Surgery Remains the Most Effective Treatment for Paget-Schroetter Syndrome: 50 Years' Experience

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Background. **Significant improvements were made in the diagnosis and management of Paget-Schroetter syndrome (thrombosis of the axillary–subclavian vein) secondary to thoracic outlet syndrome during the past 50 years. The diagnosis has often been extremely difficult.**

Methods. **Multiple approaches both in diagnosis and therapy have been tried during the years. After recognizing that the underlying pathologic process resulted from an abnormal insertion of the costoclavicular ligament laterally on the first rib, along with hypertrophy of the scalenus anticus muscle, 506 of 626 extremities have been managed by thrombolytic therapy followed by prompt transaxillary resection of the first rib. These patients have been followed up from 1 to 32 years (average of 7.2 years ± 1.0 standard deviation).**

Results. **Four hundred eighty-six patients (96%) improved. Because the pathophysiology is not well understood, many venograms suggest intraluminal disease rather than external compression. Therefore, attempts at opening the narrowed vein with intraarterial techniques do not work. Use of percutaneous venous angioplasty with stents have all occluded in our experience, making further management difficult. Venous bypass grafts fail because of low venous pressure.**

Conclusions. **Recognition that an abnormal congenital lateral insertion of the costoclavicular ligament on the first rib causes venous occlusion in Paget-Schroetter syndrome has led to acute thrombolysis, followed by prompt first rib resection, as the ideal management.**

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Paget-Schroetter syndrome (PSS), or "effort" thrombosis of the axillary subclavian vein, is most often secondary to thoracic outlet syndrome (TOS). It usually occurs in patients with excessive arm activity in the presence of one or more compressive elements in the thoracic outlet. The syndrome was described independently by Von-Schroetter [1] in 1884 in Vienna and by Paget [2] in 1875 in London. For many years, therapy included elevation of the arm with anticoagulants and subsequent return to work [3, 4]. If symptoms recurred, the patient was considered for first rib resection with or without thrombectomy [5, 6]. When the congenital lateral insertion of the costoclavicular ligament was recognized as the underlying cause, prompt transaxillary rib resection was used immediately after thrombolysis.

The availability of thrombolytic agents [7–11], combined with prompt surgical neurovascular decompression of the thoracic outlet [5], has reduced morbidity and necessity for thrombectomy, substantially improving clinical results including the return to work. For those seen late (greater than 6 weeks after the thrombotic episodes), thrombolytic agents followed by first rib resection have been successful, but slightly less so.

The purpose of this report is to define the evolution of diagnosis and therapeutic management in patients with effort thrombosis secondary to TOS [12–15], based on understanding the anatomy and pathophysiology, and to compare it with the natural history of the disease [16–18].

The axillary subclavian vein traverses the tunnel formed by the clavicle and subclavius muscle anteriorly, the scalenus anticus muscle laterally, the first rib posterior-inferiorly, and the costoclavicular ligament medially (Fig 1).

In most patients with thrombosis of the axillary subclavian vein (PSS), the costoclavicular ligament congenitally inserts further laterally than normal (Fig 2). When the scalenus anticus muscle, which is lateral to the vein, becomes hypertrophied through activity and exercise, the vein is significantly narrowed. This is not the case when the costoclavicular ligament inserts in a normal place much more medially on the first rib, even with significant scalenus anticus muscle hypertrophy.

When the vein clots secondary to TOS and PSS evolves, there is usually a severe inflammation in the area of the thoracic outlet. This markedly handicaps the anatomic dissection by obscuring the location of the first rib and the anatomy of the thoracic outlet. In addition to the inflammation, which often plasters the neurovascular structures onto the chest wall obliterating the view of the

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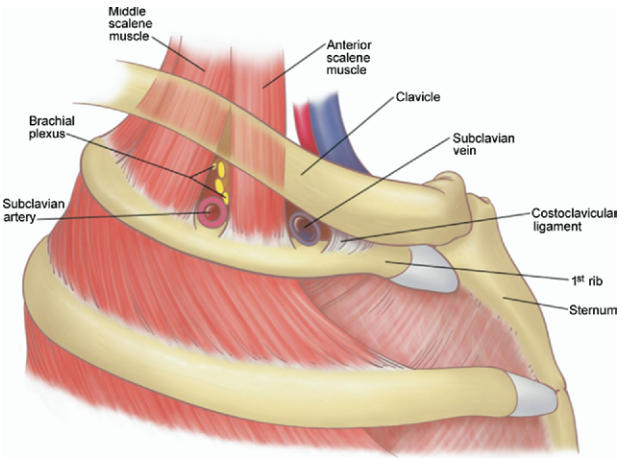

Fig 1. Normal anatomy of the thoracic outlet with conventional insertion of the costoclavicular ligament on the first rib.

first rib, there is a loss of the usual blue color of the vein because of the lack of blood flowing through it. This removes one of the best anatomic landmarks, further increasing the difficultly for the surgeon both in the acute and subacute phase of the syndrome.

Material and Methods

The chair of the institutional review board of the Baylor Research Institute reviewed the study and determined that it was exempt from institutional review board approval under 45 CFR 46.101 (b) (4).

Clinical manifestations of effort thrombosis of the axillary subclavian vein in the acute and subacute phases were evaluated in 626 extremities of 608 patients, 18 being bilateral. There were 307 women and 301 men, ranging in age from 16 to 51 years, with a mean of 32 years. (For the remainder of the discussion, the number

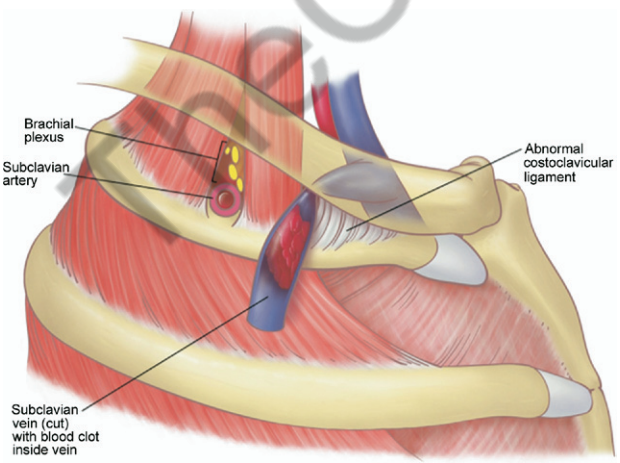

Fig 2. Congenital abnormal lateral insertion of the costoclavicular ligament on the first rib with hypertrophy of the scalenus anticus muscle lateral to the vein and thrombosis of the axillary-subclavian vein (Paget-Schroetter syndrome).

Table 1. Symptoms and Signs

Sign or Symptom	Number of Extremities
Venous distention (arm)	626
Subcutaneous venous collateral around shoulder (Urschel's sign)	618
Arm swelling	602
Bluish discoloration	591
Aching pain (with exercise)	207
Cervical ribs	62
Bilateral	18
Minimal symptoms	24

of the extremities will be assessed rather than the number of patients.) Four hundred thirty-two patients had unusual occupations that involved excessive, repetitive muscular activity of the shoulder, arm, and hand. Potentially aggravating occupations included such sports as golf, tennis, baseball, football, weight lifting, cheerleading, and drill team members, or other pursuits such as painters, beauticians, and linotype operators. The symptoms were usually exacerbated by working overhead, cold temperatures (weather or air-conditioning), or hanging the arm down for long periods.

Diagnosis

In 626 extremities, swelling or venous distention over the chest, arm, or hand occurred, suggesting the clinical diagnosis of venous obstruction (Table 1). Elevation of the arm or hand did not seem to change the configuration of the veins or swelling acutely. Bluish discoloration was observed in 544 arms, and aching pain, which was increased by exercise, occurred in 520. Cervical ribs were noted in 62 instances. The bilateral syndrome occurred in 18 patients (12 women and 6 men), simultaneously in 2 (1 with previous bilateral clavicular fractures), and sequentially in 16. In 24 patients, only minimal symptoms were present.

One hundred percent of the extremities demonstrated a positive Adson's sign, hyperabduction sign, or various other compressive signs related to the thoracic outlet compression. Diagnostic tests performed included venous ultrasound studies, venous scintillation scans, venography, plethysmography, temperature studies of the extremity, and bilateral upper extremities nerve conduction velocities including both the median and ulnar nerves.

The diagnosis was established by clinical history, physical examination, ultrasound studies, and venogram performed through a medial antecubital vein. An indwelling plastic catheter was inserted into the antecubital vein after 1980 so that serial venograms and thrombolytic injections could be performed.

Substantial narrowing or occlusion of the axillary subclavian vein in the area of the first rib and clavicle was observed in all patients on venogram. Some collateral circulation was evident in 621 extremities, although it was obviously not adequate if swelling was present. The

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pathways for the most efficient collateral communication were between the cephalic, transverse cervical, transverse scapular, and tributaries of the internal jugular veins, producing an increased subcutaneous venous plexus around the shoulder (Urschel's sign).

Differential diagnosis includes lymphangitis or lymphatic obstruction, intramuscular hemorrhage, and intermittent venous occlusion without thrombosis, as well as venacaval or innominate vein obstruction.

Surgical Considerations

INDICATIONS. Indications for surgery include (1) diagnosis of PSS (persistent symptoms such as aching pain and the feeling of a "heavy" or "fatigued" arm, and swelling of the extremity with or without venous distention); (2) a venogram positive for significant extrinsic compression in either the anatomic position or a challenging position such as the Adson's; and (3) after thrombolytic treatment regardless of whether it was successful or not.

OPERATIVE TECHNIQUE: TRANSAXILLARY RESECTION OF THE FIRST RIB AND NEUROVASCULAR DECOMPRESSION, AND RESECTION OF THE COSTOCLAVICULAR LIGAMENT. The transaxillary approach provides excellent visualization of the vein and costoclavicular ligament. Also, the neurovascular structures are away from the dissection and do not have to be retracted, thus, minimizing their injury.

The patient is placed in the lateral position with the involved extremity gently supported by forearm traction straps attached to an overhead pulley with one to two pounds of weight. Two arm holders prevent hyperabduction of the shoulder beyond 90 degrees. After prepping and draping the axilla and arm, a transverse incision is made below the axillary hairline between the pectoralis major and latissimus dorsi muscles. The dissection is carried through the skin and subcutaneous tissue to the chest wall, and extended cephalad to the first rib. Care is taken to prevent injury to the intercostal brachial cutaneous nerve, which passes between the first and second ribs to the subcutaneous tissue in the center of the operative field. With gentle dissection, the neurovascular bundle is identified and its relation to the first rib and both scalene muscles is clearly outlined to avoid injury to these structures.

In most instances of PSS there is a significant inflammatory reaction that usually "plasters" the neurovascular structures down to the chest wall. This obliterates access to the first rib, requiring careful dissection of the neurovascular structures away from the ribs.

Further contributing to the difficulty of surgical dissection is the usual occlusion of the axillary-subclavian vein, eliminating its blue color, which is usually a crucial anatomic landmark in the normal axillary dissection.

The scalenus anticus muscle is divided and resected up into the neck to avoid reattachment to Sibson's fascia. The lung on the operated side is temporarily collapsed with a double-lumen endotracheal tube to expedite safe dissection. The first rib is dissected subperiosteally and carefully separated from the underlying pleura to avoid pneumothorax. The rib is divided and a triangular portion removed, with the vertex of the triangle at the scalene tubercle. The anterior portion of the rib is dissected carefully from the vein, the costoclavicular ligament is divided and resected, and the rib is detached at its sternal cartilaginous attachment. All compression bands and adhesions are removed from the axillary subclavian vein, and the anterior venous compartment is thoroughly decompressed.

The posterior segment of the rib is carefully dissected subperiosteally from the subclavian artery and brachial plexus posteriorly. The scalenus medius muscle is dissected from the rib. The rib is divided near its articulation with the transverse process of the vertebra. Complete removal of the neck and head of the first rib is achieved with long, specially reinforced double-action Urschel-pituitary and Urschel-Leksell rongeurs (Pilling Weck Surgical, Fort Washington, PA). The eighth cervical and first thoracic nerve roots as well as the brachial plexus undergo careful neurolysis. If a cervical rib is present, it is removed and the seventh cervical nerve root is decompressed. Meticulous hemostasis is accomplished. Only the subcutaneous tissues and skin require closure, because no large muscles have been divided. The patient is encouraged to use the arm normally and can usually be discharged from the hospital on the second day after the surgical procedure.

It is preferable to remove the entire first rib, including head and neck, to avoid future bony or fibrocartilage regeneration and irritation of the plexus [19]. The infraclavicular approach has been used successfully by others. For recurrent symptoms, removal of incompletely resected or regenerated rib and lysis of adhesions can best be accomplished through the posterior "high thoracoplasty" approach.

Anticoagulants are not used after surgery.

SURGICAL OBSERVATIONS. The vein is one of the landmarks for the dissection of the first rib because of its blue color. In patients with PSS, the vein is generally occluded or markedly thickened and there is usually no blue color. Because of this, the degree of surgical difficulty is increased.

In most patients who have been operated on less than 6 weeks after thrombosis, there is usually a severe inflammatory reaction around the neurovascular structures and first rib. These structures are frequently plastered down to the first rib, increasing the dissection hazard. Several cases that had previous breast implants placed through the axilla were included in this group of patients. Extra care should be taken with the dissection of the neurovascular structures.

If an interval clot recurs or was not relieved by the thrombolytic agents, the external venous compression structures are removed (particularly the abnormal costoclavicular ligament), and the clot usually lyses spontaneously, recanalizing the vein. Three attempts to use the Roto-Rooter technique to open the occluded vein have not been ideal.

Table 2. Treatment Protocols

Protocol	Number of Extremities
Group I	36
Initial anticoagulant therapy (return to work)	36
Persistent or recurrent symptoms	21
Group II	42
Initial thrombolytic treatment (return to work)	42
Persistent or recurrent symptoms	25
Transaxillary first rib resection with thrombectomy	25 4
Group III	506
Thrombolytic treatment (within 6 weeks from thrombosis)	506
Prompt transaxillary first rib resection with thrombectomy	506 2
No anticoagulants (return to work)	506
Group IV	42
Thrombolytic treatment (after 6 weeks from thrombosis)	42
Prompt transaxillary first rib resection with thrombectomy	42
No anticoagulants (return to work)	0
Roto-rooter destruction of the clot	3
Total	626

Treatment Protocols

GROUP I. Thirty-six extremities were treated before 1975 with anticoagulant therapy (heparin sodium followed by Coumadin [crystalline warfarin sodium]). All were seen before 6 weeks from the time of thrombosis. The treatment was usually initiated in the hospital. This group was instructed to elevate the arm, and all patients were encouraged to return to work. Recurrent symptoms similar to those of the initial syndrome developed in 26 of those 36 extremities after the patient returned to work. Twenty-one of these were readmitted to the hospital, and after Coumadin (warfarin sodium) therapy was stopped, a transaxillary resection of the first rib was performed with removal of all compressive elements including the congenital or adhesive bands, the costoclavicular ligament, and the scalenus anticus muscle. In 8 extremities thrombectomy was performed. The axillary subclavian vein was opened transversely and a Fogarty catheter was used to remove clot from inside the vein. It was usually not necessary to patch the venotomy. Proximal control was difficult (Table 2).

Clavicular resection was carried out in 1 patient bilaterally because of callous formation from old fractures of the clavicles, which had initially caused the TOS. Although a clavicular resection tends to be disfiguring when performed unilaterally, a bilateral resection is less cosmetically objectionable [20]. The patient had excellent function of the arms and shoulders postoperatively. She was even able to touch her shoulders together anteriorly.

GROUP II. A second group of 42 extremities was treated after 1975. These were managed initially with heparin (sulfated glycosaminoglycan) and a venogram. Thrombolytic agents were administered in most instances through an indwelling catheter in the antecubital vein. After lysis of the thrombus, first rib resection was performed promptly in 25 extremities. In the first 10 patients, the thrombolytic agent was streptokinase (Streptase, Aventis Pharma, Australia) with the initiating dose of 250,000 U followed by 100,000 U/h intravenously for 24 to 48 hours or until lysis of the clots occurred. This was monitored with prothrombin times and partial thromboplastin times.

Because of the disadvantage of the systemic effect of streptokinase, it was replaced with urokinase (Abbokinase, Abbott Labs, Chicago, IL) in 26 extremities. The loading dose was 4,400 U/kg by bolus, and a dosage of 4,400 U/kg per hour was given until clot lysis occurred (mean duration of treatment was 26 hours). Assessment of the therapeutic effect included frequent observation as well as serial venograms through the indwelling catheter. Most patients with recent clots showed major improvement in less than 24 hours after administration of the thrombolytic agent.

Because of the experience of recurrent symptoms after discharge of group I, 25 of the 42 extremities in group II were treated promptly with surgery. Heparin and thrombolytic therapy were stopped, and the first rib was removed through a transaxillary incision in 25 extremities (a combined transaxillary-supraclavicular approach was used in 2 patients [5, 21, 22]).

It was now recognized that the primary cause of PSS was the congenital "far lateral" insertion of the costoclavicular ligament on the first rib. When there is hypertrophy of the scalenus anticus muscle (lateral to the vein), the vein occludes. Therefore, complete division of the costoclavicular ligament, the scalenus anticus muscle, and any bands or adhesions was performed. Scalenectomy was extensive to minimize the possibility of muscle fibers reattaching to Sibson's fascia. Any congenital or compressive bands were removed from the axillary subclavian vein. Venograms were occasionally obtained (in 4 patients) when the patient was on the operating table, but usually direct observation was adequate to assess patency of the vein. The apparent suggestion of retained clot and failure of the thrombolytic agents to completely lyse the clot was usually explained at operation by external compressive elements alone, even when there appeared to be "residual clot" on the venogram. The patients were discharged after several days and were followed clinically without anticoagulants. After 6 weeks, most were allowed to return to work.

Table 3. Evaluation of Results

Scale	Pain Relief	Employment	Limited Recreation
Excellent	Complete	Full	None
Good	Almost complete	Full	Some[a]
Fair	Partial	Limitation	Moderate[a]
Poor	None	No return	Severe[a]

[a] Isometric exercise is avoided.

Table 4. Clinical Results

Group	No. of Extremities	Excellent/ Good	Fair	Poor	Recurrence
I	36	16	15	4	1
II	42	25	15	2	0
III	506	486	20	0	0
IV	42	24[a]	6	12	0

[a] Three responded to Roto-Rooter clot destruction.

GROUP III. The most recent 506 extremities were treated similarly to group II. They were seen within the first 6 weeks after the thrombosis and underwent thrombolytic therapy and prompt transaxillary first rib resection without anticoagulants.

GROUP IV. Forty-two extremities were not seen until after 6 weeks from the time of venous occlusion. All were treated similarly to group II. In only 50% of these patients was it possible to decrease the amount of clot with urokinase infusion. However, none could be completely opened. All underwent prompt transaxillary first rib resection.

Results

Results were evaluated as excellent, good, fair, or poor according to the criteria in Table 3 and are summarized in Table 4. In group I, 10 extremities of the 36 showed substantial improvement with anticoagulation and evaluation therapy. In 25 extremities, symptoms either were not completely improved or recurred. The pain, swelling, and dysfunction usually occurred when the patient returned to the job that had produced the difficulty initially.

Twenty-one of those required first rib resection, and 8 also underwent thrombectomy to completely remove the clot. The long-term results demonstrated 21 patients with intermittent swelling of the arm with use; they were categorized as having fair results. Two patients had constant symptoms, and 2 others had development of phlegmasia cerulea dolens with marked chronic edema of the arm and were unable to return to work. They were considered to have poor results. One of the patients who underwent surgical intervention had recurrence.

Because of these less-than-ideal results and the observation that a first rib resection was usually necessary for patients to return to vigorous work, therapy for group II was modified. Patients were hospitalized and, through an antecubital indwelling catheter, a venogram was performed and thrombolytic therapy initiated. After lysis of the clot, prompt first rib resection with removal of compressive elements including the costoclavicular ligament and scalenus anticus muscle was performed in 25 of the 42 patients. Long-term results indicated that 25 of the 25 operated on had good to excellent results, ie, the patient returned to work without symptoms. Two patients were evaluated as having fair results, ie, intermittent swelling, but able to work. No patient had phlegmasia cerulea dolens.

Group III patients were those seen less than 6 weeks after thrombosis and treated similarly to group II. Four hundred eighty-six of 506 extremities had good to excellent results.

All patients in group IV were seen 6 weeks or more after thrombosis and also treated similarly to group II. Although none could be completely opened with thrombolytic agents, all were treated promptly with surgery, and 24 recanalized subsequently or developed excellent collateral circulation with good results later, whereas 6 had only fair results, as did 3 more who were opened with Roto-Rooter treatment and surgery; 9, however, did not open or develop collateral circulation. These were not clinically improved, with 5 exhibiting a severe postphlebitic syndrome. Coumadin did not seem to improve results, but the patients were not randomized for this therapy. None of the three venous bypass grafts in the postphlebitic group (a reversed saphenous vein from the axillary to jugular vein) remained open after 3 months in the latter group.

Patients are followed for 3 months clinically and once yearly by letter or phone.

There was no mortality in any group and no observed evidence of pulmonary embolism.

Comment

Historically, Sir James Paget [2] in 1875 in London and Von-Schroetter [1] in 1884 in Vienna described this syndrome of thrombosis of the axillary-subclavian vein, which bears their names. The word *effort* [14] was added to thrombosis because of the frequent association with exertion superimposed on anatomic compressive elements in the thoracic outlet. Trauma, unusual occupations requiring repetitive muscular activity, as has been observed in professional athletes, linotype operators, painters, and beauticians, and cold tend to increase the susceptibility to thrombosis [15]. Elements of increased thrombogenicity also exacerbate the incidence of this problem.

DeWeese and colleagues [3] reported long-term results in patients treated conservatively with elevation and Coumadin (natural history). There was a 12% incidence of pulmonary embolism, which was not observed in any of our patients. Development of occasional venous distention occurred in 18%, and late residual arm symptoms of swelling, pain, and superficial thrombophlebitis were noted in 68% of the patients (deep venous thrombosis with postphlebitic syndrome). Phlegmasia cerulea dolens was present in 1 patient.

These findings substantiate our observations from group I that a more aggressive operative approach after thrombolytic therapy is indicated. This is particularly true for younger patients with precipitating factors.

One advantage of urokinase over streptokinase is the direct action of urokinase on the thrombosis distal to the catheter, producing a more localized thrombolytic effect [10, 11]. Streptokinase produces a systemic effect involv-

ing the alteration of serum plasminogen and increasing potential complications. A decrease in the need for thrombectomy after use of the thrombolytic agent followed by aggressive surgical intervention is another advantage, as some of the long-term disability is related to more morbidity from thrombectomy as well as recurrent thrombosis [3, 5, 23]. During the last 10 years, urokinase has been withdrawn from the market and tissue plasminogen activator (Alteplase-Activase; Genetech, Inc, South San Francisco, CA) has been substituted.

The natural history of PSS suggests moderate morbidity [3, 5, 18] with conservative treatment alone. Bypass with vein or other conduits [24–26] has limited application in our experience. Open thrombectomy has been abandoned because of poor long-term results. Causes other than TOS must be treated individually using the basic principles mentioned. Intermittent obstruction of the subclavian vein [27] can lead to thrombosis, and decompression should be used prophylactically.

In the patients who were seen 6 weeks or more after thrombosis, thrombolytic agents were usually ineffective in completely removing the clot. The more aggressive Roto-Rooter techniques were helpful in only 3 patients, but usually we are not able to penetrate the hardened fibrotic area. Some of these patients did recanalize at a later stage (24 of 42 patients recanalized spontaneously or developed adequate collateral circulation and became asymptomatic after first rib resection). Of the 18 patients with persistent occlusion who did not recanalize after first rib resection, 5 experienced severe postphlebitic syndrome, 6 had moderate symptoms, and 3 responded to Roto-Rooter destruction of the clot with the relief of symptoms. Roto-Rooter also failed in 4 cases and the clot was unable to be opened.

It is obvious that the earlier that a patient with PSS (effort thrombosis) can be seen by the physician and treated with thrombolytic agents followed by prompt first rib resection, the better the results. The longer the time interval between the acute thrombolytic episode and therapeutic intervention (that is longer than 6 weeks), the less effective the therapy. Efforts at thrombolysis, or Roto-Rooter techniques, have not been as successful after 3 months.

Molina and colleagues [28] recently published a study of a group of PSS patients treated by thrombolysis, infraclavicular first rib resection, and thrombectomy. This is a more complicated operation, and long-term thrombectomy has not been successful in our experience. The balloon dilatation of a venous lesion or use of stents also has failed in our group of patients.

In our experience, there is little evidence that long-term anticoagulant therapy has any benefit, either after therapy or for conservative treatment of patients who remain occluded. Certainly, thrombogenic states such as dehydration should be avoided.

A summary of the operative procedures for relieving TOS compression [29] and the reasons for their failures [30] should be reviewed [31–35].

The contributions of Rachel Montano and Brenda Knee are of immeasurable value for their dedication and commitment to the research and completion of this publication.

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DISCUSSION

DR JOHN A. KERN (Charlottesville, VA): Dr Urschel, now I know why I didn't get a manuscript before this presentation. (Laughter).

Those of us who are crazy enough to be interested and involved in doing thoracic outlet syndrome are clearly indebted to you for your contributions over the course of recent history and past history. I completely agree with your conclusions that the best treatment for Paget-Schroetter syndrome is prompt lysis and decompression. I disagree a little bit with the necessary need for a transaxillary approach. I favor an infraclavicular approach, sometimes combined with a supraclavicular approach. I think that also allows great vision of the costoclavicular ligament, clearly the key component for extrinsic compression. The reason I utilize that approach is I think there is some benefit. Sometimes in these folks in which the situation has been going on a bit longer, I find a little bit of a need for more than just the external decompression, but I also kind of do an external venolysis and really circumferentially free it up, divide all that inflammatory tissue, and I think that helps reestablish flow through the vein.

The other thing I have been doing recently and I wonder if you could comment on, I often take these folks back down to radiology postop[erative] day 1 and I take another look, and then if they still have some residual narrowing or intraluminal abnormalities, I think they are a little more likely to respond to balloon venoplasty and they get a nice result with that. So that is one kind of question, if you could comment on the utility of that.

I just stretch it open with a balloon, just stretch it open, and I think that helps reestablish—as you said, the body naturally recanalizes. I just think that helps get rid of their symptoms a whole lot quicker.

The one really main question I have is for those patients who develop their Paget-Schroetter as a result of a malunion clavicle fracture, what is your approach for those patients? Do you take the clavicle out or do you still do a transaxillary first rib?

DR URSCHEL: The clavicle fracture is the easy one. We used to treat all thoracic outlets by claviculectomy. We used to do the venograms on the table after thrombectomy. But basically no matter what the vein looks like on the inside, if you take the external compression away, the body usually recanalizes the vein.

The infraclavicular approach is fine. It is not as easy as the transthoracic. With the supraclavicular approach you can get at it, but it is very difficult. Why do it the hard way? It is a long run for a short slide. There are many ways to approach this.

We don't ever let a physical therapist get a patient after thoracic outlet syndrome, because they do too much. We don't ever let the interventional radiologists see them again because they will always do something to them. It seems to be acutely remunerative. Whenever you send a patient to some specialist, they always want to do something—so that you have to make the decision. If you understand the pathophysiology, you will treat the patient properly. I am not saying there is only one way.