

Letters to the Editors

Arterial steal in brachiocephalic arteriovenous fistula: Could it be prevented?

To the Editors:

We read with interest the article by Papanikolaou et al¹ published in the recent issue of *Surgery*. The authors evaluated 2,422 consecutive patients who underwent 3,685 vascular access surgeries, including both arteriovenous fistulas (AVF) and arteriovenous graft (AVG) procedures. Their results revealed the superiority of autogenous accesses compared with prosthetic ones. Moreover, in diabetic and elderly patients, they recommended creation of autogenous accesses at the antecubital fossa as the first choice, which is in concordance with previous reports.^{2,3}

Arterial steal syndrome is a rare but serious complication of vascular access procedures. The choice of vascular access procedure can influence the chance of development of steal syndrome. In the study by Papanikolaou et al, the use of brachiocephalic AVF (BCAVF) as the 1st choice of vascular access was associated with the highest median primary patency among all kinds of vascular accesses; however, the rate of development of steal syndrome was 10 times greater as compared with radiocephalic AVF (RCAVF) and brachiocephalic AVF (BBAVF), a finding that was previously reported by Lok and Oliver⁴ and may adversely influence other advantages of BCAVF. In these studies, the BCAVF anastomoses had been carried out in an end-to-side fashion. We have recently introduced a new technique for creation of side-to-side BCAVFs with simultaneous perforating vein ligation.^{5,6} In these 2 studies, we performed more than 120 side-to-side BCAVF or brachial-median antecubital AVF anastomoses. Even though we used side-to-side anastomoses, none of our patients developed steal syndrome during the follow-up period. It is likely that ligation of the perforating vein prevented flow diversion from superficial veins to the deep ones via the perforating vein and resulted in reduction of arterial steal.^{7,8}

The results of our studies showed that arterial steal may be a preventable problem in BCAVF. This simple and efficient approach (ligation of the perforating vein) will reduce the rate of steal syndrome development, while it does not influence the patency rate of AVF.

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Reply to "Arterial steal in brachiocephalic arteriovenous fistula: Could it be prevented?"

To the Editors:

We read with interest the letter by Rasouli et al¹ regarding our article on the natural history of arteriovenous fistulae (AVF) and arteriovenous graft procedures (AVG) for hemodialysis.² Based on their previously reported observations, they suggest that the creation of side-to-side brachiocephalic AVF (BCAVF) with simultaneous perforating vein ligation reduces the rate of steal syndrome development, without influencing the patency rate of AVF, by preventing flow diversion from the superficial to deep veins.³

As indicated in our manuscript, for the construction of BCAVF the main trunk of the cephalic vein was anastomosed to the brachial artery. The median antecubital vein (and its tributaries) was totally resected. In addition, because further mobilization of the cephalic vein was almost always necessary, some of its more distal tributaries were ligated (and divided) as well.² This technique most likely results in a blood outflow pattern similar to that described by Moini et al.³

In our series, the incidence of moderate (arm cramping with exercise) or severe (poorly palpable radial pulse and pain) steal syndrome for a dialysis patient with BCAVF was 0.10 per patient-year,² that is, an incidence of 1% for any BCAVF that is functional 10 years after initial

construction. On the other hand, the incidence of mild (paresthesias) steal syndrome was a little bit >10 times higher (data not presented), or approximately 10% for any BCAVF that is functional 10 years after initial construction. This is very similar to the 8.95% incidence reported by Moini et al.³ At this point, we should stress the fact that lifelong clinical surveillance for the identification of steal syndrome is required, because it may develop quite late in autologous BCAVF, within 2 (or even more) years.⁴

It is true that the incidence of clinically significant steal syndrome in both our manuscript and that by Moini et al is of the lowest reported to the English literature.⁵ However, one should not immediately attribute these satisfying results to the routine ligation of the antecubital vein tributary. Indeed, in more than half of the cases, finger gangrene in hemodialyzed patients is often the result of atherosclerosis and is unrelated to the function of a fistula.⁶ Moreover, the size of the diameter of the anastomosis is directly associated with the incidence of steal syndrome.⁵ Perhaps the recommended anastomotic size of 6–7 mm is “too much” under certain circumstances (eg, diabetes,⁶ age >60,⁷ female gender,⁸ lupus⁹), leading to the development of clinically significant steal syndrome.

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Pathophysiology and treatment of the systemic inflammatory response syndrome from the perspective of evolutionary medicine

To the Editors:

As suggested in the November 2009 issue of *Surgery* by Lyte,¹ there is increasing evidence in animals indicating that afferent fibers of the vagus nerve transmit intraperitoneal information such as an infectious “threat” to the brain. Little is known, however, about the role of the vagus nerve in the crosstalk between the gut and the brain in humans. We were unable to show the influence of vagotomy on the extent of fever and acute-phase protein response in patients undergoing gastric cancer surgery.² The efferent vagus nerve has been implicated as an important anti-inflammatory pathway in animal models of sepsis and hemorrhagic shock.^{3,4} Although Lyte¹ mentions that, on the basis of evolutionary principles, the brain must initiate an appropriate response to information from the “leaky” gut, it may be difficult to explain the reason why the efferent vagus nerve sends an anti-inflammatory signal from the perspective of evolutionary medicine. I agree with the usefulness of evolutionary concept for a better understanding of the systemic inflammatory response syndrome (SIRS), but I have some concerns about the statement that current therapy, such as the use of inotropic agents and antibiotics, may represent a continuous infectious threat that is responsible for an ever-escalating inflammatory response leading to unfavorable outcomes.

SIRS is generally manifested by at least 2 of the following 4 systemic responses: hyperthermia, tachycardia, tachypnea, and leukocytosis. Sepsis is defined as the combination of SIRS and infection, and septic shock is severe sepsis associated with acute circulatory distress. Because the causes of SIRS are disparate, there are considerable differences in the disease severity among SIRS, ranging from mild SIRS, in which 2 disease criteria are met on the date of admission with a resultant mortality of 6%, to septic shock, with a mortality rate of 46%.⁵

Severe infectious SIRS is inevitably associated with capillary leakage and tissue hypoperfusion, and so the initial treatment is fluid resuscitation that helps restore cardiac function. If fluid resuscitation fails to restore sufficient perfusion and perfusion pressure of vital organs, vasoactive drugs such as catecholamines must be used to restore these, although these drugs can potentially decrease further splanchnic blood flow.⁶ What is functional from an evolutionary perspective is not necessarily functional from the perspective of patients.⁷ The appropriate treatment of SIRS may not be deducible from evolutionary considerations.

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