alterations in carotid arterial velocity-time profile produced by the blalock-taussig shunt

gerald a. serwer, m.d., brenda e. armstrong, m.d., richard j. sterba, m.d., and page a.w. anderson, m.d.

summary because the presence of a systemic artery-to-pulmonary artery communication can alter blood flow patterns in both the systemic circulation distal to the communication as well as the pulmonary circulation, the effect of a blalock-taussig anastomosis on carotid arterial flow was investigated using noninvasive, continuous-wave doppler ultrasonography. thirty-seven patients without a blalock-taussig shunt (group 1) and 17 patients with a blalock-taussig shunt (group 2) were studied using an 8-mhz continuous-wave doppler velocimeter. all group 1 patients had qualitatively similar carotid arterial velocity-time profiles and continuous antegrade flow throughout the entire cardiac cycle in both carotid arteries. eight patients in group 1 subsequently underwent creation of a blalock-taussig shunt with an increase of systemic arterial oxygen pressure greater than 20 mm hg. immediately after operation, all showed marked alterations in the ipsilateral carotid arterial velocity-time profile from the preoperative pattern. six showed the flow velocity diminishing to zero by late diastole. two showed continuous retrograde diastolic flow, and developed congestive heart failure. the velocity-time profile of the contralateral carotid artery remained normal. of the remaining nine patients in group 2 studied 6 months to 8 years postoperatively, six showed diminution of the ipsilateral carotid arterial velocity to zero by late diastole and at catheterization were shown to have an adequate-sized blalock-taussig shunt. the other three patients showed continuous forward flow in the ipsilateral carotid artery and at catheterization had a markedly stenotic blalock-taussig shunt. all nine patients showed continuous antegrade flow in the contralateral carotid artery. features of the carotid arterial velocity profiles that separated groups 1 and 2 were unaffected by age or heart rate. thus, a nonstenotic blalock-taussig shunt alters the ipsilateral carotid arterial velocity-time profile and induces characteristic changes that confirm the patency of the shunt. a shunt large enough to produce congestive heart failure produces additional distinctive alterations in the carotid arterial velocity-time profiles, e.g., diastolic retrograde flow.

the presence of a systemic artery-to-pulmonary artery communication creates a situation in which the relatively low resistance pulmonary circuit is in parallel with the relatively higher resistance systemic circuit. rudolph et al. showed marked alterations in aortic and pulmonary artery flow patterns after creation of an aortic-to-pulmonary artery communication, with marked retrograde diastolic aortic flow distal to the communication and continuous diastolic antegrade aortic flow proximal to it. naturally occurring communications, such as a patent ductus arteriosus, also alter blood flow patterns significantly. cassels, spencer and denison and serwer et al. showed alterations in the descending aortic flow velocity patterns in the presence of a patent ductus arteriosus similar to those shown by rudolph et al. that were eliminated after its ligation. spach et al. demonstrated the same findings angiographically. all of these studies showed that in the aorta distal to the communication there was a diastolic "steal" present, with retrograde flow from the distal aorta to the pulmonary artery.

anastomosis of the subclavian artery to the pulmonary artery may create the same physiologic situation present in animals when an artificial conduit is placed between the aorta and pulmonary artery and in patients with a patent ductus arteriosus. in this situation, the innominate artery is proximal to the shunt and, as such, is analogous to the ascending aorta, while the carotid artery arising from the innominate is distal to the shunt and is analogous to the descending
If the same physiologic situation does exist, alterations of carotid arterial flow should exist after creation of the Blalock-Taussig shunt, with the potential for a diastolic “steal” from the carotid arterial flow. The presence or absence of such changes was investigated using the noninvasive technique of continuous-wave Doppler ultrasonography to monitor carotid arterial flow. This technique has been used to evaluate carotid arterial flow in the normal patient and in the patient with carotid arterial occlusive disease. Our purpose in this work was to describe the carotid arterial velocity-time profiles of patients who had undergone a Blalock-Taussig shunt, to determine if alterations from the normal patient exist, and to determine the ability of such alterations to assess the adequacy of a given Blalock-Taussig anastomosis. This is particularly important clinically, as the adequacy of such a shunt can often be difficult to assess, especially in the immediate postoperative period.

**Methods**

**Patient Population**

Forty-six patients, ages 1 day to 17 years, were divided into two groups, based on whether they had undergone a Blalock-Taussig shunt. Group 1 consisted of 37 patients, ages 3 days to 17 years (mean 39.1 ± 55.9 [sd] months). Twenty patients in this group had a normal cardiovascular system based upon a normal physical examination, standard ECG, standard echocardiogram, and chest roentgenogram. The remaining 17 patients had undergone catheterization and had a variety of congenital heart lesions; three with atrial septal defect, three with ventricular septal defect, one with Epstein’s anomaly, two with pulmonic stenosis, three with tetralogy of Fallot, three with tricuspid atresia, and two with pulmonary atresia. None had a proximal aortic run-off lesion, such as aortic valve insufficiency, truncus arteriosus, or aorta–pulmonary artery window.

Group 2 consisted of 17 patients, ages 3 days to 10 years (mean 32.1 ± 38 months), who had undergone creation of a Blalock-Taussig shunt. Eight of these patients were from group 1 and thus were studied before and after creation of a Blalock-Taussig shunt. The nine remaining patients had undergone a Blalock-Taussig anastomosis 6 months to 8 years previously and were not studied before creation of the shunt. All of the Blalock-Taussig shunts were performed using the subclavian artery that arose from the innominate artery. Shunt patency in patients studied before and after surgery was confirmed by a rise in the systemic arterial oxygen pressure (Pao2) of greater than 20 mm Hg in the postoperative period. Of these eight patients, three had no new murmurs while five had continuous murmurs at the time the study was performed in the immediate postoperative period. The three patients with no new murmurs developed continuous murmurs by the time of discharge from the hospital. In addition, all eight patients had prominent peripheral pulses and a pulse pressure greater than 45 mm Hg postoperatively. The nine remaining patients all underwent catheterization to assess the status of the Blalock-Taussig shunt. None of these nine patients had a proximal aortic runoff lesion.

**Doppler Technique**

An 8-MHz, continuous-wave directional Doppler velocimeter (Sonicaid) was used for all studies. Studies were performed on both carotid arteries in all patients. All patients were studied in the supine position without the use of sedation, and no provocative maneuvers such as Valsalva maneuver were performed. The carotid artery was palpated just inferior to the angle of the mandible and the transducer was positioned over the palpated pulse. The transducer was angled at an approximate angle of 45° to the long axis of the artery and maneuvered until an arterial velocity pattern was observed. The transducer was positioned such that flow toward the head produced a positive deflection and flow away from the head produced a negative deflection. The exact angle of incidence of the sound beam with the artery was not critical. The less acute the angle, the greater the signal intensity, whereas the closer the angle was to 90°, the lower the intensity. However, the absolute magnitude of the signal was not measured and the velocity tracing was not qualitatively altered by the angulation. Studies were performed the day before catheterization in patients who underwent catheterization and in the immediate postoperative period in those who had undergone surgery, often before the adequacy of the shunt was known.

The output of the Doppler receiver was presented as an audio signal as well as being processed by a zero-crossing detector. Careful attention was paid to the audio output to eliminate low-frequency artifacts produced by simultaneous imaging of venous structures. The output of the zero-crossing detector was recorded by a Honeywell LS-6 strip-chart recorder at a paper speed of 100 mm/sec. The strip-chart recorder was also interfaced with a standard M-mode echocardiogram that permitted simultaneous recording of an M-mode echocardiogram and Doppler velocity-time profile. The zero velocity was electronically simulated by the Doppler instrument and the zero-velocity position on the strip-chart recorder was indicated by a simultaneously recorded line. The zero position of the instrument was verified before and after all recordings to ensure accurate zero-velocity position. A simultaneous limb lead II ECG was recorded. A minimum of five consecutive beats free of artifacts was required before the study was accepted for analysis.

**Results**

**Group 1: No Blalock-Taussig Shunt**

The normal patients and those with an intracardiac defect had qualitatively similar carotid arterial velocity-time tracings. After the onset of QRS, velocity increased rapidly and then declined gradually...
(fig. 1). This initial decline was terminated by a second increase in velocity followed by a second decline. The magnitude of this second rise in velocity compared with the initial increase as well as the time of its occurrence with reference to time of the initial increase in velocity varied from patient to patient. It was more prominent at a slower heart rate (figs. 1 and 2). Heart rates for patients in this group ranged from 43–176 beats/min (mean 96 ± 36 beats/min). Comparison of the simultaneous M-mode echocardiogram visualizing the aortic valve and the Doppler tracing (fig. 3) showed that the initial velocity upstroke occurred after the opening of the aortic valve, and the initial decline of velocity terminated shortly before aortic valve closure. The second increase in velocity occurred during diastole.

Continuous forward flow occurred throughout systole and diastole in all group 1 patients. At no time did the carotid arterial velocity fall to zero even in the presence of a slow ventricular rate (fig. 2). The velocity-time profiles in the group 1 patients had the same characteristics as the carotid arterial flow patterns obtained by previous workers in normal patients using intravascular catheter techniques or noninvasive, continuous-wave Doppler ultrasonographic techniques. The velocity-time profiles of both carotid arteries were identical in all group 1 patients.

**Group 2: Blalock-Taussig Shunt**

All but three group 2 patients had flow patterns in the carotid artery on the same side as the Blalock-Taussig shunt that were markedly different from those in group 1 or normal patients. All eight patients studied before and after creation of the Blalock-Taussig shunt exhibited such alterations in the ipsilateral carotid arterial velocity-time profiles as early as 1 hour after operation. Heart rates ranged from 110–153 beats/min (mean 121 ± 16 beats/min) and did not alter these characteristic changes.

Continuous systolic and diastolic antegrade flow no longer was present in the ipsilateral carotid artery (fig. 4). The velocity-time profiles of the ipsilateral carotid artery in six of the eight patients showed a rapid increase in carotid arterial flow velocity after QRS, which was followed by an initial decline, as in group 1. There was a second increase in velocity followed by a second decline, which continued until the velocity reached 0. During late diastole, the flow velocity oscillated around the zero baseline. Subsequent studies during the first week after operation showed the same

---

**Figure 1.** Normal carotid velocity time profile. The arrow denotes the second accentuation of blood flow velocity. The zero indicates zero-velocity baseline. DOP = the Doppler tracing; EKG = limb lead II electrocardiogram; + = forward flow; − = retrograde flow.

**Figure 2.** Tracing from a group 1 patient with congenital complete atrioventricular block. The Doppler tracing (DOP) never reaches the zero baseline even when diastole is prolonged.

**Figure 3.** Simultaneous carotid artery velocity-time profile and M-mode echocardiogram of aortic valve. The second increase in velocity occurs after aortic valve (AV) closure. LA = left atrium; DOP = Doppler tracing.
alterations in the velocity-time profiles present immediately postoperatively.

In the remaining two of these eight patients, marked retrograde flow occurred throughout diastole in the ipsilateral carotid artery. In figure 5, the carotid arterial velocity-time profile obtained simultaneously with an aortic valve echocardiogram in one of these two patients is shown. Retrograde flow occurred throughout diastole, in marked contrast to the other group 2 patients. These patients who had continuous diastolic retrograde flow in the ipsilateral carotid artery after operation developed congestive heart failure postoperatively manifested by marked cardiomegaly, hepatomegaly, tachypnea, tachycardia and an apical diastolic rumble not present preoperatively. With the institution of anticongestive heart failure therapy, the patients' symptoms improved, but continuous retrograde diastolic flow persisted.

Of the remaining nine patients in group 2 who were studied only postoperatively, six had velocity-time profiles that decreased to zero during late diastole in the ipsilateral carotid artery. Three patients had continuous forward flow and were indistinguishable from the group 1 patients. None of these nine patients had continuous diastolic retrograde flow. At catheterization, the six patients who had zero flow velocity during late diastole were shown to have an adequate-sized Blalock-Taussig shunt, as shown angiographically and by aortic saturations greater than 83%. All six patients had hemoglobins from 14.5-16.5 g%. The three patients who had continual forward flow, i.e., velocity tracings that did not decrease to zero, were shown to have markedly stenotic Blalock-Taussig shunts angiographically with aortic saturations below 80%. These three patients had hemoglobins of 17.5-20.5 g%. These nine patients were not in congestive heart failure.

Velocity-time profiles in the contralateral carotid artery of all 17 group 2 patients showed continuous diastolic antegrade flow. Velocity-time profiles of the contralateral carotid artery were indistinguishable from the normal or group 1 pattern. No changes were seen in the contralateral carotid artery velocity-time profiles in the patients studied before and after surgery. The two patients who developed congestive heart failure also had normal contralateral carotid arterial velocity-time profiles.

Discussion

We have shown that significant alterations in the carotid arterial velocity-time profiles are induced by the presence of a nonstenotic Blalock-Taussig shunt. In the absence of such a shunt, the carotid arterial velocity-time profiles exhibit continual antegrade or forward flow throughout systole and diastole, as has been shown invasively10, 11 and noninvasively.6, 7 The alterations in the ipsilateral carotid arterial velocity-time profiles of patients with a Blalock-Taussig shunt suggest, qualitatively at least, that the degree of flow through the shunt correlates with the type of alteration present in the diastolic portion of the velocity tracing. In the presence of no shunt or a stenotic shunt, diastolic velocity remains greater than zero. In the presence of a shunt producing clinically adequate pulmonary perfusion, flow velocity reaches zero by late diastole. In the presence of a large shunt producing congestive heart failure, continuous diastolic
retrograde flow away from the brain exists. While more patients will be needed to conclude that the appearance of continuous diastolic retrograde carotid arterial flow is diagnostic of a shunt that is too large, the two patients in group 2 with congestive heart failure certainly suggest that this may be the case. The changes induced by the Blalock-Taussig shunt were apparent immediately after operation, and subsequent studies were identical, showing no alteration from the patterns recorded in the immediate postoperative period.

Flow in the contralateral carotid artery, i.e., the carotid artery on the side opposite the Blalock-Taussig shunt, was not qualitatively affected by the presence of a shunt. Continual antegrade flow during systole and diastole persisted.

As the area under the Doppler tracing is proportional to the volume of blood flow if no change in the cross-sectional area of the vessel occurs, then changes induced by the presence of the Blalock-Taussig shunt may result in diminution of net blood flow through the ipsilateral carotid artery much the same as the presence of a patent ductus arteriosus creates a "steal" from the descending aorta. This is a consequence of placing a low-impedance, high-capacitance pulmonary vascular circuit in parallel with a higher impedance, lower capacitance circuit, i.e., the cerebral vascular system. For a given perfusion pressure and flow of blood through the innominate artery, the percentage of flow through each circuit is determined by the impedance of one relative to the other. Impedance of the pulmonary circuit is a consequence of the resistance imposed at the anastomotic site, the diameter of the subclavian artery, the vascular resistance and elasticity of the lung, neuromuscular factors and left atrial characteristics.

Two patients with a Waterston shunt and two patients with a type I truncus arteriosus with large pulmonary blood flow have similarly been studied. Unlike the patients with a Blalock-Taussig shunt, these four patients exhibited diastolic velocity diminishing to zero in both carotid arteries because the site of the "steal" was the ascending aorta rather than the innominate artery, as with the Blalock-Taussig shunt.

In this study, we did not determine whether total cerebral blood flow is decreased, but the presence of a Blalock-Taussig shunt could alter blood flow patterns throughout the cerebral vascular system. This speculation concerning altered cerebral perfusion is supported by the report of central nervous system complications after the Blalock-Taussig shunt procedure. The changes in the ipsilateral carotid arterial velocity-time profiles are similar to the changes produced by conditions that do limit cerebral blood flow, such as interventricular hemorrhage and neonatal asphyxia and carotid arterial occlusive disease. This may be particularly critical in newborn infants in whom normal autoregulation of the cerebral blood flow may be lacking. While the results of this study neither prove or disprove these concerns, the possibility is raised.

We believe that the easily monitored alterations in carotid arterial velocity-time profiles induced by the Blalock-Taussig shunt are good indicators of shunt patency and, potentially, the presence of a shunt large enough to produce congestive heart failure. Although the technique has not been used intraoperatively to monitor carotid arterial flow during performance of the Blalock-Taussig shunt, theoretically it could be used in the intraoperative setting as a potential predictor of shunt adequacy. Certainly, early recognition of a large flow shunt before the presence of clinical symptoms would permit improved care in the postoperative patient, and evaluation during the intraoperative period could allow alterations in shunt size to be performed. We conclude that carotid arterial velocity-time profiles permit clinical assessment of the patient with a Blalock-Taussig shunt.

Acknowledgement

The authors are grateful to Pat Parker for her assistance in the performance of the studies.

References

15. Milnor WB: Pulmonary hemodynamics. In Cardiovascular Fluid Dynamics, vol 2, edited by Bergel DH. New York,
Torsades de Pointes: Electrophysiologic Studies in Patients Without Transient Pharmacologic or Metabolic Abnormalities

LEONARD N. HOROWITZ, M.D., ALLAN M. GREENSPAN, M.D., SCOTT R. SPIELMAN, M.D., AND MARK E. JOSEPHSON, M.D.

SUMMARY Electrophysiologic studies were performed in 21 patients who had torsades de pointes. This ventricular tachyarrhythmia, characterized by rapid (200–250 beats/min) and irregular paroxysms and progressively varying QRS amplitude and polarity, occurred in the absence of electrolyte disturbance, antiarrhythmic drug therapy or acute ischemia. The QTc interval was prolonged in seven of 21 patients. Electrophysiologic study included ventricular pacing with the introduction of one to three extrastimuli and rapid ventricular pacing. The effect of i.v. procainamide or quinidine in these patients was also studied. Torsades de pointes was inducible in 19 of 21 patients. Induced episodes closely resembled spontaneous episodes. Torsades de pointes spontaneously progressed to ventricular tachycardia with a uniform morphology in three patients and to ventricular fibrillation in four. In eight patients, procainamide or quinidine converted torsades de pointes into typical reentrant ventricular tachycardia. Our data suggest that torsades de pointes in this setting may be a rapid reentrant ventricular tachycardia closely related to recurrent sustained ventricular tachycardia and a precursor to ventricular fibrillation and sudden death.

IN 1966, Dessertenne applied the term torsades de pointes to a distinctive ventricular tachyarrhythmia. Examples of this arrhythmia had been published under a variety of names over the preceding 50 years. It is characterized by paroxysms of ventricular tachycardia at rates typically greater than 200 beats/min in which the QRS morphology shows alternating polarity in an undulating pattern so that the complexes appear to be twisting about the baseline; hence the name torsades de pointes — "twisting of the points" (fig. 1). The designation of this arrhythmia by a specific title suggests that it is a specific electrophysiologic and clinical entity because of its etiologic and therapeutic peculiarities. Although it frequently occurs in the setting of a prolonged QT interval due to hypokalemia or antiarrhythmic drugs, it can also occur without these transient abnormalities. In the present study we investigated the electrophysiologic characteristics of this arrhythmia in patients without transient precipitating factors.

Patients

The study population included 21 patients, 13 males and eight females, ages 30–72 years, in whom torsades de pointes was induced during electrophysiologic studies or occurred spontaneously (table I). All but two patients had had at least three spontaneous episodes of torsades de pointes documented electrocardiographically, either by computer-monitored continuous electrocardiographic recording in a coronary care unit or Holter monitoring during observation periods of 4–13 days. The two patients in whom torsades de pointes occurred only during electrophysiologic studies had frequent multif orm premature ventricular depolarizations and nonsustained ventricular tachycardia, and thus did not fulfill strict criteria for the diagnosis of torsades de pointes. Eleven patients had suffered cardiac arrest, with ventricular tachycardia or fibrillation documented as the mechanism. Torsades de pointes always occurred in the absence of
Alterations in carotid arterial velocity-time profile produced by the Blalock-Taussig shunt.

G A Serwer, B E Armstrong, R J Sterba and P A Anderson

Circulation. 1981;63:1115-1120
doi: 10.1161/01.CIR.63.5.1115

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 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/63/5/1115.citation

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/