be recurrent emboli to the same vessel, 4) fragmentation of emboli. In this patient the probable cause of recurrent symptoms was recurrent emboli.

This demonstrates the observation made by Wells\(^9\) that there is a predilection for emboli to lodge consistently in the same arterial distribution.

References


Reactive Hyperemia for the Clinical Diagnosis of Subclavian Steal Syndrome: Report of a Case

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SUMMARY  The diagnosis of subclavian steal syndrome in a patient was strengthened by utilizing reactive hyperemia of the ipsilateral arm to reproduce the presenting clinical symptoms.

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Patient Report

A 76-year-old male was admitted to the Hennepin County Medical Center because of a pelvic fracture resulting from a syncopal episode. He had had syncopal episodes for 17 years, with an average frequency of 2–3 episodes per week. Synccope occurred mainly during moderate exertion or after a heavy meal. He could not relate his symptoms to upper extremity exercise. He described presyncopal symptoms of unsteadiness, vertigo, and visual blurring which lasted 30 seconds to several minutes.

His past medical history was significant for chronic alcohol abuse and coronary artery bypass surgery 8 years prior to admission. The patient denied having angina since the bypass operation and was on no medication. Holter monitoring showed infrequent premature ventricular contractions and occasional sinus pauses up to 1.6 seconds duration. His symptom complex, however, did not occur during Holter monitoring.

Physical exam revealed a blood pressure of 120/70 in the right arm and 70 palpable in the left arm. The heart rate was 78 and regular. The cardiovascular exam revealed bilateral carotid and subclavian bruits (R > L). The left brachial pulse was decreased in amplitude and delayed in arrival when compared with the right. Cardiac exam demonstrated a grade II/VI holosystolic murmur at the apex. The remainder of the exam, including neurological examination, was unremarkable.
An upper extremity exercise test as described by Patel and Toole\(^8\) could not be performed due to osteoarthritic involvement of the elbows and shoulders. We utilized reactive hyperemia to increase blood flow to the left upper extremity as a provocative maneuver to precipitate cerebral symptoms. A blood pressure cuff applied to the left upper arm was inflated to 200 mm Hg for 10 minutes, with the patient in the sitting position. The cuff was then rapidly deflated; within 2 minutes the patient experienced vertigo, visual blurring, and presyncope. These symptoms were coincident with clinical features of reactive hyperemia of the left arm, including warmth and a flushed appearance. These presyncope symptoms resolved when the patient assumed the supine position with partial re-inflation of the blood pressure cuff. Gradual deflation of the blood pressure cuff was then accomplished without incident.

Electrophysiological studies demonstrated normal intracardiac conduction intervals, sinus node recovery times, and sinoatrial conduction times. An archaortogram showed an anomalous origin of the left common carotid artery from the innominate artery. Both common carotid arteries demonstrated luminal irregularity of less than 20% to their bifurcations. There was complete occlusion of the proximal left subclavian artery and late retrograde filling of the left vertebral artery and, subsequently, the distal left subclavian artery.

Utilizing an extrathoracic approach, a saphenous vein bypass graph was placed between the left common carotid and distal left subclavian arteries. Post-operatively both pulse and blood pressure were equal in the upper extremities. At follow up examination 5 months after surgery, he had had no complaints of blurred vision or syncope although orthostatic vertigo without hypotension was present. The blood pressure was 130/70 and 125/70 in the right and left arm, respectively. The provocative test previously described was repeated and failed to elicit any symptoms of cerebral ischemia.

**Discussion**

Although many patients with subclavian steal may be asymptomatic, symptoms of central nervous system ischemia, particularly in the distribution of the vertebral basilar system, are capable of being produced by this hemodynamic abnormality. It is also possible for patients with subclavian steal to have other etiologies for recurrent cerebral symptoms. Hence it is important to document that the subclavian steal is an abnormality which is in part responsible for cerebral symptoms before proceeding to surgical treatment. Angiographic findings may be misleading, since dye injected in one vertebral artery may show retrograde flow in the other vertebral artery, without abnormalities of the great vessels or cerebral circulation.\(^8\) In addition, some patients with the subclavian steal syndrome have not shown the diagnostic angiographic changes in the basal state and have required provocation to show retrograde vertebral flow.\(^6\) Because of these considerations, a provocative test is useful.

Resting arm blood flow is relatively small compared to intracranial blood flow; however, blood flow to the arm can increase 8–10 fold with dynamic exercise\(^6\) and this is the basis for use of arm exercise as a provocative test for cerebral vascular insufficiency in subclavian steal syndrome.\(^8\) Although this test is useful, it is not always positive in this syndrome and some patients, such as the one described here, cannot perform the test because of physical limitations. Reactive hyperemia, induced after release of a sphygmomanometer placed around the upper arm at 200 mm Hg for 10 minutes, results in a greater increase in blood flow than that noted with dynamic exercise.\(^7\) Marshall and Mantini used this approach in conjunction with angiography to demonstrate retrograde flow in the vertebral artery in a patient with subclavian steal syndrome.\(^8\) Mozersky et al. utilized a directional ultrasonic velocity detector to demonstrate retrograde vertebral artery flow in 2 patients with innominate artery occlusion and symptomatic subclavian steal.\(^6\) An increase in retrograde vertebral artery flow could be demonstrated in both patients during the reactive hyperemia of the right upper extremity.

We utilized reactive hyperemia in our patient as a clinical provocative test to induce symptoms of central nervous system ischemia. Our patient had symptoms which were non-specific for the vertebral basilar distribution and we were concerned that their etiology may not have been related to the clinically apparent subclavian steal. Precipitation of this patient's symptoms during reactive hyperemia of the ipsilateral upper extremity, partial disappearance of symptoms after surgical intervention which abolished the subclavian steal, and failure to precipitate any symptoms postoperatively by the same provocative maneuver are all consistent with the concept that this patient's symptomatology was in part secondary to the subclavian steal. This patient's symptomatic improvement over a relatively short follow up period has been incomplete since orthostatic vertigo persists. However, failure to precipitate preoperative symptomatology with the same provocative maneuver is encouraging and may indicate that long-term symptomatic relief will be recognized.

We believe that this provocative test may be useful in the clinical evaluation of patients with suspected subclavian steal syndrome and deserves further clinical evaluation. This test may also be used in evaluating symptomatology after surgery. It is easy to perform, is not dependent upon physical abilities of the patient and may be reversed if undesirable symptoms occur. If the patient's symptom complex occurs in a severe form during the reactive hyperemia phase, it can be reversed by placing the patient in the supine position and inflating the blood pressure cuff to decrease retrograde vertebral flow. Then, the cuff may be gradually deflated.
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