LFCA was anastomosed on the right coronary artery in 10 cases, the first marginal branch of the circumflex artery in 5, the first diagonal branch in 5, the LAD in 3, the intermediary artery in 1, and the posterior descending artery in another case. In 21 cases, proximal anastomoses of the descending branch of the LFCA were carried out on the ascending aorta, and in 4 cases they were carried out either on the pedunculated right ITA or left ITA, forming a Y-shaped graft. The average length of the descending branch of the LFCA was 16 cm (range 14-20 cm). Clinical follow-up was complete for all patients with a mean follow-up of 8 months (range 2-26 months). One patient who was operated on on an urgent basis with an ejection fraction of 20% died of irreversible left ventricular failure on the fifth postoperative day. A postoperative coronary angiogram was repeated in 4 patients, and in only 1 of them was the descending branch of the LFCA found to be occluded. The other patients continue to be followed-up by periodic treadmill tests and remain clinically free of symptoms. During the beginning of our experience with the descending branch of the LFCA, we observed that this artery was unsuitable for use in some cases. The presence of a poorly developed artery (Fig 2A) and of macroscopic atheromatous lesions in the artery itself preclude its harvesting. For these reasons, we considered it necessary to perform bilateral femoral arteriography before surgery, during coronary angiography, for patients in whom the use of the descending branch of the LFCA might be justified. Arteriographic evaluation provided further information on the anatomic variations of this artery such as the LFCA arising from the common femoral artery or a hypoplastic LFCA being sometimes replaced by small branches taking their origin from the deep or superficial femoral arteries. The anatomy of the LFCA can be different between the right and left sides in the same person. Moreover, radiologic evaluation allows the detection of some occlusive or stenotic lesions of the superficial femoral arteries for which the descending branch of the LFCA contributes to the collateral circulation of the limb (Fig 2B). Histopathologic examination of the harvested descending branches of the LFCA revealed that this artery can contain macroscopically occult atheromatous lesions, sometimes even with microscopic calcified deposits, which could result in earlier occlusion of the graft after the operation.

In conclusion, although we totally agree with the conclusions reported by the authors regarding this new arterial conduit, we would like to emphasize the importance of a preoperative bilateral arteriographic evaluation of the femoral arteries and that of LFCAs, as we systematically do for thoracic arteries. We believe that this evaluation can avoid useless attempts at harvesting. Attractiveness of the descending branch of the LFCA as an arterial conduit will depend on its patency rate in the long term and the enthusiasm of surgeons to use it.

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12/8/97601

A successful treatment of serous leakage from a polytetrafluoroethylene Blalock-Taussig shunt with intravenous fibrinogen administration

To the Editor:

Occasionally, excessive serous fluid leakage occurs as a complication after the modified polytetrafluoroethylene (PTFE) Blalock-Taussig (B-T) shunt. The estimated frequency of this complication is reported as approximately 20%,[1,2] but the mechanism is not clearly defined. We present a case of serous leakage through a modified B-T shunt treated with intravenous fibrinogen administration and describe our hypothesis about the role of fibrinolysis in this problem.

A 3-year-old boy (11.5 kg) with single right ventricle and pulmonary stenosis was admitted for the treatment of cyanosis and exercise intolerance. A blood test revealed polycythemia with a hemoglobin level of 20.8 g/dL. We decided not to proceed directly to a Fontan procedure because of the lack of direct measurement of pulmonary arterial pressure and pulmonary vascular resistance (the catheter did not go through the narrow ventricular outflow tract). As a preparation for a Fontan-type procedure, the boy underwent a left modified B-T shunt with a 5-mm PTFE shunt. Anticoagulation was initiated on the first postoperative day with a continuous heparin infusion (200 units/kg per day; Novo Heparin, Hoechst Marion Roussel, AG, Frankfurt, Germany) and an oral administration of ticlopidine hydrochloride (5 mg/kg per day; Panaldine, Dai-Ichi, Inc, Tokyo, Japan). On the third postoperative day, the chest tube drainage increased appreciably and the anticoagulation therapy was discontinued. The drainage continued for more than a week, with excessive serous fluid losses averaging 700 mL daily. The blood test revealed a marked decrease in total plasma protein (4.2 g/dL) and plasma fibrinogen (93.6 mg/dL) levels. The massive leakage continued and plasma protein and fibrinogen did not increase despite the infusion of packs of frozen plasma. We administered fibrinogen (heated and freeze-dried human fibrinogen; Fibrinogen HT, Yoshitomi, Inc, Osaka, Japan) intravenously, 200 mg/kg per day, for 2 days. The leakage stopped instantly and the fibrinogen level increased to more than 300 mg/dL. The chest tube was removed and the patient was discharged after a catheterization. Anticoagulation therapy was never resumed.

Three weeks later, the boy was readmitted with dyspnea, chest pain, and increasing cyanosis. A chest x-ray film revealed a massive pleural effusion on the left side, and a chest tube was inserted. Chest drainage continued for several days, with excessive serous fluid losses and decreased plasma protein (4.4 g/dL) and fibrinogen (71.3 mg/dL) levels. Fibrinogen (200 mg/kg per day for 2 days) was administered again, and the fluid leakage stopped completely within 2 days. On the basis of a high D-dimer level (736 ng/mL) detected after the second fibrinogen administration, we speculated that the fibrinolytic system had been greatly amplified in this cyanotic patient. Therefore, we started an oral medication of tranexamic acid (Transamin-G; Dai-Ichi, Inc, Tokyo, Japan) 30 mg/kg daily as an antifibrinolytic therapy. Additional prophylactic fibrinogen infusion was achieved twice at 2-week intervals. Since then, the plasma fibrinogen level has been within normal range (no less than 170 mg/dL), and the plasma D-dimer level has been maintained at a relatively lower level (200 to 600 ng/mL), leaving the patient in stable condition without any fluid leakage. Neither thrombocytopenia nor bleeding tendency was observed. No thrombotic events occurred. A bidirectional Glenn shunt was successfully done 6 months after the B-T shunt.

LeBlanc and associates[2] first described excessive fluid leakage after a modified B-T shunt. Bolton and Cannon[3] suggested that rapid wetting of the graft with organic solvents or a high blood flow with faulty formation of fibrin may be responsible for the leakage. However, the mechanism still remains unclear. In most of the cases previously described, conservative management failed and surgical interventions were required. Maitland and coworkers[4] reported 2 cases in which leakage was controlled with intraluminal fibrin glue. Noyez and Daenen[1] reported the case of a patient treated with fibrin glue and collagen fleece wrapping. Intravenous fibrinogen administration was first described by Suzuki and associates[5] as an effective conservative management of the leakage. Human fibrinogen products were originally developed for congenital fibrinogenopenia and are commercially available in Germany and Japan. According to their report, 7 patients were successfully treated with fibrinogen administration (150-200 mg/kg per day for 3 or 4 days), and in 2 of those cases the plasma fibrinogen level was much lower than the normal range at the beginning of the leakage. As we described, the fact that plasma fibrinogen level decreased and plasma D-dimer level increased at the beginning of the fluid losses may be an important clue to the mechanism of leakage. We speculate that the fibrinolytic system could be greatly amplified in some cyanotic patients with polycythemia against great thrombotic tendency owing to high blood viscosity. A probable explanation is that the fibrin cross-linking step and newly formed fibrin clots in a PTFE graft wall could be easily impaired and degraded when this overwhelming fibrinolytic regulation occurs. Once the strong fibrinolysis begins, a large amount of fibrinogen may need to be supplied until newly formed cross-linked fibrin is tightened and subsequent clot formation is completed. Platelet function of patients with polycythemia may play another important role in this problem. In our preliminary data of the patients, very weak aggregation and poor α-granule release response have been observed. We hypothesize that both strong fibrinolytic response and weakened platelet function could be the physiologic adaptation phenomenon against the great thrombotic risk. Intravenous fibrinogen administration is effective and the combination of fibrinogen and antifibrinolytic therapy may be the best conservative management available for serous leakage through PTFE so far. Further investigations are needed to prove our hypothesis.

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Aortic function in patients during intra-aortic balloon pumping determined by the pressure-diameter relation

To the Editor:

Stefanadis and associates[1] provide an important analysis of aortic function in patients during intra-aortic balloon pumping, which aids in the understanding of the improved circulatory function that results from its use. However, two points need further clarification.

First, in Fig 4 they state that the fourth derivative of the aortic pressure and diameter was used to estimate (1) minimum aortic pressure and diameter and (2) the point of inflection of the aortic pressure and diameter, thus allowing calculation of the pressure augmentation index. The fourth derivative has been previously described as the optimum way of deriving these points.[2]

The use of the fourth derivative is against the basic principles of calculus, in which the first derivative is used to evaluate minima and maxima, with the second derivative used to distinguish between these and points of inflexion, negative at