INTRODUCTION

Stroke is the 3rd cause of death in the U.S.A., being preceded only by heart disease and cancer. It is estimated approximately 300,000 Americans suffer new strokes each year, that 1/3rd of those with acute thrombotic strokes die within 30 days of onset, and that 200,000 people in the U.S.A. die yearly from this disease. There are alive today, in the U.S.A., two million people who have had strokes. The social and economic consequences to the nation and the families of those individuals are incalculable.1

Increasing awareness of extra cranial location and segmented nature of atherosclerotic occlusive lesions in a large proportion of patients with cerebrovascular insufficiency has been followed by the development and use of removing or bypassing the offending plaques. It is now estimated that 75% of patients with ischemic stroke syndrome have at least one obstructive lesion at a surgically accessible site and that more than 40% have the principal occlusion confined to extra cranial vasculature.2

More than 75% of patients who suffer strokes have warning symptoms in the form of transient ischemic attacks (TIAs).3 Likewise 30-35% of untreated patients with TIA develop frank strokes if followed 3-5 years, or longer.4 5

PATHOLOGY

The Atheromatous Plaque consists of nodular deposition of fat, primarily cholesterol, in the arterial intima. There is associated inflammatory response resulting in fibroblastic proliferation. Calcium salts may precipitate in the primary plaque. As the lesion enlarges, the central portion undergoes softening and may rupture through the over-lying intimal surface and result in a discharge of the atheromatous debris (emboli) into the lumen of the vessel. An open cavity remains in the central portion of the lesion. This cavity (ulcer) can be the nidus for platelets aggregate or thrombus formation. Most frequently these aggregates are attached loosely and can be swept into the arterial stream as secondary emboli causing TIA or frank strokes. Hemorrhage may occur within the soft portion of the atheromatous lesion and result in a precipitous extension of the lesion producing acute occlusion of the artery at that location.6

Atheromatous lesions occur at branches or arterial bifurcations. The commonest location of significant lesions occurs at the carotid bifurcation. The ratio of extra-cranial to intracranial carotid lesions is 2 to 1.7

Stenosis of the carotid artery occurs in 34% of extra-cranial to intracranial carotid lesions; while right vertebral artery is 8.4% and left vertebral artery 22.3%; the innominate artery 12.5% and the left subclavian artery is 12.4%.

CLINICAL MANIFESTATION

The spectrum of symptoms in cerebrovascular disease can vary from minor episodes of neurologic dysfunction lasting less than 24 hours, to a major episode of cerebral infarction resulting in a permanent neurologic deficit.

Trans-Ischemic Attack is defined as producing an episode of neurologic dysfunction that lasts no more than 24 hours and usually lasts just a few minutes following the attack. The patient returns to pre-attack status without residual neurologic deficit. TIA may produce lateralizing symptoms in which case their arterial origin and subsequent distribution can be labelled clearly. They also can produce generalized symptoms such as ataxia, dizziness, vertigo and syncope, which suggests a brain stem distribution or posterior circulation. Ischemic, however, these attacks cannot be labelled routinely due collateral to circulatory interrelationships of the carotid and vertebral basilar systems.

The subclavian steal syndrome is one manifestation of vertebral-basilar insufficiency that occurs by way of an unusual manifestation of collateral circulatory redistribution. This abnormality was defined by Reivich et al8 in 1961 following an angiographic description in 1960 by Cantorni of retrograde flow in the involved vertebral artery.

When the origin of the subclavian artery becomes compromised, reversal of blood flow occurs in the branches of the first portion of the subclavian artery as well as the vertebral artery which can become a major collateral contributor for upper extremity blood flow; thus serving as a conduit through which the basilar artery blood flow may be siphoned off to collateralize the upper extremity exercise.

The classic symptoms complex, associated with this syndrome, includes vertigo and/or pre-syncope flowing upper extremity exercise. Other symptoms include bilateral visual disturbance, dysarthria, disorder of equilibrium, impairment of consciousness and drop attacks. There may be monoparesis or paralysis shifting from side to side and involving any of the extremities.

Oclusion of the innominate artery may cause the same symptom complex in addition to symptoms of carotid insufficiency, since the reversal of blood flow occurs also via the carotid which include headaches, dizziness, black out spells, loss of memory and buzzing
FIGURE I:
Pre-op arteriogram showing "Arrows" complete occlusion of innominate artery and the backflow via the carotid and vertebral arteries "Steal".

FIGURE II:
Post-op arteriogram showing the subclavian-subclavian graft with riling of both the right carotid and subclavian arteries.

FIGURE III:
The pulsatile graft can be seen. Lower neck incisions also are seen.
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It is significant to mention that a patient may demonstrate angiographic subclavian lesions without having any symptoms.

**EVALUATION OF PATIENTS**

A good history of the specific sequence of events involving neurologic dysfunction is the single most important step in evaluation of patients who may have cerebrovascular insufficiency. Careful history review will be helpful in identifying other vascular occlusive diseases and risk factors. The following points are important in examination of these patients:

1. Bilateral upper extremity blood pressure. A difference in arm pressure may be the first clue to occlusive diseases of the arch vessels.
2. Peripheral pulses evaluation.
3. Determination of neck bruits in the area of carotid bifurcation and over the subclavian arteries.
5. Neurologic examination.
6. Ophthalmic examination.
7. Other tests to evaluate internal carotid artery use Ophthalmosonometry (OSM), Ophthalmophotysthymography and Ophthalmodynamometry, Brain scan and cerebral blood flow.
8. Angiography which visualizes all aortic branches and cerebral blood vessels as well as using subtraction films.

The diagnosis of subclavian steal syndrome can easily be made by finding decreased pulse and blood pressure in the affected side in association with supraclavicular bruit. Serial angiogram can easily demonstrate the site of occlusion and the reversal blood flow to the affected site.

**TREATMENT**

Since the introduction in 1957 of the extra-thoracic bypass grafts for occlusive lesions of the aortic arch branches, a noticeable decline in the operative mortality (1%) and post operative morbidity (2-5%), has been achieved when compared with direct intra-thoracic approach.

Since then various authors have confirmed the effectiveness of various extra thoracic procedures. Various procedures have been used including carotid-subclavian, carotid-carotid, axillary-axillary, subclavian-vertebral and the most recent one carotid-vertebral by pass graft. The first report about using a subclavian-subclavian graft was by Ehrenfeld et al. 1968, in which they used a vein graft. The graft was layed in front of the trachea and involving transection of Sterno Mestoid and scalenus anticus. The first series to report about subclavian-subclavian dacron graft was by Norman Finkelstein et al. in which they reported their experience during 6 years and involving 13 patients. They retracted Sterno Mastoid and transected scalenus anticus on the affected side and placed the graft sub-cutaneously.

Searching the literature we could not find more reports about this procedure. We would like to make a plea for usage of this procedure more often because of the advantages it offers. It is easier, less traumatic, less time consuming, does not affect the carotid flow during or after surgery and the graft is short and less affected by motion. The subclavian artery is not a fixed artery and can be mobilized from the neck without angulation of the graft.

This bypass graft can be used in occlusions of the left subclavian artery, innominate artery, right subclavian artery and vertebral artery. Other grafts can be connected to it, to carotid arteries and vertebral arteries as needed.

The carotid-subclavian bypass graft has the following disadvantages. It may siphon the blood from the distal carotid artery with subsequent decrease in blood flow. Also, it carries the usual risks of any carotid surgery.

The axillary-axillary bypass graft avoids the complications of carotid surgery but it is a longer procedure, and using a longer graft, with more wound complication and a greater potential for mal-position causing compression of the graft.

The carotid-carotid bypass which recently has been advocated carries double risk of carotid surgery and should be preserved for those rare cases of occlusion of innominate artery and left subclavian artery simultaneously.

We would like to report a case of right innominate artery occlusion using modified subclavian-subclavian dacron bypass graft.

**CASE:** 67 year old white male, complaining of weakness and numbness of left hand and forearm, cramps of right leg which started several months ago and progressively getting worse to the point where he is getting rest cramps. Patient was also complaining of dizziness, light-headedness, drop attacks and falling several times while walking and working around the house. Patient is a right handed person and has been using his right hand more because of weakness of left hand. Now he is experiencing more attacks of dizziness, light-headedness and drop attacks. He noticed that his memory has been getting poor recently. His medication consists of Hydromorphone 25 mg twice daily, Zylorprim 300 mg daily and Vasodilan 20 mg 4 times daily.

Smokes cigarettes and drinks alcohol.

**EXAM:** Well nourished white male with the positive physical finding. B.P. right arm 70/?, left arm 150/86. Right carotid pulse is weak to absent. Right radial pulse is very weak. Right popliteal pulse is 1/4. Left popliteal 3/4 rt. Post-tibial 0, left post-tibial 2/4, right
di vi di n g ei th er th e Sterno Ma st o id o r sca le nu .. a nti cus. ubcl avi an-ubclavian 8mn1 dacron graft without muscle and the graft w a placed under the platysm a. recovery room. Th e Pul sa t ible graft w a ' 'a ily superficial femoral artery with good popliteal antera l ph ase .

HOSPITAL via the right caroti d art e r y and ri ght vertebral artery t rif u rc ati n ru

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