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**


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

MARK SHARON, M.D., RICHARD W. ASINGER, M.D., AND MORRISON HODGES, M.D.

**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.

Stroke, Vol 12, No 3, 1981

**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. Syncope 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.

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An upper extremity exercise test as described by Patel and Toole 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 presyncopal 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. Postoperatively both pulse and blood pressure were equal in the upper extremities. At follow-up examination 5 months after surgery, he has had no complaints of blurred vision or syncope although orthostatic vertigo persists. 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.

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.
SUBCLAVIAN STEAL SYNDROME/Sharon et al.

Acknowledgment

We wish to thank Dr. David C. Anderson for his review of this manuscript. Ms. Barbara Nelson provided secretarial services for this manuscript.

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Reactive hyperemia for the clinical diagnosis of subclavian steal syndrome: report of a case.
M Sharon, R W Asinger and M Hodges

doi: 10.1161/01.STR.12.3.369

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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