UPPER EXTREMITY ARTERIAL ISCHEMIA

By

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INTRODUCTION

Ischemic arterial lesions of the upper extremity are not viewed with the same degree of concern as lower extremity lesions by both, the patient and physician. Only five percent of patients symptomatic from peripheral vascular disease seek treatment for upper extremity arterial system disease (1). Such lesions are less common, occur in association with a wide variety of underlying causative factors, and are often ascribed to minor trauma or cold weather. Consequently, the primary cause remains untreated until there is interference with work, the end organ i.e., digits, faces amputations, or when multiple symptomatic episodes direct attention to the problem. Severe arterial ischemia leading to minor (digital) or major amputation of the upper limb is an even more catastrophic event than loss of the lower limb in terms of impediment to daily chores, ability to earn a livelihood, and most important, patient self image. In addition, significant morbidity results from a poorly functioning upper extremity as a result of untreated prolonged arterial ischemia or where treatment has been delayed.

It is the purpose of this paper to review the pathophysiologic mechanisms, etiology, clinical features, differential diagnosis, invasive and noninvasive diagnostic methods, and finally, outline briefly the therapeutic approach to the ischemic upper limb.

Pathophysiology:

Due to increased vascularity of the fingers, the blood flow to the upper extremeities is larger in the hand than the forearm. Ludbrook measured this plethysmographically to be 1.5 to 12 cc/100 cc/minute at room temperature (2). This is higher than the 4.1 to 5 cc/100 cc/minute flow when expressed per unit volume of the forearm. Fingertip flow depends on surrounding temperature and degree of sympathetic activity and can vary from one to 150 cc/100 cc/minute. This variation is because of the large muscular and cutaneous arteriovenous shunts in the fingertips, estimated at 501 anastomoses per square centimeter in the nail bed and 236 per square centimeter in the fingertip (3).

The vasodilator drugs may work by lowering peripheral resistance in the peripheral vascular bed, but objective data do not support their action on the larger collaterals (4). So, their efficacy in tissue loss or rest pain is doubtful, and may in fact be harmful as valuable blood flow may be diverted to normal areas and away from severely diseased areas. Lassen and associates state "vasodilator therapy of any form is contraindicated in patients with impending or manifest gangrene(5). The rationale for sympa-

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thectomy rests on the fact that it results in loss of vascular tone in vessels supplied by vasoconstrictor fibers. The increased blood flow following sympathectomy is limited to cutaneous vessels where the arteriovenous shunts are affected more than the capillaries (6).

Etiology:

The commonly encountered causes of upper extremity arterial ischemia are listed in Table 1. Trauma, arterial embolism and thrombosis are most frequent serious causes.

Table I: Causes of Upper Extremity Arterial Ischemia

Arterial Embolism	Cardiac, Thoracic Outlet Syndrome, Atheroemboli
Arterial Thrombosis	Arteriosclerosis, Polycythemia, Frostbite, Drug Injection, Aortic Dissection, Iatrogenic.
Venous Thrombosis	Phlegmasia Cerulea Dolens.
Arterial Trauma	Acute Blunt Or Penetrating, Thermal, Occupational, Crutch Trauma.
Vasospastic Disorders	Raynauds Disease—Primary and Secondary, Collagen Diseases.
Buerger's Disease	
Arteritis	Nonspecific, Drugs, Takayasu.

Arterial Embolism: Peripheral arterial embolism is being more frequently seen due to an aging population with associated arteriosclerosis. A cardiac source of an arterial embolus can be identified in 75-90% of patients (7). Chronic atrial fibrillation associated with arteriosclerosis or rheumatic heart disease is the commonest case, followed by acute myocardial infarction. James and colleagues reviewed 36 patients with upper extremity thromboembolism, constituting 15% of all patients with arterial thromboembolism (1). A cardiac origin was suspected in 88% and arteriosclerotic heart disease was documented in 80% of these patients. The brachial artery was the most common location of the embolus. Bacterial endocarditis, prosthetic heart valves, atrial myxoma, atheroemboli, and paradoxial emboli are other causes of arterial emboli to the upper extremity. A high percentage of patients in whom a source cannot be identified, persistent and close followup will often reveal an underlying cardiac source. Sudden onset of pain, numbness, coolness, and occasional sensory or motor disturbances of the upper limb are common presenting features. Physical examination usually indicates absent or weak pulses, coolness, pallor, and often sensory or motor deficits(7).

Microemboli from non cardiac sources are an important cause of upper extremity arterial ischemia, if only because they are not thought of often enough (8). Foci for microemboli include subclavian or axillary artery lesions from thoracic outlet compression, arterial aneurysms, and atheromatous intraluminal lesions. Mural thrombus inside the subclavian artery compressed between the first rib and clavicle is the most frequent cause, followed by embolization form a true arteriosclerotic aneurysm. Complete cervical ribs, anomalous ribs, or deformed clavicles from old fractures can also compress the subclavian artery with a post stenotic dilated vessel which harbors a thrombus with the potential for embolization. Propagation of throm-

bus proximal to smaller vessels occluded by showers of emboli makes embolectomy often a futile procedure in these cases (8).

Arterial Thrombosis: Danto has emphasized three basic mechanisms for arterial thrombosis (9): change in the arterial wall (e.g. arteriosclerosis), alteration in the blood itself (e.g. polycythemia), and decreased blood flow (e.g. dehydration). Most patients are male and may have signs of atherosclerosis elsewhere. The left subclavian artery is involved at its origin three times more frequently than the right, with most often, no symptoms. Patients presenting with multiple, small punctate necrotic areas over the fingers and hands should remind the observer of a blood dyscrasia such as polycythemia vera.

Iatrogenic arterial thrombosis is being more often seen due to large numbers of cardiac catheterizations being carried out. James et al., described nine patients with brachial artery thrombosis following retrograde catheterization (1). Delay in diagnosis or procrastination following loss of a previously present pulse was a major factor in morbidity and limb loss. Extensive use of radial artery catheters and frequent arterial blood gas analysis has increased the need for closer monitoring of the palmar circulation. Mozersky and colleagues in a study of 140 normal volunteers estimated that between 4.8 and 12.8 patients per 1000 can be expected to develop acute hand ischemia after radial artery cannulation (10). They also pointed out that an additional 25 to 50 patients per 1000 would have a viable hand solely dependent on poor collateral circulation. A correctly performed Allen test or doppler ultrasonic evaluation is helpful in preventing a large majority of these problems.

The problem of intra arterial drug injection, once rarely seen because of accidental barbiturate injection by anesthesia per-sonnel, is now a common problem in drug abusers. Drugs that have been implicated include: chlorpromazine, propoxyphene hydrochloride, hydroxyzine hydrochloride, pentazocaine hydrochloride and ergot. Pathogenesis usually involves intimal damage followed by arterial thrombosis. An experimental study in the rabbit ear with sequential angiograms following intra arterial injection of thiopental sodium demonstrated an interesting pattern of injury (11). Acute venoconstriction was the initial injury followed by venous thrombosis and gangrene with variable patency of the arterial circulation. The femoral triangle and the antecubital fossa are the usual locations for the injections. Severe pain occurs instantaneously along with blanching, cyanosis, and swelling.

Venous Thrombosis: Massive acute venous thrombosis (phlegmasia cerulea dolens) is uncommon in the upper extremity and is even more rare as a cause of arterial ischemia. Concomitant arterial and venous thrombosis can certainly exist in a patient where the first rib or scalenous anticus muscle compresses and occludes both vessels. Ischemic venous thrombosis results in severe pain, cyanosis, and edema. Excessive fluid loss occurs in the extremity and circulatory collapse may occur. Venous gangrene occurs five to seven days following the onset of ischemia (12).

Arterial Trauma: Increasing high speed traffic and the use of firearms and other weapons to settle disputes has provided an unwelcome challenge for the modern day vascular surgeon. Blunt trauma to the upper chest with the fracture of the first or second ribs should lead one

to suspect arterial injury in the region of the subclavian or axillary arteries. Often in the absence of evidence of direct trauma to this region the subclavian or axillary artery can be completely avulsed and result in severe arterial ischemia of the upper limb. Trauma to this region is a surgical challenge, and carries significant morbidity and mortality from hemorrhage and associated nerve injuries. It is also important to realize that blood vessels do not have to be transected or directly injured with a missile to produce arterial thrombosis. A high velocity missile even in the vicinity of a major vessel can produce intimal thrombosis and arterial ischemia because of a cavitation effect.

Percutaneous radial artery cannulation is increasingly used for monitoring and diagnostic purposes. Justifiable concern exists regarding the incidence of post cannulation thrombosis(13). Bedfor studied 100 patients with 105 radial artery cannulations(14). Radial artery thrombosis resulted in 38% of patients. A pulse was palpable in 73% and doppler signals were audible 90% of the time in the radial artery distal to the thrombus by way of the ulnar artery. All the thrombosed vessels which were followed up, recannalized and no major ischemic complications were noted. In contrast Baker and colleagues(15) reported five patients seen over a 20 month period who developed severe ischemia of the hand following the use of indwelling radial artery catheters, all of whom lost part of one or more digits. An obvious alternative, should the Allen's test or doppler test be even questionable, is the use of the superficial temporal artery for monitoring or diagnostic purposes. Chronic, repeated trauma can also produce thrombosis of the microvasculature and small arterioles in the digits. Operators of drilling equipment or other heavy machinery, pianists, and typists can present with ischemic finger lesions with or without skin necrosis. The "vibrating tool" syndrome is well recognized in patients involved in occupations involving use of pneumatic drills(8).

Improper, prolonged use of crutches is well known to cause nerve palsy but injury to the artery is less commonly recognized(16). Acute thrombotic occlusion or embolization of loose thrombus from the lumen of an axillary artery aneurysm are the two common presentations. Prevention requires the use of elbow supporting crutches which do not cause trauma to the axillary vessels. The implication of recognizing this entity is that resection of the segment of artery rather than simple embolectomy is required.

Vasospastic Disorders: Primary Raynauds is typically episodic and seen in women, involves both upper limbs, and is usually precipitated by cold weather or emotional problems. Tissue loss is minimal. At least in its early stages, this is believed to be a purely vasospastic condition without organic arterial obstruction(17). A diagnosis of primary Raynauds Disease can only be made tentatively as a minimum followup of 3-5 years is necessary to exclude "secondary" Raynauds Disease. The latter can be due to underlying collagen diseases, arterial occlusion from embolism, arterial thrombosis, heavy metals, ergot, etc. "Secondary" Raynauds Disease can present with Raynauds phenomena months or years prior to a clinical diagnosis of collagen disorders such as disseminated lupus, scleroderma, dermatomyositis or rheumatoid arthritis, and malignancies such as multiple meloma and leukemia.

Sumner and Strandness studied 105 patients with Raynauds phenomena and found primary Raynauds Disease in only 16, autoimmune disease in 25, and suspected autoimmune disease in another 22 patients(18). Porter and colleagues prospectively investigated 100 patients with Raynauds phenomena(19). The incidence of immunologic disease was 81% and conditions like Buerger's Disease and arteriosclerosis were not found to be a cause of Raynauds phenomena. They proposed an intensive search for underlying autoimmune or connective tissue disorders. Such studies included a complete blood count, sedimentation rate, chest x-ray, and complete gastrointestinal radiologic workup, including a barium swallow. Immunologic tests included serum protein electrophoresis, quantification of immunoglobulins, rheumatoid factor, veneral disease reaearch laboratory test, lupus erythematosis cell preparation, antiglobulin test, cold agglutination titers, and antinuclear antibody. Detailed magnification hand arteriography was also performed on these patients.

Buerger's Disease: Buerger's Disease is currently not a fashionable diagnosis since clinicians are now more attuned to arteriosclerosis obliterans. It usually affects cigarette smoking males and is thought to predominently involve the lower extremities. Concomitant superficial venous thrombosis involves non-varicose veins in about 40% of patients(8). When diagnosed in the upper extremity, it is frequently bilateral, and multiple segmental occlusion of smaller arteries of the hand is noted on arteriography.

Arteritis: Since obstruction of hand arteries is not typical of arteriosclerosis, arteritis has been advanced as a possible etiology. Digital ischemia may be caused by arteritis associated with the collagen diseases already mentioned. Ergot intoxication and heavy metal ingestion can cause digital ischemia. However, the former usually affects the lower extremity to a much greater degree. Uremic arteritis can also present with digital ischemia and plain x-rays of the hand typically reveal calcification of the vessels(13).

Takayasu Arterities is an uncomnion condition affecting young females, with an onset mimicking a rhumatic illness which is often overlooked(20). The patient later presents with loss of pulses in the upper extremity. Cerebral hypoperfusion may co-exist. Prognosis depends on the course of the disease but generally cardiac and neurologic complications ensue, with few patients surviving beyound five years.

Clinical Features:

The clinical presentation of upper extremity arterial ischemia is generally similar to lower limb ischemia, and the acuteness of presentation will depend on the cause of arterial occlusion. Complaints can vary from simple claudication of the forearm and upper arm muscles to gangrenous digits. Claudication can occur in the forearm muscles, but are not as severe because of the lesser degree of heavy activity involved. Housewives typically notice an aching or cramp in their forearms during housework or brushing their hair. Men involved in heavy machinery work complain of a "charley horse". Spontaneous ulceration of the fingertip or nonhealing following minor trauma to the skin is occasionally the only presenting feature of upper extremity arterial ischemia. Nonhealing often follows an infection occuring in the nail bed area, such as paronychia, due to poor nutrient blood supply. An ischemic lesion of a fingertip typically presents

as a tender, cyanotic fingertip followed by diminution of the pain and formation of a hard eschar over this area. Weeks or months following this, a small depression may be left as the only evidence of the ischemic episode. Gangrenous changes may appear as multiple, small areas of cutaneous necrosis following embolic occlusion of terminal arterioles.

An occupational history may provide a clue regarding chronic recurrent digital trauma or heavy metal injection. A prior history of drug abuse or evidence of multiple needle tracks may suggest intra arterial drug injection or mycotic aneurysms proximally. Examination should consist of verifying the presence of subclavian, axillary, brachial, carotid, radial, and ulnar pulses. Bilateral brachial pulses should be recorded for comparison. Allen's test at the wrists should be performed to detect occlusion of the radial or ulnar artery. Obliteration of the radial pulse with the arm in a 90° abduction, external rotation with the head turned towards the opposite side can be present in 50-70% of the normal population. However, the reproduction of the patient's usual symptoms along with disappearance of the pulse does indicate the presence of a narrow thoracic outlet.

Noninvasive Laboratory Diagnosis:

The percutaneous doppler ultrasonic flowmeter is a valuable tool to detect the presence or absence of adequate palmar collateral circulation prior to radial arterycannulation(10). The superficial palmar arch is located with the doppler, the direction of flow is noted, and the response to compression of both the radial and ulnar arteries is also noted. Because the ulnar artery is the dominant vessel in making up the superficial palmar arch, radial artery occulsion does not threaten limb viability in the majority of hands.

Objective methods of diagnosis such as digital pulse wave forms or pulse volume recordings and digital blood pressure measurement can be used to separate exaggerated cold sensitivity from organic small vessel blockage(21,22). Digital plethysmography is an extremely sensitive method for documenting peripheral arterial occlusive disease. Digital pulse volume recordings are utilized both to confirm the presence of a lesion as well as evaluate sympathetic activity prior to and after surgical sympathectomy (Fig 1).

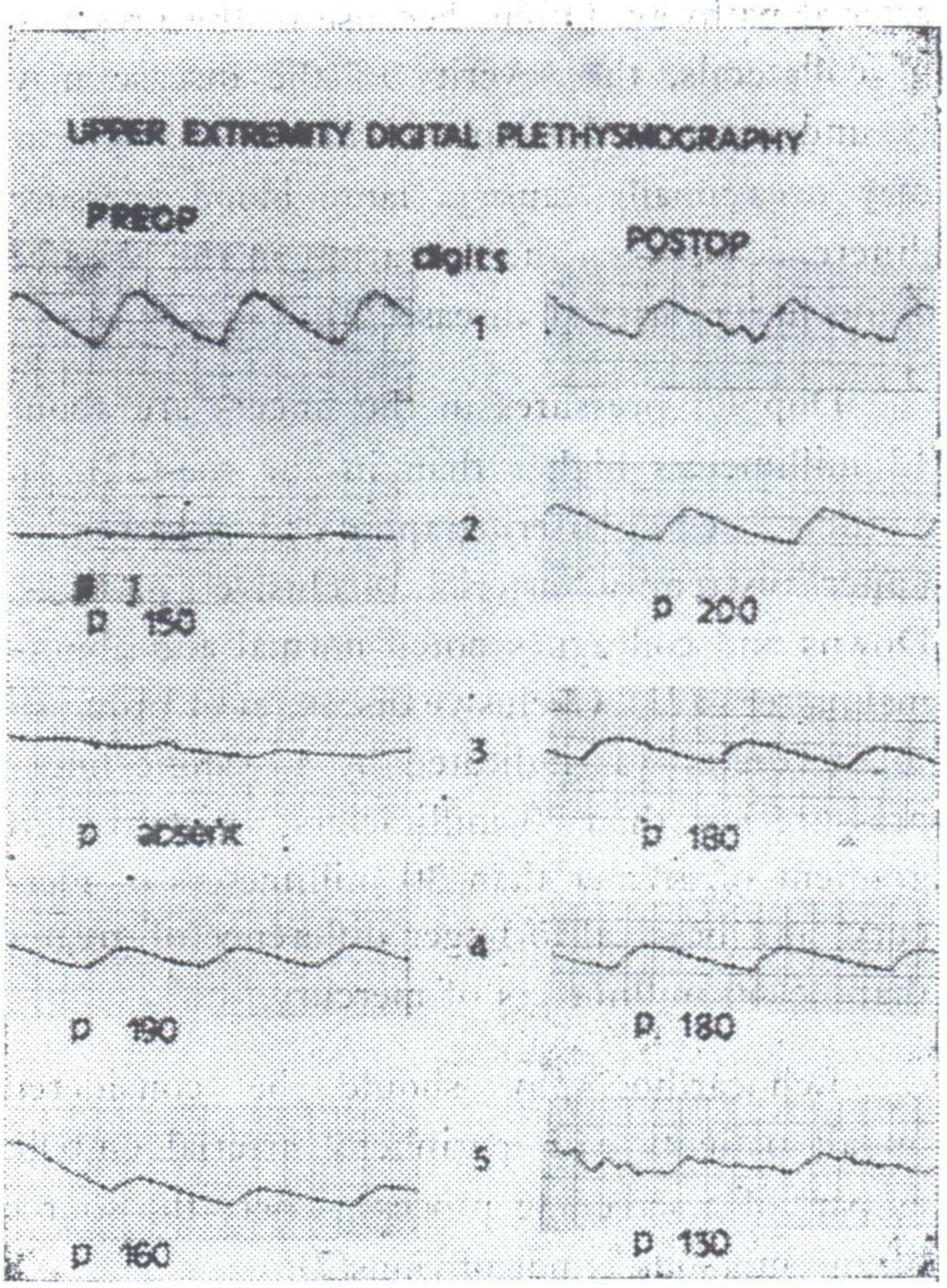

Fig. 1*:* Clinically successful transthoracic cervicodorsal sympathectomy for distal ischemia confirmed by improvement in postoperative pulse volume recordings.

Vasospasm produces blunting, loss of reflected wave, and a reduced amplitude of the arterial wave form. Stressing the patient with immersion of the hand in ice water and recording tracings upto five minutes afterwards, helps in the diagnosis of early cases(18).

Measurement of systolic doppler blood pressure in the upper extremity helps document the presence, location, and severity of the occlusive disease process. Difference in the blood pressure between the two arms greater than 15-20 millimeters of mercury should arouse suspicion of a stenotic or occulsive lesion in the arterial pathway. Often, because of the presence of collaterals, the severity of the disease may be underestimated. Supravalvular aortic stenosis may occasionally cause a large blood pressure difference between the two arms, in the absence of occlusive arterial disease(23).

Doppler pressures in the fingers are about 13 millimeters higher than in the toes(22). In a study based on arteriographic demonstration of upper extremity arterial occlusive patterns, Downs and colleagues noted normal and abnormal patterns(21). Occlusive disease at or proximal to the digits was indicated by a systolic finger pressure less than 70 millimeters, wrist to digit gradient of greater than 30 millimeters of mercury, and brachial to finger difference of greater than 35-40 millimeters of mercury.

Echocardiography should be considered on all patients with peripheral arterial emboli, as part of a screening process, where the source of an embolous is not obvious(7).

Invasive Diagnosis:

Arteriography remains the mainstay of accurate diagnosis and for planning therapy. If evaluation of the entire arterial tree from the subclavian artery to the digital vessels is desired, transfemoral catheterization is required under local anesthesia. Percutaneous brachial cannulation provides excellent visualization of the digital vessel where the proximal vessels are not suspected to be involved. Intra arterial injection of papaverine is frequently helpful in assessing the response to intra arterial vasodilators, as well as visualizing the digital vessels. Arteriography to outline the subclavian or axillary artery in suspected thoracic outlet syndrome should be performed with the arm in neutral and hyperabduction positions. An arterial embolus is suggested on arteriography by the presence of a sharply demarcated, filling defect in the dye column, normal arteries elsewhere and insufficient colateral formation around the obstruction. Cardiac catheterization and ventriculograms are often necessary to confirm echocardiographic findings of intra ventricular thrombus in patients with arterial emboli.

Treatment:

Non Surgical: Non operative measures in general for small vessel occlusive disease include cessation of tobacco in any form, an attempt to obtain relief with vasodilators and inculcating an obsession to protect the involved extremity. Often, cessation of tobacco and avoidance of cold is all that is necessary in younger patients with Raynauds phenomena(13). Protection of the extremity involves the use of heavy, warm gloves in even moderately cold weather, prevention of even minor trauma to the fingers and a change in occupation if necessary, to avoid repetitive minor trauma to the digits.

Anticoagulation with continuous intravenous heparin may be used either as primary treat-

ment for small vessel thrombosis or secondary to prepare for surgical treatment and following procedures such as arterial embolectomy. Heparinization alone may be useful in the prevention of further thrombosis following digital artery occlusion where direct surgical repair is not feasible. Longterm oral anticoagulation may be chosen for certain patients with arterial emboli secondary to atrial fibrillation where a high likelihood of recurrence exists. This will not prevent recurrent emboli in all patients and certainly, the substantial hazards of anticoagulation have to be weighed against possible benefits(7). Thrombolytic therapy in peripheral arterial occlusion has not been extensively used. Amery and colleagues performed post treatment arteriograms in patients with occlusions of less than 12 hour duration and noted clearing in 75% of the cases(24). If occlusion was present longer than 3-5 days the chance for clearing the obstruction was markedly reduced. Because patients with an arterial embolus have a higher risk of re-embolization from the original source, thrombolytic therapy is not a viable alternative as far as definitive therapy is concerned.

Stellate ganglion block can be performed percutaneously under local anesthesia to try and predict success of subsequent surgical sympathectomy. Relief of symptoms in the absence of blockage of the adjacent brachial plexis portends relief following sympathetic nerve section. Intra arterial injection of one milligram of Reserpine at three to six month intervals offers an alternative to surgical sympathectomy in selected cases. Unfortunately, the effect is often shortlived and the response variable(17,19). The mechanism of action apparently involves depletion of the arterial wall of norepinephrine, which persists for about two weeks.

Surgical Treatment : Acute upper limb ischemia secondary to an embolus is treated by arterial embolectomy, usually performed under local anesthesia unless access to other areas is desired, or the need for extensive reconstruction is anticipated. Exposure of the brachial artery in the mid upper arm and proximal and distal control is followed by a transverse arteriotomy. Withdrawal of the clot cranial and caudad with the help of a Fogarty embolectomy catheter is performed. After restoration of flow, intraoperative arteriography is often obtained to confirm complete evacuation of the clot. In delayed treatment of ischemic limbs, compartment syndromes can develop and fasiotomy should be utilized liberally. The limb salvage rate following arterial embolectomy overall is in the range of 85-90%(7). Following arterial embolectomy, the source of the embolus should be sought.

Chronic obstruction of the subclavian artery alone rarely causes severe ischemia reguiring reconstruction. However, if the obstruction is extensive and collaterals are poorly developed, secondary surgical options are available. Direct endarterectomy or bypass from the arch to a suitable recipient vessel can be performed. However, the current approach favors an extrathoracic bypass with much less morbidity and mortality. A subcutaneous vein or prosthetic bypass from a patent donor vessel (e.g. opposite subclavian or axillary artery) to the ipsilateral axillary or brachial vessel can be performed under general or local anesthesia.

Where thoracic outlet syndrome is the cause of arterial compression, transaxillary resection of the first rib decompresses the space effectively. Arterial reconstruction and grafting is usually necessary to remove the diseased

segment of artery and the supraclavicular approach is preferred in this instance. Resection of the artery and rib will prevent further emboli, but embolectomy to remove small vessel thrombi or large organized thrombi is not indicated and return of distal pulses should not be expected.

Severe hand ischemia from diagnostic procedures, i.e. radial artery puncture or cannulation, is treated with thrombectomy under local anesthesia and reconstruction of the artery with autogenous vein patch if necessary. Systemic anticoagulation or Dextran may be necessary postoperatively following small vessel repair. Arterial insufficiency to a terminal part of a digit can sometimes be treated effectively by resection of a part of the thrombosed artery (Leriche principle of sympathetic denervation) (15). With the current microvascular surgical techniques, a small segment of reversed arm vein can be used to bypass symptomatic localized arterial obstructive lesions even in the peripheral part of a digit. Surgical biopsy of the digital neurovascular bundle is rarely feasible for fear of nonhealing of the incision. If it is essential to the diagnosis, a small incision is made on the lateral or medial aspect of the involved distal phalanx to remove a small length of the bundle.

Thoracic sympathectomy for selected patients with small vessel occlusion of the digits gives satisfactory results. Resection of the chain from T1 through T5 produces effective sympathetic denervation of the upper extremity. Resection of only the lower half of stellate ganglion will avoid Horner's Syndrome. Many routes for sympathectomy have been proposed—cervical, posteror, thoracic, and anterior thoracic. The axillary transpleural approach provides the best exposure, is simple, and does not involve transecting muscle. Postoperative intercostal neuralgia is a common problem, as with any thoracotomy. This can be minimized by avoiding heavy pericostal sutures around the ribs. Excellent long-term relief is obtained following a complete upper extremity sympathectomy in properly selected patients. Sympathectomy for primary Raynauds Disease in the absence of complications is usually not indicated(17). In patients with longstanding ischemia secondary to arterial emboli from subclavian artery compression by the first rib, sympathectomy along with first rib resection is often performed through the same incision(8).

Treatment of limbs with intra arterial drug injection consists of elevation, anticoagulants or Dextran to limit small vessel thrombosis, analgesics and occasionally intra arterial injection of Reserpine for relief of arterial spasm. Fasciotomy should be utilized early to prevent ischemic muscle contracture and nerve dysfunction.

SUMMARY

The majority of patients with upper extremity arterial ischemia, including those with hand or digital ischemia, can be diagnosed by a detailed history, physical examination, and a few simple tests. Complex hematologic, immunologic, and arteriographic investigation is useful in sorting out the more difficult problems. Noninvasive arterial testing is valuable in confirming the clinical diagnosis and in followup of patients on various drug regimens or after surgical procedures. It is now recognized that severe hand or digital ischemia is not caused by vasospasm and is almost always due to organic arterial occlusion. Modern techniques have enabled vascular surgeons to effectively deal with occlusive lesions of the upper extremity, restore function, and in most cases, achieve limb salvage.

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