Vertebral Grand Larceny

By Marvin L. Daves, M.D., and Albert Treger, M.D.

The "subclavian steal" is a clinical syndrome that has been described only recently.1-10 Patients so afflicted are usually elderly, with an atherosclerotic plaque blocking the left subclavian artery between the aortic arch and the origin of the left vertebral artery. The left vertebral artery carries blood in retrograde fashion, siphoning part of the brain's supply from the circle of Willis to fulfill the demands of the left arm. With the stimulus of exercise of the left arm, these patients may become syncopal. This syndrome focuses attention on the vertebral arterial confluence as a collateral pathway in patients with peripheral vascular disease.

An infant in whom retrograde flow of the left vertebral artery supplied blood to the left arm, the trunk, and the lower extremities, and to some extent the lungs, made us aware of the possible importance of this pathway in patients with congenital obstructive lesions of the aortic arch.

Case Report

F.G., CGH no. 189642, was the product of an uneventful gestation and delivery and appeared to be a normal 5 lb., 8¾ oz. infant at birth. At the age of 1 month, vomiting and cyanosis were first noted. He was treated at another hospital for pneumonia and congestive failure. When he failed to respond, he was transferred to this hospital at age 3 months.

The remarkable physical findings were absent femoral and left brachial pulses with a bounding right brachial pulse. The liver was down two fingerbreadths below the costal margin and the spleen was down one fingerbreadth. Palpation of the precordium revealed a lift and thrust of right ventricle and a faint systolic thrill along the left sternal border, felt best at the fourth right intercostal space. Auscultation revealed a loud, split, second heart sound, the second component being louder than the first. A third heart sound was heard at the apex. There was a grade-IV/VI harsh pansystolic murmur heard all over the precordium but loudest at the third and fourth intercostal spaces. The infant's color was described as normal, dusky, or cyanotic by various observers and seemed to fluctuate with the degree of respiratory distress. (Clinically and radiographically, varying degrees of pneumonia and atelectasis were present throughout the hospital course.) No difference in color was evident in comparing the upper and lower body.

Plain films of the chest revealed moderate generalized cardiomegaly and increased pulmonary vascularity, in addition to the pulmonary changes already mentioned. Repeated electrocardiograms showed sinus tachycardia, probable left atrial enlargement, biventricular enlargement, and right axis deviation. An electroencephalogram was not done. Cardiac catheterization demonstrated positive hydrogen curves in the pulmonary artery and right ventricle and a negative curve in the right atrium. Pulmonary artery pressure was 60 to 82/45 mm. Hg and femoral artery pressure was 70 to 75/50 mm. Hg. Oxygen saturation in volumes per cent was as follows: mid right atrium 3.85, outflow of right ventricle 7.11, pulmonary artery 9.39, femoral artery 11.03. Pulmonary flow measured five times systemic flow and pulmonary resistance at rest was 340 dynes sec. cm.-5 M². Left-to-right shunts at the level of the right ventricle and pulmonary artery confirmed the clinical impression of ventricular septal defect and patent ductus arteriosus, and a retrograde right brachial arteriogram was performed to outline the clinically apparent aortic coarctation (fig. 1). Evident from this study was the fact that circulation reached the descending aorta via an ascending route to the basilar artery and circle of Willis through the right vertebral and right (and perhaps left) carotid arteries and thence by retrograde flow down the left vertebral artery to the left subclavian artery. The origin of the latter was unusually low on the aorta. There was bidirectional flow in the left subclavian artery: toward the arm distal to the vertebral origin and toward the aorta proximal thereto. Some of the contrast medium that reached the aorta in this way shunted left to right through the patent ductus arteriosus, demonstrating the pulmonary artery. Apparently the coarctation completely obstructed the aorta proximal to the origin of the left subclavian artery.

Early in the fourth month the patent ductus arteriosus was ligated. No vestige of aorta was noted.

From the Departments of Radiology and Medicine (Division of Cardiology), University of Colorado Medical Center, Denver, Colorado.

Supported by General Research Support Grants 21 and 91, Institutional grants from the U. S. Public Health Service.

Circulation, Volume XXIX, June 1964 911
immediately proximal to the ductus. Banding of the main pulmonary artery failed to decrease pressure distal to the band. While the band was being adjusted, cardiac arrest ensued and the patient died, despite attempts at resuscitation. At autopsy the ligated patent ductus arteriosus and ventricular septal defect were confirmed. The banded pulmonary artery was large. A normal ascending aorta gave origin to innominate and left common carotid vessels but was totally absent from this point to the rather low level of origin of the left subclavian artery.

**Discussion**

In essence, this case represented a predudal or infantile coarctation. Complicating factors were the ventricular septal defect and agenesis of the aortic isthmus. Two unusual features were the low pulmonary resistance that permitted left-to-right flow through the ductus despite the predudal position of the aortic obstruction and maintenance of systemic circulation (all but the right arm and the head) via retrograde left vertebral arterial flow.

Roberts et al. have summarized 55 reported cases of complete interruption of the aortic arch. In at least 32 of these, the obstruction was proximal to the origin of the left subclavian artery. All such cases have a patent ductus arteriosus and constitute a form of predudal or infantile coarctation of the aorta. The large series of reported cases of predudal coaractations that we were able to find usually fail to mention the position of the left subclavian artery. In view of the anatomy encountered in Roberts' series, it seems likely that a significant number of patients with predudal coaractation would have a congenital collateral pathway to the descending aorta via the vertebral arterial confluence.

Edwards et al. originally hypothesized that the compatibility of isthmus obstruction with normal fetal circulation vs. the necessity for

---

*Figure 1*

Infantile vertebral "grand larceny." Right brachial artery injection. Top. The right subclavian and right vertebral arteries are filled and a little contrast medium is evident in the right common carotid artery. Early collateral filling is present. Center. Contrast material has gone up the right vertebral artery, down the left, and fills the proximal left subclavian artery. Collateral vessels from one thyrocervical trunk to the other are seen crossing the midline and a rich collateral network over the right upper lobe from the thyrocervical and costocervical trunks is beginning to fill a large bronchial artery. Bottom. Five-eighths second later, the bronchial collateral, the descending aorta, and the pulmonary artery are well demonstrated. The patent ductus arteriosus could be seen in the simultaneous lateral projection.
VERTEBRAL GRAND LARCENY

development in utero of collateral circulation to the descending aorta in patients with postductal coarctation constituted a reason for the graver prognosis of the isthmus defect. It is obvious that in evaluation of this hypothesis, the position of the left subclavian artery relative to the obstruction must henceforth be considered.

North et al.² have indicated five collateral pathways to the left subclavian artery from right brachiocephalic vessels including the vertebral confluence. In our enthusiasm for "vertebral grand larceny" we should not fail to emphasize that all these pathways may play a part in our patient's collateral circulation. The angiogram demonstrates the vertebral-vertebral communication and a network of intercommunications between branches of the thyrocervical trunks. In addition, the area of the right upper chest demonstrates a rich network of vessels from the right subclavian artery reaching the descending aorta via a large bronchial artery. Paradoxically, the congenital collateral pathway that one would expect to be well developed is not seen in our patient—or at least not on the angiogram—namely, the internal mammary, superior epigastric, inferior epigastric, and external iliac circuit. At any rate, the sum of these collateral pathways was able to maintain a pressure in the descending aorta in excess of the elevated pulmonary artery pressure. This situation is not unusual in patients with preductal coarctation. The absence of arterial pulsation in the lower body reflects the lack of a systolic thrust and does not necessarily indicate a low mean pressure. The importance of a reasonably normal mean descending aortic pressure is the possibility of a left-to-right shunt through the ductus despite its postcoarctation position. Therefore, in theorizing on the etiology of pulmonary hypertension and pulmonary arterial changes in patients with preductal as opposed to postductal coarctation, it is not reasonable to assume a right-to-left shunt through the ductus. The pressures during life in the descending aorta and the pulmonary artery and the exact nature of the collateral pathways should be established. A detailed discussion of this facet is beyond the scope of this paper but constitutes a continuing subject of investigation in this institution.

Summary

A case is presented of atresia of the aortic arch between the left common carotid artery proximally and the left subclavian artery and a patent ductus arteriosus distally. Collateral circulation including a pathway via the vertebral arteries maintained an adequate mean pressure in the descending aorta to produce a left-to-right shunt in the patent ductus, despite an elevated pulmonary arterial pressure.

References


Circulation, Volume XXIX, June 1964
Vertebral Grand Larceny
MARVIN L. DAVES and ALBERT TREGER

Circulation. 1964;29:911-913
doi: 10.1161/01.CIR.29.6.911
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1964 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/29/6/911

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/