radial and ulnar digital arteries in each digit. Areas of cyanosis, ulceration, or gangrene in the hand were documented with photographs. Plain x-ray films were obtained, especially in those patients with ulceration or gangrene, to exclude osteomyelitis.

Rheumatologic consultations were obtained in six patients who had been referred directly to the hand surgery service. Erythrocyte sedimentation rate, rheumatoid factor, antinuclear antibody, anticitrullinated antibody, and serum protein electrophoresis were determined in patients suspected of having connective tissue disorders. Cardiologic consultation and cardiac ultrasound studies were obtained for three patients found to have an irregular pulse rate.

The 30 patients with symptoms of chronic ischemia of the hand were investigated further with digital plethysmography (16 cases) and cold stress testing (9 cases) in our vascular laboratory and with intraarterial digital subtraction angiography (22 cases) by a vascular radiologist. As a prelude to plethysmography, segmental blood pressures were measured with the pencil Doppler probe over the brachial artery at the elbow and over the radial and ulnar arteries at the wrist. A gradient of greater than 20 mm Hg between the elbow and the wrist or a 20 mm Hg difference between the ischemic hand and the contralateral uninvolved extremity were used as the criteria for significant stenosis of the distal brachial artery or radial and ulnar arteries in the forearm. Digital plethysmography provided an absolute blood pressure and waveform analysis for each digit. A difference of 15 mm Hg between adjacent fingers or a gradient of greater than 30 mm Hg between the radial and ulnar blood pressures and the digital blood pressure were used.
Magnification provided better definition of the common digital and proper digital arteries. If vasospasm was suspected of being a component of the patient’s symptoms, angiography was performed with the use of axillary block anesthesia. Vasospasm was also prevented by intraarterial injection of 25 mg tolazoline immediately before injection of the contrast medium. Patients with the following indications were investigated angiographically: (1) unilateral Raynaud’s phenomenon; (2) progressive digital ulceration or gangrene in spite of good medical treatment; (3) recurrent digital ulceration; and (4) Doppler evidence of occlusion of the distal radial or ulnar arteries.

Finally, in addition to the specific cause, an attempt was made to determine the pathophysiologic mechanism responsible for producing the ischemia. This was based on the patient’s history and physical examination, as well as other investigations.

The 20 patients with acute ischemia of the hand were immediately investigated by means of Doppler examination of the distal radial and ulnar arteries, the superficial palmar arch, and the radial and ulnar digital arteries in each digit and, if necessary, by angiography (14 cases). Ten patients required emergency medical treatment with intraarterial streptokinase, intravenous heparin, or dextran 40 and continuous stellate ganlion blocks. If angiography had documented emboli lodging in the distal radial or ulnar arteries, a fine cannula was introduced immediately proximal to the site of the embolus and 100,000 units of streptokinase was infused over 30 minutes as a test dose, followed by continuous infusion of 5,000 units of streptokinase per hour with titration of the serum fibrinogen level. Angiography was usually repeated 24 or 48 hours later to determine whether there had been any improvement in visualization of the distal arterial tree. Low-molecular-weight dextran 40 was infused continuously at 25 ml per hour for 5 days. Emergency surgical intervention was performed in three patients who developed acute ischemia of the thumb and index finger after radial artery cannulation. It proved impossible to remove the thrombus adequately, even with the smallest Fogarty catheter, since damaged endothelium was left behind to act as a focus for further thrombosis. Consequently, arterial inflow was restored by resection of the involved segment of the radial artery through the first web space until normal-appearing intima was seen under the operating microscope. The segment was then replaced with a reversed interposition vein graft by standard microsurgical techniques (Fig. 3, B).

Patients with symptoms of chronic ischemia of the hand were advised to give up smoking and to avoid exposure to cold, including refrigerators and air-conditioning.
Fig. 3. A, Angiogram of a patient with atherosclerosis and ischemia of the thumb and index finger showing occlusion of the radial artery. B, Arterial inflow to the hand was improved by resection of the occluded segment of radial artery and interposition vein grafting from the distal radial artery to the origin of the arteria princeps pollicis.

out to the level of the web spaces (Fig. 2). If ischemia was localized more specifically to one digit, the radial and ulnar digital arteries were stripped out to the level of the proximal interphalangeal joint of that digit by extending the inverted J-shaped incision in the palm into a Brunner incision over the proximal phalanx.

Finally, patients who had unremitting rest pain, Raynaud’s phenomenon, or multiple recurrent digital ulcerations that could not be controlled with medical therapy, and who had angiographic evidence of occlusion of the distal radial or ulnar arteries, were considered for resection of the occluded arterial segments and reconstruction of a new “palmar arch” with reversed interposition vein grafts by standard microsurgical techniques (Figs. 3 and 4). The interposition vein graft was anastomosed end to end to the distal radial or ulnar artery and the common digital arteries were anastomosed end to side to the graft itself.

**Results**

A specific etiologic factor could be determined in 49 patients, with the predominant diagnoses being connective tissue disease (38%), atherosclerosis (18%), and renal vascular disease (16%) (Table II). Angiograms were performed in 30 patients (60%) and pulse
Fig. 4. A, Distal tip ulcerations of the left index, long, ring, and small fingers in a patient with chronic renal disease and scleroderma. B, Angiogram shows occlusion of both the distal radial and ulnar arteries but reconstitution of a superficial palmar arch. C, After resection of the occluded distal ulnar artery and superficial palmar arch, the common digital arteries to the second, third, and fourth web spaces were anastomosed end to side to an interposition vein graft, which was anastomosed end to end to the distal ulnar artery to reconstruct a new "palmar arch." D and E, Postoperative pulse volume recordings confirmed both improved wave forms (Table V) and increased digital pressures, and this correlated with clinical signs of healing of the tip ulcerations.

volume recordings in 27 patients (54%). Together these two investigations confirmed more frequent involvement of the ulnar artery, superficial palmar arch, and proper digital arteries compared with relative sparing of the radial artery and common digital arteries. On the basis of the patient's history, clinical examination, and results of the angiogram and pulse volume recordings, a pathophysiologic mechanism responsible for the ischemia could be inferred. Emboli were considered to be the cause in 6%, thrombosis or sludging in low flow states in 28%, occlusive disease alone in 26%, and in combination with vasospasm or external compression in 30%, and pure vasospasm only in 10% of patients (Table III).
Emboli, radial artery thrombosis after cannulation, intraarterial drug injections, meningococcal septicemia, and low flow states in acutely ill patients, especially those receiving dopamine infusions, were the predominant causes of acute ischemia affecting the hand. Emboli lodging at the bifurcation of the brachial artery are traditionally managed with embolectomy by the vascular surgeon, and one such patient seen by the hand surgery service was referred to a vascular surgeon for embolectomy. Three patients with incomplete arches had acute ischemia of the thumb and index finger after radial artery cannulation and were successfully treated by resection of the thrombosed segment and revascularization with a reversed interposition vein graft. In two patients with acute ischemia due to intraarterial drug injections, extensive gangrene of the digits developed despite intraarterial streptokinase given within 12 hours of the injection. Streptokinase was beneficial in only one patient, in whom an embolus from an atherosclerotic plaque in the subclavian artery lodged in the distal radial artery. Follow-up angiograms revealed improved visualization of the digital arteries to the thumb and the radial digital artery to the index finger but incomplete lysis of the embolus. In two children with meningococcal septicemia fulminant digital gangrene developed presumably as a result of disseminated intravascular coagulation. Intravenous heparin was ineffective in both cases.

Thirty-nine patients (78%) required surgical intervention at some stage in the evolution of their ischemia. Amputations alone were required in 18 patients; only three of these experienced delayed healing of amputation stumps, usually because of hypoproteinemia or high steroid dosage. Since the incidence of vasospasm as the sole mechanism causing ischemia has been quite low (10%), only seven patients have undergone digital sympathectomy. Elective microsurgical revascularization of the distal radial and ulnar arteries and superficial palmar arch was performed in 14 patients—six patients with connective tissue disorders, three with atherosclerosis, three with renal vascular disease, and two with vibration-induced ulnar artery thrombosis.

Follow-up has ranged from 1 year 4 months to 5 years 4 months. Relief of hand pain, decreased frequency of Raynaud’s phenomenon, and healing of dig-
ital ulcers have been used as criteria for evaluation of the efficacy of medical and surgical treatment. Improvement in digital blood flow has been documented objectively by postoperative pulse volume recordings and by comparison with preoperative values (Tables IV and V and Fig. 4, E), and the patency of vein grafts has been confirmed by pencil Doppler examination. In short-term follow-up at 1 year, the majority of patients obtained subjective relief of their pain, with healing of digital ulcers, after amputations, digital sympathectomy, or microsurgical revascularization. In longer follow-up, however, recurrent digital ulcerations have developed in 10 patients (20%), predominantly patients with connective tissue disease and the combination of renal vascular disease and diabetes. Among the patients undergoing microsurgical revascularization, thrombosis of the vein graft has been documented thus far in five patients, corresponding to a patency rate of 65%. There have been six deaths (12%) during the follow-up period, five of them due to myocardial infarction.

Discussion

Although there are multiple causes of ischemia of the hand, a classification based on the underlying physiologic mechanism responsible for the ischemia would seem to be appropriate as a rationale for specific treatment. The pathologic processes that produce ischemia in any end organ—and the hand can be considered such an end organ—are emboli, “sludging” of blood in low flow states, thrombosis, external compression, intimal proliferation progressing to occlusion (atherosclerosis), and vasospasm. A tentative mechanism can be inferred from consideration of the patient’s history, clinical presentation, and investigations, but obviously the precise mechanism can be confirmed only at the time of surgical exploration or by histologic examination of the involved artery.

Large emboli from the heart due to atrial fibrillation or myocardial infarction lodging at the bifurcation of the brachial artery are traditionally managed with embolectomy by the vascular surgeon. However, smaller emboli or a shower of microemboli dislodged from an ulcerated atherosclerotic plaque in the subclavian artery may lodge in the distal radial or ulnar arteries or appear as digital tip ulcerations or gangrene and therefore fall within the domain of the hand surgeon. Localized thrombosis may be precipitated by direct endothelial injury to the brachial artery during cardiac catheterization or to the radial artery during monitoring of arterial pressure. Local thrombosis of the distal ulnar artery through Guyon’s canal as a result of repetitive blunt trauma together with a component of external compression constitutes the hypothenar hammer syndrome.

Sludging of blood in low flow states, which may progress to global thrombosis throughout the entire arterial system of the hand, may account for the ischemia seen after intraarterial drug injections, polycythemia, meningococcal septicemia, and malignant disease. Intimal proliferation resulting in progressive narrowing of the internal lumen of the common and proper digital arteries would seem to be the mechanism responsible for producing focal stenoses and segmental occlusions of these arteries in patients with connective tissue disorders, atherosclerosis, and renal vascular disease, but vasospasm and external compression by periadventitial fibrosis may also play an associated role. It has been our impression, however, that the ischemic conditions in these patients may deteriorate acutely as a result of secondary thrombosis of the previously narrowed distal radial or ulnar arteries or of the superficial palmar arch. Whether Raynaud’s phenomenon is due to pure sympathetic overactivity producing vasoconstriction of the digital arteries or whether it is due to underlying occlusive disease of the digital arteries remains controversial.

Whatever the mechanism, patients with incomplete palmar arches (22% in Coleman and Anson’s study) are more at risk if one of their inflow arterial systems becomes compromised by any of these pathophysiologic mechanisms. Ischemia may continue to progress by extension of thrombosis into the common digital arteries, by seeding of microemboli into the distal digital arteries, or by secondary vasospasm of the remaining collateral circulation because of the increased sympathetic discharge from a thrombosed distal radial or ulnar artery.

In our experience, the pencil Doppler probe has been the simplest, most informative technique for investigation of patients with acute and chronic ischemia of the hand, allowing precise determination of the patency of the distal radial and ulnar arteries, the superficial arch, and the proper digital arteries in each digit. Doppler evidence of occlusion of either the distal radial or the ulnar artery has become one of our criteria for proceeding to angiography. Whereas digital plethysmography can diagnose occlusive disease in the hand, the most significant benefits of pulse volume recordings are that they allow a noninvasive, objective comparison of preoperative and postoperative perfusion in the digits after various treatment modalities (Tables IV and V). Wilgis has combined the technique of pulse volume recordings with cold stress testing to evaluate the contribution of vasospasm in arterial insufficiency.
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Painful overactivity can be designated as the primary mechanism producing ischemia only in those patients in whom the decrease in absolute blood pressure and flattening of the pulse volume wave form after cold stress testing can be reversed by local anesthetic block in the distal palm. However, the majority of our patients with connective tissue disorders or renal vascular disease have complained that cold immersion is extremely painful, and, even if they were able to complete preoperative testing, they have been very reluctant to undergo postoperative comparison testing. Angiography has proved to be the definitive technique in this series for delineation of the site of involvement by emboli, thrombosis, or occlusive disease. Allergic reactions have not occurred with the newer contrast agents and computerized digital subtraction techniques, which require only small volumes to be injected. The entire arterial system from the arch of the aorta to the hand should be scanned to exclude proximal stenosis or a source of emboli in the subclavian artery. Absence of the common digital arteries to the second, third, and fourth web spaces is indicative of occlusive disease of the superficial palmar arch. However, normal visualization of the superficial palmar arch and the common digital arteries after injection of tolazoline implies that vasospasm may be the predominant cause of the patient’s ischemia. Angiography is not necessary in every patient but should be strongly considered in the circumstances previously described.

Immediate diagnosis is crucial in patients with acute ischemia of the hand, and this has been accomplished most expeditiously with the pencil Doppler probe and emergency angiography. Because of the concomitant endothelial injury in cannulation injuries of the radial artery, we would advocate immediate resection of the involved segment and reconstruction with an interposition vein graft by standard microsurgical techniques (Fig. 3, B) rather than ineffective removal of the thrombus with small Fogarty catheters. However, cannulation injuries of the radial artery should be largely preventable with preoperative Allen testing, prophylactic aspirin, constant irrigation of the catheter, and early removal of the catheter. Patients with incomplete arches are at greatest risk and can be identified preoperatively by pencil Doppler testing of the ulnar digital artery to the thumb with temporary occlusion of the radial artery. Whereas Jalalian et al. have successfully used intrarterial infusion of streptokinase to salvage patients with acute ischemia due to drug injections, we have been unsuccessful in preventing the inevitable progression to digital gangrene in two patients in this series who were treated with streptokinase within 12 hours of the drug injection. Treatment of patients with acute ischemia of the hand due to low flow states, especially those receiving dopamine infusions, continues to be a frustrating problem.

The most effective pharmacologic agents for the conservative management of chronic symptoms of Raynaud’s phenomenon and digital ulceration have been the calcium channel blockers, nifedipine, and the rheologic agent, pentoxifylline. Formal surgical amputation is obviously a salvage procedure that will relieve pain and remove the areas affected by ulceration or gangrene. However, interest now focuses on whether prophylactic procedures, such as digital sympathectomy and microsurgical revascularization, will increase digital blood flow and thereby relieve symptoms of ischemic pain, heal ulcers, and prevent the progression to frank gangrene.

Flatt first proposed that sympathetic vasoconstrictive control of the digital arteries could be interrupted by stripping the adventitia from the common digital arteries in the palm. Wilgis further emphasized that only those patients who demonstrate improvement in pulse volume recordings by local anesthetic block before cold stress testing are potential candidates for digital sympathectomy. However, the incidence of vasospasm as the sole pathophysiologic mechanism producing ischemia in this series has been surprisingly quite low. On the basis of surgical exploration of the superficial palmar arch system of patients with various connective tissue diseases in this series, it would appear that three different components may be contributing to their ischemia: (1) an exaggerated vasoconstrictive response, (2) periadventitial fibrosis producing external compression of the digital arteries, and (3) intimal proliferation within the digital arteries. Our technique of digital sympathectomy has therefore evolved into a much more extensive technique than that originally advocated by Flatt, Wilgis, and Egloff et al. Patients with scleroderma, mixed connective tissue disease, and systemic lupus erythematosus seem to have a significant degree of proliferative fibrous tissue extending down on either side of the neurovascular bundles, similar to that seen in Dupuytren’s contracture, together with increased periadventitial fibrosis around the common digital arteries and superficial palmar arch. Consequently, the entire superficial palmar arch system should be inspected, not only to release these fibrous septa but also to determine whether there are any segmental occlusions amenable to resection and interposition vein grafting. The adventitia is then stripped from the superficial palmar arch, the radial digital artery to the index finger, the common digital arteries to the
second, third, and fourth web spaces and their continuation as the radial and ulnar proper digital arteries, and the ulnar digital artery to the little finger. Under the operating microscope, common digital arteries that initially appeared occluded have sometimes become soft and patent after resection of the adventitia, similar to the dilatation of vein grafts after stripping off of the adventitia with jeweler's forceps (Fig. 2). Digital sympathectomy may therefore produce improved blood flow in the digital arteries, not only by interruption of the sympathetic control but more simply by removal of the constrictive cuff of periadventitial fibrosis from around the arteries. We therefore consider digital sympathectomy to be a "decompression arteriolyis," analogous to epineurectomy of a peripheral nerve in a compression neuropathy. Long-term follow-up to document the incidence of recurrent digital ulceration will be necessary to determine whether digital sympathectomy is in fact effective, not only in patients with an exaggerated sympathetic vasoconstrictive response to cold after crush injuries and cold injuries but, more important, in patients with connective tissue disorders. In this series patients with connective tissue diseases have reported a lessening of the severity of their pain and cold intolerance and healing of ulcers in the short term, but it is our impression that these results have not been maintained over the long term, probably because of occlusive disease becoming the predominant component in these diseases.  

Resection of the thrombosed segment of ulnar artery and reconstruction with interposition vein grafting is well established for hypothenar hammer syndrome. Occlusion of the distal radial or ulnar arteries has been documented by angiography in various systemic diseases, such as connective tissue disorders, renal vascular disease, and atherosclerosis. Consequently, restoration of arterial inflow by reconstruction of a new "palmar arch" with an interposition vein graft from the distal radial or ulnar arteries to the common digital arteries might be expected to increase the inflow perfusion pressure to the involved digits in such patients. However, microsurgical revascularization can be considered only if satisfactory distal runoff can be documented by visualization of the common digital arteries on angiography and if there is satisfactory backflow from the common digital arteries at the time of exploration. Only patients with chronic systemic disease meeting our stringent angiographic and clinical criteria have undergone revascularization, and the long-term patency of these vein graft reconstructions has been comparable to those reported by Wilgis and Koman and Urbaniak in the hypothenar hammer syndrome. Although microsurgical revascularization might be expected empirically to increase the inflow perfusion pressure to the digits, two alternative mechanisms may also play a role. Since Zook et al. have suggested that resection of a thrombosed digital artery may itself improve digital perfusion by reduction of any secondary vasospasm, it is conceivable that resection alone of a thrombosed segment of the distal radial or ulnar arteries or superficial palmar arch may reduce the sympathetic discharge from this thrombosed segment and prevent it from causing vasoconstriction of the remaining collateral circulation. In addition, the extensive dissection required to expose the superficial palmar arch and common digital arteries to allow resection of the occluded segment may, in itself, be a form of distal sympathectomy and may therefore alleviate some of the secondary vasospasm. Whatever the mechanism, microsurgical revascularization can be expected to be only a palliative intervention since it is likely that any underlying arteriopathy will continue to progress, not only in the remaining digital arteries but also in the reconstructed vein grafts themselves.

REFERENCES

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