Conversely, initial anastomosis of the opposite iliac artery decreases the duration of ischemia of the transplanted kidney. Total duration of ischemia in our case did not exceed 20 minutes. Had the aneurysm involved the common iliac artery bearing the transplanted kidney, as in the case reported by Jebara, there would have been 2 ischemic periods, corresponding to Figures 1B and C, respectively. But these 2 periods would have been interrupted by a non-ischemic period, during which blood supply would have been restored to the graft by the natural connections of the internal iliac arteries. Therefore, the longest ischemic period incurred with our technique would never exceed 20 minutes.

In conclusion, the method associated with the minimal ischemic duration is that of first anastomosing a branch of the graft to the opposite iliac artery and reestablishing arterial flow, before performing the anastomosis of the iliac artery bearing the transplanted kidney. Only when the internal iliac arteries are not patent can this method not be used. In such a case, the method described by M. Lacombe[2]—using retrograde flow from lumbar and inferior mesenteric arteries during the proximal anastomosis—may be sufficient. Finally, temporary shunts are necessary only in the absence of collateral blood flow.

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References

1. Jebara VA, Fabiani J-L, Moulonguet-Deloris L, et al. Abdominal aortic aneurysmectomy in renal transplant patients. Tex Heart Inst J 1990;17:240-4.
2. Lacombe M. Abdominal aortic aneurysmectomy in renal transplant patients. Ann Surg 1986;203:62-8.

The above letter was referred to Dr. Jebara, who replies in this manner:

We thank Doctor Mellière for his very interesting remarks, and we accept the fact that it is possible to treat vascular lesions proximal to renal allografts without adjunctive techniques to protect the graft. However, 3 points of concern deserve to be mentioned: the difficulty of the vascular procedure and the time required to do it cannot always be judged preoperatively; the tolerance of the transplanted kidney to warm ischemia is unknown; and the quality of collateral blood flow through the pelvic anastomosis is questionable.

Moreover, in Dr. Mellière's case the aneurysm did not include the iliac vessels; hence the use of an aorto-femoral graft as was described in our paper would have allowed him to perform the whole procedure with no renal graft ischemia.

The technique we used is simple and safe, offering total security for the renal graft. It also gives the surgical team all the time necessary to perform the procedure.

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The Role of Arterial Tissue Susceptibility in Atherogenesis

To the Editor:

Your last issue carried a letter from Dr. Meyer Texon of New York University Medical Center, arguing in behalf of a hemodynamic pathogenesis for atherosclerosis: "In brief, atherosclerosis may be considered the reactive biologic response of arteries to the diminished lateral pressure generated by flowing blood" (Tex Heart Inst J 1990;17:355). My own experience and that of my colleagues, in working with canine models, has strongly suggested that the nature of the arterial tissue itself—rather than its location at branches, bifurcations, and other areas of turbulence—is a primary factor in accounting for segmental differences in susceptibility to the atherogenic process.

The predilection of certain arterial segments for developing atherosclerosis is most often illustrated in the aorta. In contrast to the abdominal segment (the site of maximal involvement), the thoracic aorta in the dog appears largely refractory to atherosclerosis; and in man, too, the thoracic segment is less susceptible to the disease process. While hemodynamic factors have been advanced by Dr. Texon[1-3] to explain this difference between the two aortic segments, our studies of arterial tissue metabolism[4] have suggested that the difference can be accounted for by a biological dissimilarity between the two segments.

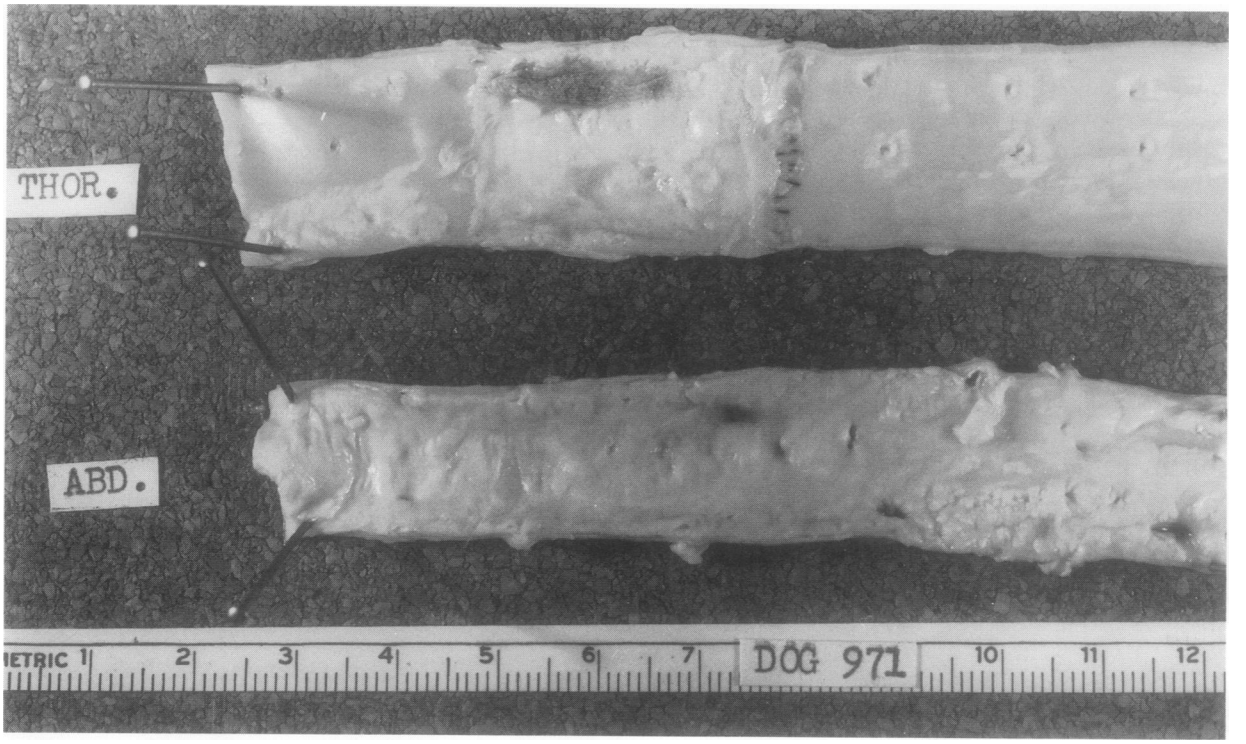

Fig. 1 *Host abdominal aorta and abdominal aortic homograft implanted into thoracic aorta of dog, shown after 11 months on cholesterol-thiouracil regimen. The average serum cholesterol level was 1547 mg/100 mL. Note massive atherosclerosis (75 to 100% of intimal surface) of both graft and host abdominal aorta, and minimal-to-moderate lesions in host thoracic aorta.*

(From Haimovici and Maier,[6] with permission.)

In other studies,[5,6] we transposed canine aortic grafts of abdominal and thoracic segments before rendering the dogs hypercholesterolemic, and learned that: 1) the abdominal implants were severely involved by atheromatous lesions, regardless of whether they were transposed to the thorax or left in place (Fig. 1); and 2) the thoracic segments disclosed minimal or no lesions, irrespective of location.

In order to exclude high arterial pressure as a factor, and to test our concept further,[7] we implanted autogenous abdominal aortic segments into the canine jugular vein, a low pressure system. The results were positive, and similar to those obtained with abdominal segments left in place or exchanged with thoracic aortic segments.

Other investigators[8] using the arterial transposition technique (Haimovici) also used dogs—but exchanged the pulmonary artery (known to be resistant to spontaneous or induced atherosclerosis) with the abdominal aorta (prone to atherosclerosis). Their results were similar to ours, i.e., no lesions in the pulmonary segments implanted into the abdominal aorta, together with atheromatous lesions in the abdominal aortic segments implanted in the pulmonary location.

Our studies of arterial tissue metabolism in dogs[4] have demonstrated differences in the enzymatic components of thoracic and abdominal aortic walls; and another research team[9] has proposed that differences in the structure and vasal supply of human aortic segments are related to the differences in their vulnerability to atherosclerotic involvement.

In no way does our concept exclude other primary risk factors in atherogenesis—especially hypercholesterolemia.

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References

1. Texon M. Hemodynamic basis of atherosclerosis. Washington: Hemisphere, 1980. 95 pp.
2. Texon M. The hemodynamic basis of atherosclerosis. Further observations: the linear lesion. Bull N Y Acad Med 1986;62(2nd series):875-80.
3. Texon M. The cholesterol-heart disease hypothesis (critique): time to change course? [editorial] Bull N Y Acad Med 1989;65 (2nd series):836-41.
4. Maier N, Haimovici H. Oxidative capacity of atherosclerotic tissues of rabbit and dog, with special reference to succinic dehydrogenase and cytochrome oxidase. Circ Res 1965;16:65.

5. Haimovici H, Maier N. Fate of aortic homografts in canine atherosclerosis. III. Study of fresh abdominal and thoracic aortic implants into thoracic aorta: role of tissue susceptibility in atherogenesis. Arch Surg 1964;89:961-9.
6. Haimovici H, Maier N. Role of arterial tissue susceptibility in experimental canine atherosclerosis. J Atherosclerosis Research. 1966;91:62-74.
7. Haimovici H, Maier N. Experimental canine atherosclerosis in autogenous abdominal aortic grafts implanted into the jugular vein. J Atherosclerosis Research. 1971;13:375-84.
8. Woyda WC, Berkas EM, Ferguson DJ. The atherosclerosis of aortic and pulmonary artery exchange autografts. Surg Forum 1960;11:174.
9. Wolinsky H, Glagov S. Comparison of abdominal and thoracic aortic medial structure in mammals. Deviation of man from the usual pattern. Circ Res 1969;25:677.

The above letter was referred to Dr. Texon, who replies in this manner:

The vascular system has both mechanical and biological properties. Mechanically, a blood vessel is a conduit for blood; its mechanical characteristics depend on its physical structure and geometrical or anatomical design. These may include caliber of lumen, bifurcation, tapering, branching characteristics, curvature, thickness, elasticity, and strength of bond between layers. The biological properties of the vascular system are concerned with the cellular life processes of the blood vessels themselves: namely, their metabolic maintenance, growth, repair, and response to noxious stimuli, including mechanical or hydraulic stress.

It is specious reasoning to believe that a curvature lesion such as I illustrate here (Fig. 1) has developed on the convex side of the vessel because the metabolic or enzymatic activity on that side differs from the metabolic or enzymatic activity on the concave side of the same segment. Furthermore, if atherosclerosis were indeed initiated by inherent biological susceptibility in certain vascular tissue segments, what would account for the greatly increased rate of intimal hyperplasia in saphenous veins transplanted to the coronary vasculature for use as bypass grafts? Surely it is plausible to attribute the stenosis of these vessels to the very different fluid mechanics in their new location; in such cases, intimal proliferation is the reactive biological response to hemodynamics. Arterial lesions can also be induced when vascular hemodynamics are experimentally altered by the surgical production of curvatures in transplanted femoral or carotid arteries in dogs.[1]

We should all agree that atherosclerosis involves first an injury to the arterial wall and second a response to injury, in the form of repair. Dr. Haimovici's research findings are directed at segmental differences in the metabolic activity of various blood vessels that appear to render the vessels susceptible to atherosclerosis. While differences in metabolic or enzymatic activity may influence the biological response to injury, they cannot be considered the primary stimulus, which is the injury itself—hemodynamically induced.

All my research findings as documented in *The Hemodynamic Basis of Atherosclerosis*[2] support and compel the conclusion that the effect of the laws of fluid mechanics is the primary factor in the localization and progressive development of atherosclerosis.

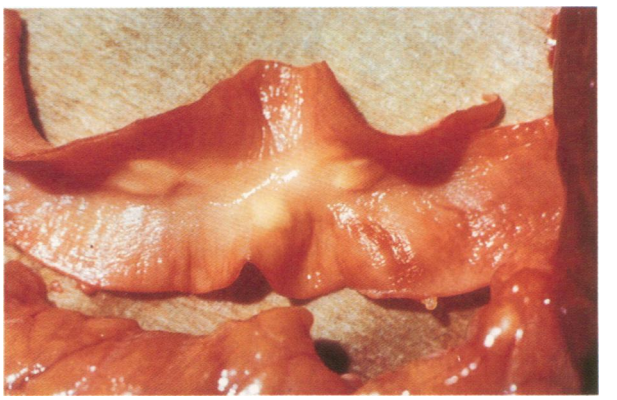

Fig. 1 *Human splenic artery. Note the atherosclerotic plaque on the convex surface of each of three curvatures.*

(From Hemodynamic Basis of Atherosclerosis,[2] *with permission.)*

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References

1. Texon M, Imparato AM, Jord JW, Helpern M. The experimental production of arterial lesions furthering the hemodynamic concept of atherosclerosis. Arch Intern Med 1962; 110:50.
2. Texon M. Hemodynamic basis of atherosclerosis. Washington: Hemisphere, 1980. 95 pp.

Letters to the editor should be no longer than 2 double-spaced typewritten pages and should contain no more than 4 references. They should be signed, with the expectation that the letters will be published if appropriate. The right to edit all correspondence in accordance with Journal *style is reserved by the editors.*