Thoracic Outlet Syndrome:
Review and Reference to Stroke in a Major League Pitcher

The thoracic outlet compression syndrome is reviewed. It should be considered in all neurologic and vascular complaints of the upper extremity. Symptoms are related to arm position and use and are aggravated by sustained activity that stresses the shoulder. A participant in any sport that requires a repetitive violent throwing motion is a potential candidate for this condition. A detailed report of a major league pitcher illustrates the importance of early diagnosis and appropriate treatment, inasmuch as delay can be catastrophic, as in the case of this career athlete. A thorough history is the best aid in arriving at a diagnosis. When the symptoms are mild or moderate, conservative therapy may suffice. When surgery is indicated, the object is to create adequate space for passage of the brachial plexus, subclavian artery, and subclavian vein. Patients with thrombosis and occlusion of a major artery supplying the upper extremity require an additional arterial reconstructive procedure plus a cervicothoracic sympathectomy.

Vascular compression in the thoracic outlet, with or without the presence of local musculoskeletal anomalies, is a well-documented entity [1]. When thoracic outlet syndrome occurs in athletes involved in repeated violent exercise of the arms, the subclavian artery may be subjected to a severe degree of recurrent trauma, resulting in intramural hematoma, dissection, and thrombosis of the artery. Antegrade or, less often, retrograde propagation of a thrombus or extension of an intramural hematoma places the cerebral circulation in great jeopardy by exposing the vertebral artery and, on the right side, the common carotid artery to proximal occlusion and/or distal embolization.

Case Report

A 30-year-old, right-handed major league pitcher was first seen in 1980. During spring training of that year he complained of progressive fatigue and a sense of heaviness in his pitching arm after a lengthy workout or after he had pitched five or six innings. Once he began the pitching rotation during regular season, it became even more of a problem because he could not go past the third or fourth inning without great difficulty. After that, the greater the amount of effort, the more serious the problem. He was seen on several occasions by the team physician and was subsequently referred to an orthopedic surgeon specializing in bone and joint problems of baseball players.

In June 1980, during the third or fourth inning of a game, his arm suddenly "went dead," and he had numbness and tingling in the first three digits of the right hand. He was unable to continue.

He was admitted to a hospital a few weeks later for diagnostic evaluation, and on July 25, 1980, angiography demonstrated complete occlusion of the right subclavian artery just distal to the origin of the thyrocervical trunk and immediately proximal to the medial edge of the first rib (fig. 1A). The brachial artery was reconstituted via thyrocervical/subscapular collaterals (fig. 1B).

No surgery was performed at that time, and the patient was discharged from the hospital and allowed to resume his workouts. Early in the morning on July 30, 1980, he went to a chiropractor for manipulation of his neck. Later in the afternoon of that same day, while
working out on the field, the patient experienced a sudden onset of confusion, headache, tinnitus, nausea, and generalized weakness, but did not lose consciousness. He was readmitted to the same hospital, where it was noted that he had suffered a stroke, manifested by left motor and sensory paralysis and left homonymous hemianopia. Arteriography revealed occlusion of the innominate artery, presumably by a thrombus (fig. 2). He was taken immediately to surgery, where a large amount of clot was removed from the innominate and right subclavian arteries and a smaller amount from the proximal segment of the right common carotid artery. After slow, but satisfactory, recovery, the patient began a rehabilitation program.

He first came under the care of one of us (W. S. F.) on September 29, 1980, and was admitted to another hospital for further assessment. At that time he was walking with only minimal difficulty, but
had residual muscular weakness of the entire left upper extremity and left facial weakness.

His visual fields were full to confrontation testing when a single stimulus was presented from either side, but there was obvious inattention to the left visual field when simultaneous stimuli were presented from both sides. Neuroophthalmologic assessment revealed an incongruous left upper homonymous quadrantanopia. Some acquired myopia secondary to ciliary spasm was also present.

Examination by computed tomography (CT) on September 30, 1980, revealed large infarcts in the right basal ganglia, the right occipital lobe, and the right cerebellar hemisphere (fig. 3). It was presumed that these were embolic and had originated at the time of acute occlusion of the innominate artery on July 30, 1980. The appearance of the infarcts has remained basically unchanged on follow-up CT studies despite progressive clinical recovery. Arteriography on October 1, 1980, showed rounding-off of the subclavian artery after surgery (fig. 4A). Collateralization of the brachial artery remained slow (fig. 4B), to the extent that one could not study the distal upper extremity even with selective injections into the suprascapular collateral. During the late phase of the suprascapular study, the subclavian vein was seen to narrow considerably as it crossed the first rib (fig. 4C). The intracranial circulation was unremarkable except for a slightly delayed flow in the posterior temporal branch of the right middle cerebral artery.

It was believed that the best hope for complete athletic rehabilitation would be a subclavian-brachial bypass, using an external iliac artery as an autograft. This was the procedure the patient chose after all options, both surgical and nonsurgical, had been discussed with him. He was then transferred to a third hospital (out of state), where surgery was performed on October 14, 1980.

Because of the patient's height (203 cm) and weight (118 kg), both external iliac arteries were harvested and sutured end-to-end to form a new subclavian-axillary conduit. The external iliac arteries were replaced with synthetic grafts. In addition, a right first rib resection, a scalenectomy, and an upper thoracic sympathectomy were performed. The patient tolerated the lengthy procedure very well, and his recovery was uneventful. When he was released from the hospital on October 25, 1980, the blood pressure in the right arm was normal and equal to that in his left arm, and there was no bruit audible over the graft. Slight numbness remained in the right middle finger but appeared to be resolving.

He returned to the care of one of us (W. S. F.) and entered a physical fitness program. By January 1981, the strength of his left upper extremity was almost normal, but both still showed residual increase of tendon reflexes. The left facial weakness was no longer apparent. Although repeat neuroophthalmologic assessment revealed no left visual field defect, a perceptual deficit still seemed to be present. As is often observed in patients with right cerebral hemisphere lesions, both his depth perception and his ability to transfer two-dimensional planes into three-dimensional displays were
The left external iliac artery graft was bypassed, using a graft made of different material. The patient's recovery was uneventful, and repeat arteriography demonstrated a patent, smooth left iliac graft with normal blood flow.

The patient once again began a reconditioning program and was able to increase his activity without any recurrence of the claudication. In June 1983, he returned to active training, pitching regularly and participating in other physical pursuits.

Before the start of the 1984 season, however, his contract expired, and he was placed on waivers. He was not picked up by any other team and, therefore, decided to retire from professional baseball. At the present time he is working regularly, supervising a sports program for minority youths, and continues to keep himself in good physical condition. He is neurologically normal on examination, but brain CT still shows the same hypodense lesions that were present at the time of his initial evaluation in 1980.

Discussion

Our mammalian predecessors carried their front legs in a dependent position so that they could stand on all four limbs. When we became bipeds and assumed an upright posture, our arms became parallel to the trunk. This moved the nerves, arteries, and veins (which leave the thorax to pass into the arm) laterally and increased their angle of flexion. The combination of this acute angle with stretching caused by the weight of the upper limbs might be expected to produce a thoracic outlet syndrome in everyone, but the support provided by the muscles of the shoulder girdle is normally adequate to compensate for this anatomic arrangement. If, however, this support fails or if the neurovascular bundle or the bony outlet from the thoracic cage is anatomically abnormal, the nerves, artery, or vein may become stretched or compressed [2].

All shoulder girdle compression syndromes have one common feature, namely, compression of the brachial plexus, the subclavian artery, and subclavian vein, usually between the first rib and the clavicle [3]. With elevation of the upper limb, there is a scissorlike approximation of the clavicle superiorly and the first rib inferiorly [4]. Grouping the various conditions under the single heading of thoracic outlet syndrome has resulted in more correct diagnoses and improved therapy. This syndrome should be considered in all neurologic and vascular complaints of the arm previously reported as scalenus anticus, hyperabduction, costoclavicular, cervical rib, fractured clavicle, cervicothoracic compression, pneumatic hammer, effort vein thrombosis, subcoracoid pectoralis minor, and first thoracic rib syndromes [5].

Our case involved retrograde propagation of a right subclavian artery thrombus into the brachiocephalic trunk. Such an occurrence, although rare, was first reported by Gould in 1884 [6] and again in 1887 [7]. Symonds [8] subsequently reported two cases of thrombosis of the subclavian artery accompanied by contralateral hemiplegia of sudden onset, probably caused by embolic infarction. As one might expect, the lesion in all published cases has been on the right side, since only on that side does circulation to both the head and arm originate in a common trunk from the aorta [9]. In all cases thus far reported, a latent period of 3 months to 3 years

![Subclavian arteriogram 3 years after stroke. Arterial conduit constructed from external iliac arteries serves as subclavian/axillary bypass autograft.](image)
has elapsed between the onset of upper limb symptoms and
the occurrence of hemiplegia. Although this complication is
uncommon, it must be anticipated and prevented because its
consequences are potentially devastating. We believe that
the chiropractic manipulation on our patient had no bearing
on the events that followed. Strokes after such maneuvers
involve the territory supplied by the vertebrobasilar arterial
circulation. To our knowledge no cases have been reported
that involve the carotid middle cerebral circulation [10].

Diagnosis

A detailed history from the patient is the best aid in arriving
at a proper diagnosis of thoracic outlet compression syn-
drome. Symptoms may originate from nerve compression
(paresthesias, numbness, pain, weakness), from arterial
compression (coldness, pallor, claudication, pain, numbness,
weakness), or from venous compression (edema, skin disclo-
oration, fatigue, prominent venous pattern over arm, shoulder,
or chest) [11]. If the sympathetic nervous system is involved,
the condition can lead to Raynaud phenomenon. When the
subclavian artery is compressed, poststenotic dilatation or an
aneurysm can be produced distal to the site of stenosis.
These dilatations or aneurysms can become thrombosed or
can act as foci for repeated small distal emboli [4]. In some
instances, amputation has been necessary because of re-
peated embolization and gangrene.

The symptoms in thoracic outlet syndrome are related to
arm position and use and are aggravated by sustained activity
that stresses the shoulder girdle such as combing or blow-
drying the hair; reaching; holding a newspaper, telephone, or
steering wheel; or hammering. A participant in any sport that
requires a repetitive violent throwing motion is a potential
candidate for thoracic outlet compression.

Symptoms are more common in those baseball players
whose throwing motion is overhand rather than sidearm. They
also occur in cricket bowlers whose throwing motion is tra-
ditionally overhand and in carmen who engage in repetitive
hyperabduction of both shoulders.

Strukel and Garrick [12] published what they believed to
be the first case reports of thoracic outlet compression in
athletes. Three of their four cases were in baseball players,
aged 16, 17, and 20 years. One of us previously reported
brief case histories of three other major league pitchers who
suffered from thoracic outlet compression symptoms [1]. This
author has learned subsequently of two others undergoing
surgery successfully during 1984; these athletes have both
resumed pitching (Yao JST, personal communication). We
are also aware of another case report of a left-handed major
league pitcher in whom a subclavian artery thrombosis had
occurred. After surgical intervention had solved his problem,
he was able to return to pitching [13]. He was not at risk for
a stroke because his occlusion was on the left side.

Objective findings are often minimal or absent and leave
one with a clinical diagnosis based largely on subjective
symptoms. The Adson, costoclavicular, and hyperabduction
tests are not consistently and unequivocally positive in repro-
ducing symptoms and, conversely, may produce radial pulse
obliteration in asymptomatic subjects [14]. Telford and Mot-
tershead [15] found that, of 120 healthy medical students,
38% had obliteration of the radial pulse with shoulder depres-
sion, 68% with shoulder retraction, and 54% with shoulder
abduction. These tests are helpful only when the symptoms
produced clearly simulate the patient’s complaints and cor-
relate with accompanying changes in the radial pulse.

Radiographs of the chest, cervical spine, and shoulder may
suggest the cause of a thoracic outlet compression only when
a gross anomaly such as a visible cervical rib is present.
Blood-flow studies using the Doppler technique are useful in
confirming that arterial flow diminishes or ceases during pos-
tural tests. Nerve conduction-velocity studies are valuable in
differentiating thoracic outlet syndrome from other neurologic
diseases and also as a guide to therapy [16]. Angiography
offers the most precise method of locating the site of
compression when the artery or vein is involved; it should be
performed with the arm in a neutral position and repeated
during postural maneuvers that produce alterations in the
distal pulse [14, 17].

Treatment

Nonoperative. In early and mild cases, conservative therapy
may suffice. Any of several techniques may be effective:
avoiding hyperabduction and obesity; improving posture by
elevating shoulders; exercising to strengthen shoulder girdle
muscles; cervical traction; moist heat; ultrasound; and rest
from all activities [4].

Operative. The aim of surgery is to create an adequate
passage for the neurovascular bundle. Certain patients re-
quire resection of bony abnormalities, such as prolonged
transverse processes, a cervical rib, or other congenital or
acquired abnormalities. In most patients, transaxillary resec-
tion of the first rib and scalenotomy is favored today [4].

Once vascular symptoms appear and a cervical rib is pre-
sent, it should be removed. Schein et al. [18] recommend
replacement of the affected segment of the subclavian artery
with a homograft to restore normal blood flow and prevent
further emboli or thrombosis. (In athletes, a homograft from
an artery bank) may not withstand repetitive stress as well as
an autograft from the patient’s body, as was used in our
patient.) In cases of brachial embolus, the distal vessel is
obliterated, and placing a graft does not improve the circula-
tion to the hand and forearm to an extent greater than would
be accomplished by a sympathectomy. Rob and Standeven [19]
subscribed to the idea that, since an upper thoracic ganglo-
necotomy could be done in a few minutes, it should always be
done to secure rapid and maximal dilatation of the collateral
channels.

Patients with thromboembolism and angiospastic phenom-
enia of a severe degree, or those with thrombosis and occlu-
sion of a major artery in the arm, require some arterial surgi-
cal procedure and, in addition, a cervicothoracic sympathectomy.
For this type of operation, most surgeons have used the
transaxillary approach.

When feasible, the artery should be directly reconstructed
or bypassed to restore adequate flow in the arm and prevent
further emboli or retrograde thrombosis from the affected
segment of the subclavian artery [20]. When this is impossible
or inadvisable, further distal embolization should be prevented by ligating the artery distal to the points of compression. One should recognize that a cervical rib is not an essential requirement for this syndrome, since any situation that results in prolonged or repetitive compression of the subclavian artery can lead to the mural and intimal changes that predispose to thrombosis and distal embolization.

The importance of early diagnosis and treatment of the syndrome must be emphasized. Vigorous investigation is required, particularly in the symptomatic athlete. A progressively shorter time interval from beginning of daily workout to onset of symptoms is an ominous sign of impending disaster, and investigation and treatment become matters of great urgency. With its typical historic/clinical presentation, the syndrome requires only our most basic diagnostic modality: Listen to the patient; he may be telling you the diagnosis.

REFERENCES
8. Symonds CP. Two cases of thrombosis of subclavian artery with contralateral hemiplegia of sudden onset, probably embolic. Brain 1927;50:259–260