Subclavian steal syndrome

Part 1: Proximal vertebral to common carotid artery transposition in three patients, and historical review

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Three patients with central nervous system symptoms due to subclavian steal syndrome were treated with proximal vertebral to common carotid artery transposition. Neurological symptoms were relieved or improved in all three, with no decrease in blood pressure or pulse in the ipsilateral upper extremity. The colorful history of this syndrome is reviewed, and the various surgical approaches to its treatment are discussed. Although the literature suggests that the commonly used carotid to subclavian artery bypass graft and other similar extrathoracic procedures are generally safe and effective for relief of symptoms of the steal, there is also evidence that these bypasses may fail to restore antegrade flow in the vertebral artery, and, in fact, may steal from the carotid artery. Thus, the blood flow provided to the brain by these procedures may be hardly more than that provided by vertebral artery ligation, whereas the principal effect is to restore blood flow into the upper extremity. Vertebral artery ligation alone has been used in 20 patients, with neurological improvement in all cases and production of persistent intermittent brachial claudication in only one. These considerations and our patient experience suggest that a relatively simple operation, proximal vertebral to common carotid artery transposition, which emphasizes restoration of flow to the brain rather than to the upper extremity, may be preferable for most patients with neurological symptoms of subclavian steal syndrome.

KEY WORDS • basilar artery • carotid artery • cerebral angiography • cerebral artery • transient cerebral ischemia • subclavian steal syndrome • vertebral artery

A subclavian steal occurs when blood flows away from the circle of Willis, retrograde down the vertebral artery into the distal subclavian artery. Most cases are due to atherosclerosis, which causes stenosis or occlusion of the subclavian or innominate artery proximal to the origin of the vertebral artery. The subclavian artery distal to the stenosis or occlusion then functions as a sink. Retrograde flow occurs down the left vertebral artery in about 75% of cases. Other less common causes of this syndrome are trauma to, or embolization of, the subclavian artery. A few cases subsequent to surgical treatment of contralateral subclavian steal have been reported. A number of congenital cardiovascular anomalies, or occasionally even the surgical procedures used to treat these anomalies, may produce this syndrome. A principal example of this is the Blalock-Taussig procedure (subclavian to pulmonary artery end-to-side anastomosis) for tetralogy of Fallot, which involves division of the subclavian artery.

The natural history of subclavian steal syndrome is variable. Although a subclavian steal may be seen by angiography with moderate frequency, it is often asymptomatic. When the steal does become symptomatic, it may be incapacitating to the patient. Although most patients complain of neurological symptoms, a small percentage present with symptoms of brachial ischemia. Production of neurological symptoms by exercising the relatively ischemic arm is common in some series and rare in others. The neurological symptoms usually result from brain-stem and cerebellar ischemia, although cerebral symptoms may also occur, in the
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absence of carotid artery occlusive disease\textsuperscript{13,14,21,33,36,39} Completed stroke is uncommon in this syndrome\textsuperscript{21}; however, when stroke does occur, the cerebrum is often affected, as well as the brain stem or cerebellum.\textsuperscript{33,36,44,78} This paradox, of neurological deficit in areas not normally supplied by the vertebral or basilar artery, may result from secondary intracranial steal.\textsuperscript{75} Finally, there are even reports of spontaneous remission of symptoms and, in a case caused by trauma, return of antegrade vertebral artery blood flow documented angiographically.\textsuperscript{44,71,79}

We report on three patients recently treated for this syndrome. We describe and discuss a procedure, proximal vertebral artery to common carotid artery transposition, performed in these patients, and review the history of this syndrome. We discuss the various surgical approaches to its treatment.

Case Reports

Case 1

This 57-year-old right-handed high school maintenance foreman experienced progressively severe episodes of vertigo and dysequilibrium for 2 years. These episodes prevented him from working. He could not relate the episodes to arm exercise, but he noted that they frequently followed arising from a squatting position.

Examination. Both radial pulses were diminished, but they were easily felt, and there was no pulse delay. His brachial blood pressures were 74/40 bilaterally. At the level of the thigh, blood pressures were 150/90 bilaterally. There were soft bilateral carotid bruits. Arm exercise did not precipitate symptoms. His general and neurological examinations were otherwise unremarkable.

Angiograms of the aortic arch demonstrated complete occlusion of the proximal left subclavian artery with rapid appearance of contrast medium flowing retrograde down the left vertebral artery to the subclavian artery (Fig. 1). An enlarged branch of the thyrocervical trunk ran parallel with the vertebral artery into the subclavian artery. Moderate stenosis, less than 50\%, of the left common carotid artery bifurcation was present. Selective left common carotid artery injection also resulted in prompt visualization of the left vertebral and thyrocervical arteries by collateral feeding from the external carotid artery. A right retrograde brachial angiogram further demonstrated the rapid steal and moderate right proximal subclavian artery stenosis. The intracranial circulation was otherwise unremarkable.

Operation and Postoperative Course. On January 31, 1978, the proximal left vertebral artery was transposed to the left common carotid artery. The patient has experienced no neurological symptoms in 13 months since the operation, even with maneuvers that readily produced symptoms preoperatively. Blood pressure in the left arm was unchanged by the procedure, and the left radial pulse is readily palpable. The patient has returned full-time to his previous job.

An angiogram 1 year postoperatively demonstrated prompt antegrade filling of the left vertebral artery from the common carotid artery (Fig. 2). The left subclavian artery received collateral flow from the distal vertebral and external carotid arteries through the thyrocervical trunk (Fig. 3). Abundant filling of the basilar artery circulation was seen, and a minimal contralateral steal was demonstrated down the right vertebral artery. The left carotid bifurcation plaque had not progressed over the period of observation.

Case 2

This 64-year-old right-handed woman had experienced vertigo, staggering, and multiple falls for 5 months. During one of these episodes, she fractured her wrist. She had no symptoms of brachial ischemia, and she could not relate her spells to arm exercise.

Examination. The blood pressures in her upper extremities were 200/100 on the right and 150/90 on the left. She had soft left carotid and supraclavicular
bruits. The remainder of her general and neurological examinations was normal. Arm exercise did not produce neurological symptoms.

Transfemoral aortic arch and selective cerebral angiograms revealed very slight proximal left internal carotid artery stenosis and generalized intracranial vascular disease. The left subclavian artery was stenotic near the aortic arch, and flow in the left vertebral artery was retrograde into the subclavian artery (Fig. 4). Surgical correction was recommended, but the patient minimized her symptoms and refused the operation.

Eight months later she returned. Her symptoms had intensified and recurred daily, and she was unable to do her housework. She had also experienced occasional paresthesias in the right extremities. Her blood pressures were unchanged from before. There was a pulse delay at the left wrist. The left carotid bruit was definitely louder and higher in pitch than earlier. She had no neurological deficits. A second angiogram showed marked progression of the stenosis in both the left internal and external carotid arteries (Fig. 5). The left subclavian artery was more stenotic. The left vertebral to left subclavian steal was unchanged.

**Operation.** At this time, the patient had symptoms of both vertebrobasilar and left carotid artery insufficiency. Accordingly, on August 1, 1978, the proximal left vertebral artery was transposed to the left common carotid artery, and endarterectomy of the left common, internal, and external carotid arteries was performed.

**Postoperative Course.** She has done well, and has had no central nervous system or brachial symptoms. The blood pressure and pulse in the left upper extremity were unchanged by the procedure. An angiogram before discharge demonstrated prompt antegrade flow in the left vertebral artery and patency of the entire left carotid artery distribution (Fig. 6).

**Case 3**

This 48-year-old right-handed woman had been aware of asymmetric brachial blood pressures for at least 10 years, with pulse and blood pressure always difficult to obtain on the left. She complained of frequent episodes of vertigo, often accompanied by dysarthria, and bilateral hand and perioral numbness, during the same period. Six months before admission she experienced a frightening episode described as...
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“other worldliness,” with vertigo, dysarthria, and light headedness, lasting 24 hours. Diplopia followed for another 24 hours. On the day of admission, she suddenly developed left upper extremity paresis, followed within minutes by complete left hemiparesis. She had no symptoms of brachial ischemia, and could not relate her spells to arm exercise.

Examination. The brachial blood pressures were 130/80 on the right and 90/50 on the left. There was a definite delay in the left radial pulse compared to the right side. No arterial bruits were heard. The patient was irritable and lethargic. She exhibited a left central facial paresis. Visual fields were full to confrontation. She retained only antigravity power in the left upper extremity, with no ability to flex the fingers. The left lower extremity was mildly paretic. Sensory perception was impaired to all modalities on the left side. Exercise and cuff compression of her left arm to above systolic pressure neither aggravated nor alleviated her findings.

Transfemoral arch and selective cerebral angiograms revealed proximal occlusion of the left subclavian artery and no other occlusive or ulcerated lesions. Contrast medium promptly refluxed down the left vertebral artery into the left subclavian artery (Fig. 7). The right vertebral artery briefly and incompletely filled the superior cerebellar arteries, but the posterior cerebral arteries did not fill (Fig. 8). On right common carotid artery injection, the right posterior cerebral artery filled persistently from the right internal carotid artery. Both anterior cerebral arteries

Fig. 4. Case 2. Left: Initial arch angiogram shows stenosis of proximal left subclavian artery (arrow) and non-visualization of left vertebral artery. Right: Slightly later in the sequence the left vertebral artery (curved arrow) fills retrograde into the distal left subclavian artery (straight arrow). Collateral channels from the aortic arch to the distal subclavian artery are also seen.

Fig. 5. Case 2. Initial (left) and subsequent (right) left common carotid angiograms, anteroposterior projection, showing progression of stenosis of the proximal internal and external carotid arteries (arrows). During the interval of 8 months, the patient developed symptoms of left carotid insufficiency.

Fig. 6. Case 2. Postoperative arch (left) and left common carotid (right) angiograms showing vertebral to common carotid artery anastomosis (solid curved arrows), with antegrade flow in the vertebral artery (solid straight arrows) and prompt filling of the basilar artery (open curved arrow). The left internal and external carotid arteries are widely patent (open straight arrows).
FIG. 7. Case 3. Arch angiograms. Left: Early phase showing occlusion of proximal left subclavian artery (curved arrow), normal right vertebral artery (open arrow) and carotid artery bifurcations (straight arrows). Right: In the later phase there is visualization of the left vertebral artery (straight arrow). Collateral channels to the thyrocervical trunk (circumflex arrow) fill retrograde to supply the distal left subclavian artery (open arrow).

filled from the right internal carotid artery. An early draining vein emerged from the upper region of the right postcentral gyrus in the midst of a faint vascular blush, suggestive of luxury perfusion (Fig. 9). No occluded vessel was seen. The right common, internal, and external carotid arteries were normal. The left common carotid artery injection showed prompt, persistent, bilateral filling of the posterior cerebral and superior cerebellar arteries via the left posterior communicating artery. The left vertebral artery and cervical muscular branches were faintly opacified in a retrograde direction by collateral flow from the left external carotid artery. Computerized tomography (CT) scans with and without contrast enhancement on the second and seventh hospital days did not demonstrate cerebral infarction.

The cause of the patient's acute left hemiparesis was not entirely clear. Her past history indicated chronic vertebrobasilar insufficiency, and the distal basilar artery branches did not fill from the vertebral artery injection. However, additional angiographic findings indicated that an intracranial steal had also developed from the internal carotid to the vertebrobasilar distribution in response to the vertebral to subclavian artery steal. Evidence for this anterior to posterior circulation intracranial steal was the prompt and persistent bilateral filling of the posterior cerebral and superior cerebellar arteries from the internal carotid
arteries, as well as non-filling of the posterior cerebral vessels and the distal basilar artery by the vertebral arteries. Further, both anterior cerebral arteries filled from the right side. These flow patterns placed abnormal demands on the right internal carotid artery circulation. As a result of intracranial steal, the watershed area of perfusion between the right anterior and middle cerebral arteries may have become underperfused and subsequently ischemic. This explanation is supported by the location of the early draining vein and vascular blush in the watershed area, absence of a visible occluded vessel on the angiogram, and lack of a source of emboli from the proximal carotid arteries or from the heart.

The patient's left hemiparesis improved steadily. However, by the second week she still had only fair finger movement.

Operation and Postoperative Course. On February 13, 1979, the 12th hospital day, the proximal left vertebral artery was transposed to the left common carotid artery. Postoperatively, the left brachial blood pressure is 90/60, the left radial pulse is unchanged, and neurological function continues to improve.

Surgical Procedure

The critical technical points of proximal vertebral to common carotid artery transposition are as follows. The sternocleidomastoid and anterior scalene muscles are divided near their origins, for exposure of the subclavian and common carotid arteries. During operation on the patient's left side, the thoracic duct may be identified medially, and either protected or ligated, if entered (Fig. 10 left). After dissection of the vertebral artery, 3500 U of heparin are given intravenously, a temporary clip is placed high on the vertebral artery, and the vessel is divided distal to a ligature at its origin. The common carotid artery is briefly occluded, its wall incised, and an ellipse removed with a 4.5-mm aortic punch. A partially occluding vascular clamp is then applied to the common carotid artery, to allow distal flow to the brain during the anastomosis. The end-to-side anastomosis is performed with continuous 7-0 suture under the operating microscope (Fig. 10 center and right).

Carotid endarterectomy, when performed at the same operation, should follow the vertebral to common carotid artery anastomosis. This permits the restored antegrade flow in the vertebral artery to contribute to, and not steal from, the cerebral circulation during carotid occlusion. Furthermore, if endarterectomy were performed first, the fresh operative site might promote thrombus formation in cases where the force or volume of blood flow across it is reduced during partial occlusion of the proximal common carotid artery.

Historical Review and Discussion

The first reported case of subclavian steal syndrome and its surgical treatment seems to have been that of Mr. William Banks, a patient treated by Dr. Andrew Smyth at the Charity Hospital in New Orleans in 1864. It is of interest that Banks' subclavian steal was iatrogenic. The 32-year-old ship's steward developed a traumatic right subclavian aneurysm after clinging to an anchor with another man in turn holding on to him, as a result of a collision with
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Fig. 10. Anatomy for left proximal vertebral to common carotid artery transposition. Left: The sternocleidomastoid and anterior scalene muscles have been transected. After dissection of the vertebral artery from its origin off the subclavian artery to its entrance into the C-6 transverse foramen, the internal jugular vein and vagus nerve are retracted laterally for the anastomosis. Center and Right: A temporary clip is placed high on the vertebral artery, and the artery is secured with a clip and a suture ligature near its origin. The common carotid artery is briefly occluded and an ellipse is removed from its lateral aspect with an aortic punch. A partial occlusion clamp is then applied during the anastomosis. A double-ended 7-0 suture is used for a continuous stitch.

Another ship, Smyth employed a variation of Hunterian proximal ligation for treatment of the aneurysm, as recommended by Rogers, a surgeon visiting from New York. The procedure involved ligation of the innominate artery and the common carotid artery, in order to “intercept a retrograde current through [the carotid artery], which he supposed had occurred in former cases.” Fourteen days later profuse hemorrhage ensued, “causing syncope rapidly.” Smyth was struck by the rapid occurrence of syncope during a second bleed weeks later, and thought the vertebral artery was carrying blood away from the brain as collateral to the subclavian artery distal to the aneurysm. This pathway had been described in 1829 by Robert Harrison, in his text, The Surgical Anatomy of the Arteries of the Human Body. Smyth, therefore, ligated the vertebral artery. Banks survived, and worked for 10 years, at which time the aneurysm recurred. Despite ligation of the internal mammary artery and an attempt at packing the sac, he died from postoperative hemorrhage, a result of collateral flow to the aneurysm through the subscapular artery. As a principal in the “first truly original surgical work ever done in Louisiana,” the patient had become a local celebrity. Consequently, upon his death, there was great competition for his remains, between his friends who sought to bury him “in a style befitting such an illustrious personage,” and the pathologist, Souchon, who “was doggedly determined that they should not have him without dividing with me, and I wanted the lion’s share.” The lion’s share was obtained, through no small effort, and after spending a year secreted away “quietly unknown to all but myself in an old whiskey barrel filled with water and alcohol,” it was sent to the Army Medical Museum (now the Armed Forces Institute of Pathology), where it remains today, “the admiration of all who love subclavian aneurisms of the third portion.”22 Fields,20 who publicized Smyth’s report in 1970, implied that the subclavian steal syndrome would have become generally recognized decades earlier, had Smyth’s paper been published in a more widely read journal than the first issue of the New Orleans Medical Record, in the wake of the War Between the States. However, Contorni10 later noted that Smyth’s “brilliant intuitions” were indeed well disseminated, especially in Europe. His contributions subsequently became overlooked and forgotten after newer surgical techniques for aneurysms were developed near the turn of the century. Failure to recognize this syndrome earlier in this century was also “no doubt the result of our ignorance of many aspects of the development of medical science through the years.”10

Retrograde flow in the vertebral artery was first demonstrated angiographically in the Italian literature by Contorni in 1960, in a neurologically asymptomatic patient with absent left upper extremity pulses. Contrast medium injected into the right subclavian artery passed up the right vertebral and refluxed down the left vertebral into the left subclavian artery. Similar arteriographic findings in four
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patients, three of whom had symptoms of basilar insufficiency, were presented during the third Princeton conference on cerebral vascular disease in January, 1961, by Fields and Toole. The widely recognized article by Reivich, Holling, Roberts, and Toole, relating reversal of flow in the vertebral artery to cerebral ischemia, appeared in November, 1961. This paper included complete descriptions of two patients whose arteriograms were presented earlier that year by Toole at the Princeton conference. These authors are generally credited as the first to associate reversal of blood flow to brain ischemia. However, at the same Princeton conference, Rob, discussing techniques of surgical therapy for incipient stroke, had clearly recognized that the transient cerebral ischemia in one of his patients was due to right subclavian artery occlusion, which resulted in "blood being withdrawn from the cerebral circulation into the right arm when that limb was exercised." These authors were all apparently unaware of Contorni's publication in the previous year and Smyth's paper published in the previous century. In the editorial accompanying the article by Reivich, et al., C. Miller Fisher proposed the term, "subclavian steal" to describe the syndrome. Shortly thereafter, numerous cases rapidly appeared in the literature. Some authors suggested more anatomical terms, such as "brachial-basilar insufficiency syndrome." Contorni recently urged belated recognition of the original contributors, by adopting the eponym "Harrison and Smyth's syndrome," but Fisher's alliterative sobriquet still stands.

A variety of operations rapidly evolved as surgeons became aware of the syndrome. In the modern era, Rob appears to have been the first to recognize and treat, surgically, a patient with symptomatic subclavian steal. He, like Smyth, 100 years before, ligated the vertebral artery. Reivich, et al., treated one of their patients with subclavian endarterectomy, but the postoperative result was not described. The subclavian steal of their other patient was not clearly symptomatic, and his operation was directed to a carotid occlusion. During 1962, at least four papers appeared reporting experience with the new syndrome in 13 patients. Subclavian endarterectomy via sternal splitting was performed with success and failure by Mannick, et al., and Simon, et al., as well as DeBakey's group. The latter group introduced extrathoracic reconstruction in the same paper, performing a supraclavicular subclavian endarterectomy in one patient, and interposing a carotid to subclavian artery bypass graft in four others.

It is of interest that Lyons and Galbriath had previously reported the use of subclavian to carotid artery bypass grafts for treatment of common carotid artery occlusion. Blood, in that situation, is shunted directly from the subclavian artery, beyond the occluded common carotid artery, into the internal and external carotid arteries to the brain. In contrast, the carotid to subclavian bypass, when used to treat subclavian steal, directs blood away from the common carotid artery into the subclavian artery. Blood flow in the subclavian artery then must be bidirectional, retrograde to the origin of the vertebral artery, and antegrade out to an extremity.

Controversy arose, and continues, as to whether or not a carotid to subclavian steal develops after the carotid to subclavian bypass graft procedure. Intraoperative blood pressure and flow measurements in several patients suggested that this steal does not occur, but in another report a steal was demonstrated. Animal experiments designed to evaluate this point have also produced conflicting results. In one preparation of carotid to subclavian artery bypass, there was no change in proximal or distal common carotid artery blood flow with the upper extremity at rest. When exercise of the extremity was simulated by creation of a distal subclavian arteriovenous fistula, flow in the common carotid artery proximal to the graft increased, and there was no decrease in flow to the distal carotid artery. Hence, the authors believed there was no steal from arteries supplying the brain in their experimental model. However, several other animal experiments did demonstrate decreases in distal carotid artery flow under similar circumstances, particularly if there was stenosis at the origin of the internal carotid artery. Although there have been no well documented reports of symptoms of such a steal in patients, this bypass has failed to relieve vertebrobasilar symptoms in some cases. We have seen a patient whose vertigo was incompletely relieved by a carotid to subclavian bypass graft. Angiography showed the graft to be patent, but flow was preferential into the arm. There was no longer a steal down the vertebral artery, but the dye column in the vertebral artery was essentially stagnant. Thus, in regard to the brain, the hemodynamic effect of the bypass graft was hardly better than vertebral artery ligation. This problem was also found in the animal preparations in which retrograde flow in the vertebral artery was induced by subclavian artery ligation. In several animals the retrograde flow was only diminished or arrested, without becoming antegrade, upon opening the carotid to subclavian bypass graft. The appropriate index of success for this and other indirect bypass procedures for subclavian steal syndrome is, therefore, not merely equalization of brachial blood pressures, but restoration of antegrade flow in the vertebral artery. Although the carotid to subclavian bypass graft currently appears to be the most frequently used method for treating subclavian steal syndrome, these suboptimal features indicate the need for more effective extrathoracic techniques which are equally safe.

Variations on the carotid to subclavian artery bypass procedure have been reported. A more lateral distal anastomosis (to the axillary instead of the subclavian artery) is thought to be technically easier and
harmodynamically equally effective.\textsuperscript{40} Direct common carotid to vertebral artery side-to-side anastomosis is a variant of carotid to subclavian bypass that requires only one anastomosis. This procedure was facilitated by an elongated, tortuous vertebral artery in the one case reported.\textsuperscript{67} Edwards and Wright\textsuperscript{16} favored direct side-to-side or end-to-side subclavian to carotid artery anastomosis for the same reason. Mehigan, \textit{et al.},\textsuperscript{50} recommended subclavian to common carotid artery transposition, and stressed the additional advantage of  

The subclavian to subclavian artery bypass was introduced in 1968 to avoid manipulation of the carotid artery.\textsuperscript{22} Experimental studies in dogs indicate that this procedure, like carotid to subclavian artery bypass grafts, is usually effective in restoring antegrade flow in the vertebral artery.\textsuperscript{25} Criticism of the procedure relates to possible injury to the thoracic duct, and to phrenic and recurrent laryngeal nerves, and some critics believe the dissection is technically difficult.\textsuperscript{34,46} Others object to the subcutaneous location of the graft.\textsuperscript{31}

Axillo-axillary bypass was popularized as having the same benefits as the subclavian to subclavian procedure, with the advantage of being easier to perform.\textsuperscript{14,34,46} The procedure can be done under local anesthesia, as can some of the other operations, and two operating teams can complete the procedure in less than 1 hour.\textsuperscript{70} There have been isolated reports of graft occlusion, both spontaneous and as a result of ligation of the graft against the sternum.\textsuperscript{59,70} Transient problems with injury to the surrounding brachial plexus have also been reported.\textsuperscript{38,70} Herring\textsuperscript{38} raised the interesting consideration that, inasmuch as 10\% of patients with subclavian steal develop coronary artery occlusion during an extended follow-up period,\textsuperscript{21} the presternal graft might interfere with the approach to a coronary bypass operation. Nonetheless, axillo-axillary bypass has a good clinical record.\textsuperscript{38} It is supported by data demonstrating restoration of pre-anastomotic blood flow in the vertebral artery, and has a mortality approaching zero.\textsuperscript{53}

The vertebralbasilar symptoms of subclavian steal may often be relieved by treatment of coincidental carotid artery stenosis alone.\textsuperscript{21,54,64,67,79} Carotid endarterectomy presumably increases the volume of intracranial collateral flow to structures in the posterior fossa that are prone to ischemia because of the steal. Najafi, \textit{et al.},\textsuperscript{84} however, reported several patients treated with carotid endarterectomy alone whose vertebralbasilar insufficiency symptoms persisted. A second operation directed to the subclavian steal was required. His group therefore began performing, in selected patients with ipsilateral disease, concomitant carotid endarterectomy and carotid to subclavian artery bypass grafts. Their results were satisfactory.\textsuperscript{84} The operation of least magnitude for symptomatic subclavian steal is vertebral artery ligation. This was done successfully in the first two reported cases, as mentioned earlier. Despite his success, in 1964, Rob\textsuperscript{61} recommended using this "inferior" procedure only "exceptionally in poor risk patients." Twenty instances of vertebral artery ligation for symptomatic subclavian steal were found in the literature, and useful clinical information is available for 10 (Table I).\textsuperscript{3,5,7,16,31,32,46,64,68,69,72,76,80} Neurological symptoms were relieved or improved in all instances. None of the patients suffered any neurological deficit as a result of the ligation. Although in two cases radial pulses disappeared after the ligation, and in one the arm briefly appeared threatened, these signs universally cleared, and the patients regained good use of their upper extremities.\textsuperscript{7,68} The only instance of severe, albeit transient, brachial ischemia appeared in Smyth's case, reported in 1866.\textsuperscript{89} This probably occurred because adequate collateral flow to the extremity had not had time to develop. His patient, however, subsequently enjoyed normal use of the arm for 10 years.\textsuperscript{1} Only one case of persistent intermittent brachial claudication as a result of vertebral artery ligation for subclavian steal syndrome could be found in the literature.\textsuperscript{21} In some instances, preoperative symptoms of brachial ischemia were not made worse by vertebral artery ligation.\textsuperscript{5,58} Another case frequently cited in criticism of this procedure involved ligation of the subclavian artery, as well as its first four branches, including the vertebral artery, in a 1-year-old child undergoing a Blalock-Taussig anastomosis for tetralogy of Fallot.\textsuperscript{77} The comparison of acute ligation of the subclavian artery and its proximal branches in that case with vertebral artery ligation for subclavian steal syndrome due to senile atherosclerosis is inappropriate. During gradual occlusion of the subclavian artery by atherosclerosis, rich collateral circulation to the extremity develops from several sources, apart from the vertebral artery. Webb and Burford,\textsuperscript{77} discussing their unfortunate patient with tetralogy of Fallot, enumerated the many vessels that readily contribute to the upper extremity when needed. The internal mammary, thyrocervical, and costocervical arteries become the principal conduits when the subclavian artery is ligated, with the vertebral artery contributing "only a very few further collateral channels." Even when those principal branches must be ligated in addition to the subclavian artery, which the authors state, "has been done on other occasions without ill effect," the intercostal, subscapular, and lateral thoracic arteries suffice to supply the extremity.\textsuperscript{77} Although interruption of retrograde vertebral artery collateral blood flow to the subclavian artery may result in exercise-induced brachial ischemia, fear of more substantial com-
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**TABLE 1**

<table>
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<th>Author, Year</th>
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*NR = not reported; sc = subclavian; inn = innominate; CNS = central nervous system.*

promise is unfounded. Our three cases, with no significant reduction in brachial blood pressure or symptoms of brachial ischemia after vertebral to carotid artery transposition, further support this concept.

Similarly, it is not likely that brain-stem or cerebellar infarction will occur after vertebral artery ligation performed to arrest retrograde flow. In articles denouncing vertebral artery ligation as appropriate treatment for subclavian steal syndrome, authors cite famous instances of neurological disasters subsequent to vertebral artery ligation. However, all of those cases involved interruption of antegrade blood flow in the vertebral artery. The hemodynamics of that situation are diametrically opposite those of the subclavian steal syndrome. Excepting the unlikely propagation of a thrombus into the distal vertebral artery, it would be unreasonable, on a hemodynamic basis alone, to expect that ligation of a vertebral artery carrying retrograde flow would do other than improve perfusion of the posterior fossa structures. Universal improvement in vertebrobasilar insufficiency symptoms of subclavian steal syndrome by vertebral artery ligation attests to this point.

At least three real advantages favor bypass procedures that restore antegrade flow over simple ligation of the vertebral artery for the treatment of this syndrome. First, flow to the brain is greater. Second, the potential ischemic consequences of occlusive disease of the contralateral vertebral artery causing insufficient flow from the contralateral side may be avoided. Third, it is not uncommon for collateral channels to the subclavian artery to develop between the distal vertebral artery and branches of the thyrocervical trunk, as seen in our first patient (Fig. 3). These channels remain open and continue to supply collateral flow to the upper extremity after vertebral artery ligation. This persistent collateral flow, however, is compensated for by those bypass operations that restore adequate antegrade flow in the vertebral artery.

Although most of the available procedures for treatment of the subclavian steal syndrome have a good record with respect to functional result, they vary in complexity and technical difficulty. The large variety of procedures reported also suggests that the ideal operation, suitable for all patients, has not been developed. Indeed, problems with interposed synthetic or autogenous graft materials, thrombosis of these grafts, multiple or difficult anastomoses, carotid artery occlusion, and difficult or hazardous dissections, may be minimized or avoided with simpler and more direct procedures. Vertebral to carotid artery anastomosis appears to fulfill these objectives.

In 1966, Clark and Perry reported success in one case with anastomosis of the distal vertebral artery to the external carotid artery. Their patient had pre-
vously refused vertebral artery ligation. Post-operatively he was improved neurologically, and there was no reduction of blood pressure in the involved arm. Their procedure involved a fairly extensive dissection to remove the vertebral artery from the upper cervical transverse foramina in order to obtain adequate length. In 1977, Corkill, et al.12 reported conceptually similar, but seemingly difficult external carotid to vertebral artery anastomoses in two patients. In their cases the external carotid was attached end-to-side to the vertebral artery between transverse foramina.

A more proximal anastomosis was first reported by Galbriath and McDowell in 1969.14 They briefly mentioned treating vertebral origin stenosis in one patient by end-to-side proximal vertebral artery to common carotid artery transposition. They noted, parenthetically, that they treated vertebral steal (subclavian steal) with carotid to subclavian bypass grafts. Wylie and Ehrenfeld7 reported a patient with contralateral vertebral artery occlusion and stenosis at the origin of her remaining vertebral artery. They, too, transposed the proximal vertebral artery, just beyond the stenosis, end-to-side to the common carotid artery, and this procedure relieved the patient's symptoms.

Proximal end-to-side vertebral to common carotid artery transposition is a relatively simple procedure for the treatment of neurological symptoms of subclavian steal syndrome. It involves only one anastomosis, it may be done without complete carotid occlusion, and it neither requires an artificial graft, nor a separate incision and sacrifice of a vein for an autogenous graft. Direct antegrade flow in the vertebral artery is immediately established without jeopardy to the upper extremity. The remaining collateral flow from the distal vertebral muscular branches to the subclavian artery, by way of the thyrocervical trunk, is adequately compensated for by the high-volume antegrade flow in the vertebral artery. The only apparent contraindication to this bypass operation is stenosis or occlusion of the proximal common carotid artery. Such occlusion or stenosis is rare.79

The three patients we have reported are now free of vertebrobasilar and brachial insufficiency symptoms. They have retained good radial pulses with no decrease in brachial blood pressure. These results were predictable from a careful review of the literature relative to the favorable treatment of the subclavian steal syndrome by vertebral artery ligation alone. The advantages of restoring antegrade flow in the vertebral artery as opposed to simple vertebral artery ligation have been discussed. Failure to establish antegrade flow in the vertebral artery by carotid to subclavian bypass grafts has occurred both in patients and in laboratory preparations. In this regard, intraoperative blood flow measurements in the vertebral arteries were performed in two of our patients. These data confirmed retrograde flow in the vertebral artery before transposition and, subsequently, a generous antegrade flow after transposition. These findings are reported and discussed in a separate paper.8

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References

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61. Rob C: Subclavian occlusive disease and reversal of the flow in the ipsilateral vertebral artery: treatment, in


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