Blood Flow in the Diabetic Leg

By Hendrick B. Barner, M.D., George C. Kaiser, M.D.,
and Vallee L. Willman, M.D.

SUMMARY

Femoropopliteal bypass grafting with reversed saphenous vein was performed in 47 nondiabetic and 27 diabetic extremities. Graft flow was measured with an electromagnetic flow probe before and after injection of 15 mg of papaverine into the graft. Basal flow (nondiabetic, 74 ± 6; diabetic, 76 ± 11 ml/min) was similar in the two groups. Peak flow was significantly higher in the nondiabetic group (217 ± 14 ml/min) than in the diabetic group (169 ± 18 ml/min, P < 0.05), and the outflow tract was significantly better in the nondiabetics (12.2 ± 0.5) than in the diabetics (8.6 ± 0.7, P < 0.001). When flow was related to the quality of the outflow tract, there was no significant difference between nondiabetics and diabetics. Diabetics with occlusive vascular disease have greater involvement of the tibial and peroneal arteries than do nondiabetics. Vascular reactivity is not significantly impaired, and arterial reconstruction should not be withheld on the basis of arteriolar-capillary involvement.

Additional Indexing Words:
Electromagnetic flowmeter Occlusive arterial disease Saphenous vein bypass graft

THROUGH the years, emphasis on the occurrence of severe generalized small vessel disease in diabetics has pervaded the thinking of many clinicians to such a degree that vascular reconstruction is considered, at the least, ill founded or, more commonly, unfeasible in diabetics with occlusive vascular disease. It has become clear to vascular surgeons1,2 and to others3 that, although vascular disease is more extensive in the cognate arteries in diabetics than in nondiabetics, small vessel involvement is not of such a degree as to preclude arterial reconstruction.

This report on blood flow at the time of vascular reconstruction provides physiologic data to support this position.

Methods

Femoropopliteal bypass grafting using reversed, autogenous saphenous vein was performed in a standard manner in 47 nondiabetic and 27 diabetic extremities. Patients' ages ranged from 52 to 92 years (average, 62) in the nondiabetic group, and from 45 to 78 years (average, 64) in the diabetic group. There were seven female nondiabetics and 12 female diabetics. Claudication was the indication for operation in 24 of 47 nondiabetic and four of 27 diabetic reconstructions. Rest pain or gangrene was the indication for operation in the others. Preoperative and intraoperative arteriography allowed numerical grading of the popliteal outflow tract and division into groups I, II, and III (I = 12–16, II = 7–11, III = 0–6) on the basis of decreasing outflow.4 A perfect outflow tract received a score of 16, with four points each for popliteal, anterior tibial, posterior tibial, and peroneal arteries. The duration of diabetes treated with medication averaged 9, 9, and 12 years in the three groups (overall average, 10 years).

After completion of the reconstruction, flow through the vein graft was measured with a noncannulating electromagnetic flow probe (Carolina Medical Electronics). Basal flow and peak...
In group I there were 32 nondiabetics and seven diabetics; in group II there were 10 nondiabetics and 10 diabetics; in group III there were five nondiabetics and 10 diabetics. Basal and peak flows within each group were not statistically different for nondiabetics and diabetics, nor were the outflow tracts significantly different (table 1). These data are presented graphically in figure 1 and show the decline in both basal and peak flow with decreasing quality of the outflow tract, although the numerically small samples are not ideal for statistical analysis.

Combination of the three groups provides a larger sample with statistically different outflow tracts (nondiabetics, 12.2 ± 0.5, diabetics, 8.6 ± 0.7, \( P < 0.001 \)), nearly identical basal flows (nondiabetics, 74 ± 6, diabetics, 76 ± 11 ml/min, \( P = 0.90 \)), and significantly different peak flows (nondiabetics, 217 ± 14, diabetics, 169 ± 18 ml/min, \( P < 0.05 \)) as shown in table 1 and figure 2.

Representative flow tracings (fig. 3) reveal a brisk flow response to papaverine in a
Capillary occlusion.1-3, described in nondiabetic arteries.1-3, it is higher in nondiabetic anterior tibial artery). However, when peak flow was related to the outflow tract, the difference between diabetics and nondiabetics was not significant. This response of the resistance vessels to papaverine vasodilation indicates that they are not extensively diseased and that it is the diseased outflow tract (tibial and peroneal arteries) that limits flow during vasodilation in diabetics. If the resistance vessels were severely involved, there should be no response to papaverine.

The group III grafts are of some interest in that the popliteal segment was “isolated” (occlusion of the popliteal trifurcation as well as proximal occlusion) in five out of five nondiabetics and in six out of ten diabetics, and outflow was dependent on the geniculate arteries and other collaterals. Despite the lack of conventional outflow, the basal flow (nondiabetics, 67 ± 14, diabetics, 37 ± 8 ml/min, P > 0.05) and peak flow (nondiabetics,

**Discussion**

Accumulating evidence indicates that occlusive arterial disease in diabetics is similar to that in nondiabetics at the femoral and popliteal level, although it tends to be more diffuse in diabetics.6 However, diabetics have a higher incidence of involvement of the anterior and posterior tibial and the peroneal arteries.1-3, 6 Although specific changes have been described in the arterioles of diabetics,7, 8 they apparently do not frequently result in occlusion.1, 3 Capillary basement membrane thickening is a common but not characteristic feature of diabetes.9 This is not an occlusive or flow restrictive lesion, but it may impair diffusion and contribute to diabetic neuropathy.1

Overall basal blood flow was nearly identical in nondiabetics and diabetics despite a statistically poorer outflow tract in the diabetic group. This observation is consistent with the existence of resting vascular tone (vasoconstriction) in the resistance vessels so that they, and not the outflow tract, limit flow. Peak flow was 128% greater than basal flow in the diabetics and 193% greater in the nondiabetics. However, when peak flow was related to the outflow tract, the difference between diabetics and nondiabetics was not significant. This response of the resistance vessels to papaverine vasodilation indicates that they are not extensively diseased and that it is the diseased outflow tract (tibial and peroneal arteries) that limits flow during vasodilation in diabetics. If the resistance vessels were severely involved, there should be no response to papaverine.

The group III grafts are of some interest in that the popliteal segment was “isolated” (occlusion of the popliteal trifurcation as well as proximal occlusion) in five out of five nondiabetics and in six out of ten diabetics, and outflow was dependent on the geniculate arteries and other collaterals. Despite the lack of conventional outflow, the basal flow (nondiabetics, 67 ± 14, diabetics, 37 ± 8 ml/min, P > 0.05) and peak flow (nondiabetics,
198 ± 64, diabetics, 79 ± 16 ml/min, P > 0.05) were surprisingly good in these isolated popliteal segments. Although the number of grafts studied is small, these data suggest that with an isolated popliteal segment nondiabetics have a greater capacity to develop collateral circulation than do diabetics.

These data indicate that the diabetic with symptomatic occlusive arterial disease has a vascular bed that is nearly as reactive as the vascular bed of the nondiabetic with similar arterial disease. Arterial reconstruction may not be technically feasible on the basis of cognate artery disease in a significant number of diabetics, but it should not be withheld on the basis of suspected or apparent small vessel disease.

References
Blood Flow in the Diabetic Leg
HENDRICK B. BARNER, GEORGE C. KAISER and VALLEE L. WILLMAN

Circulation. 1971;43:391-394
doi: 10.1161/01.CIR.43.3.391
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1971 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/43/3/391

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/