hemostatic disorder. However, while there is a general understanding of the fibrinolytic response to CPB, little has been reported on the relationship between the type of cardiac surgical operation and the intensity of the changes observed in the fibrinolytic system. The aim of this preliminary study is to determine whether the modality of open heart surgery influences the magnitude of these changes.

We have prospectively analyzed the plasma levels of tissue-type plasminogen activator (tPA), plasminogen activator inhibitor type 1 (PAI-1), and fibrinogen and fibrin(ogen) degradation products (FDP) in 69 patients who underwent heart surgery with CPB (35 valve replacements: 19 aortic, 9 mitral, 7 both aortic and mitral; and 35 coronary bypass) and in 55 control subjects (surgery without CPB) matched for sex and age. CPB operations were all done in elective fashion and none of the patients were in congestive heart failure before surgery. Blood samples were collected three times: before surgery, and on postoperative days 1 and 7. We did not find differences in the plasma concentrations of tPA between CPB and control groups, and there were no changes in tPA levels after CPB compared with levels before surgery. However, levels of PAI-1 were significantly higher in the CPB group even before surgery (p<0.05) and on postoperative days 1 (p<0.0005) and 7 (p<0.005) compared with the control group. Among the subjects of the CPB group, the concentration of PAI-1 increased significantly on postoperative days 1 (p<0.0005) and 7 (p<0.005). A significant increment in fibrinogen and FDP levels was observed only on day 7 (p<0.01 for each). When valve replacement and coronary bypass groups were compared, the persistence of an elevated PAI-1 level on postoperative day 7 was observed only in the first group (p<0.025), while it returned to normal in the second (Fig 1). Duration of surgery was similar in the valve replacement and coronary bypass groups (274±80 vs 280±109 min). The median duration of the CPB procedure was 104±52 min, and duration of ischemia ranged from 21 to 91 min. There was no association between changes in PAI-1 levels and duration of CPB or ischemia. Two patients in the CPB group and five control subjects (no statistically significant difference) developed a venous thromboembolic event in the postoperative period, but only in the CPB group was it related with a higher PAI-1 plasma concentration (p<0.001). These two CPB patients were in the valve replacement subgroup.

A prior study has shown that an increase in tPA during CPB (occurring intraoperatively, within the first 30 min of CPB) is associated with valve surgery to a greater extent than coronary artery bypass grafting.9 We have not measured fibrinolytic parameters during surgical intervention. However, our study demonstrated that the postoperative increase in PAI-1 is more durable in patients who undergo valve replacement than in those undergoing coronary bypass. Biologic mechanisms that would explain this difference are unclear to us; perhaps more intense endothelial damage occurs secondary to surgical technique in valve replacements. On the other hand, elevated PAI-1 levels postoperatively are associated with an increased risk of venous thromboembolism.7 Curiously, in our study, those patients in the CPB group who had a thromboembolic event in the postoperative period had a significantly higher PAI-1 plasma concentration. In conclusion, it seems that the valve replacement modality of cardiac surgery is associated with more durable changes in the fibrinolytic system than the coronary bypass modality, and this contingency may have clinical consequences. Certainly, further studies are needed to elucidate the mechanism and the clinical significance of these findings.

J. M. Baga Sánchez, MD
J. M. Rodríguez Martín, MD

Servicio de Hematología y Hemoterapia
Hospital Universitario de Canarias
Universidad de La Laguna
Tenerife, Spain

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The Case for Troponin T

To the Editor:

In the January 1997 issue of CHEST, Brown and Bertoloté make a compelling argument for the use of troponin rather than creatine kinase (CK) as the marker of choice for detecting myocardial injury. I agree, but I must differ with several issues leading them to conclude that troponin I is more sensitive than troponin T. Specifically, Brown and Bertoloté state that there has been no head-to-head comparison of the two troponins, and that troponin T is appropriately elevated in patients with end-stage renal disease who show no signs of myocardial disease.1 Recently, two reports have been published refuting their arguments—possibly the authors were not aware of these reports when writing their article.

CHEST / 112 / 3 / SEPTEMBER, 1997 857

Figure 1. Evolution of PAI-1 activity levels compared with valve replacement and coronary bypass patients. AU=arbitrary units.
Ohman et al did compare troponin T to troponin I as predictors of adverse cardiac events in more than 600 patients with the acute coronary syndrome. Using receiver-operator curves and a multiple regression model, they concluded that troponin T had a greater sensitivity than troponin I in detecting patients who would eventually have a myocardial infarction and/or die within 30 days. Furthermore, the concern that troponin T is inappropriately elevated in patients with renal failure has been clarified and rejected in an abstract by Haller et al. They assessed the cardiac troponin T levels in 97 patients with renal failure and classified them into three groups: 22 with coronary artery disease proved by angiography or prior myocardial infarction, 40 with two or more recognized risk factors, and 25 with no risk factors. In each, the troponin T value was correlated with cardiac risk: mean±SD values were 0.26±0.08 ng/mL, 0.23±0.06 ng/mL, and 0.07±0.02 ng/mL for the three groups, respectively. Increased troponin T in patients with renal disease was a function of severity of cardiac risk and most likely indicated minimal cardiac injury in this patient population.

Another benefit of troponin T that has not been discussed is the low clinical cutoff (0.1 ng/mL) of troponin T assays, allowing detection of even minor infarcts. Cutoffs for troponin I assays vary by manufacturer and are often too high to detect micro-infarcts, and lower cutoffs can result in false positives. According to Ohman et al., troponin T has been shown to be an excellent prognostic indicator of adverse cardiac events in patients with unstable angina, due to its analytical sensitivity at very low concentrations.

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1 Brown CS, Bertolet BD. Cardiac troponin: see ya later. CK. Chest 1997; 111:2-4

To the Editor:

We thank Dr. Statland for his comments. We have examined the abstract by Haller et al. According to the standardized troponin T test, each group had elevated levels of troponin T. However, according to a new more specific enzyme-linked immunosorbent assay (ELISA), patients with proven coronary artery disease (CAD) and patients with two or more cardiac risk factors but without CAD had elevated levels. Therefore, in patients with and without CAD and chronic renal failure, cardiac troponin T is unable to distinguish patients with acute myocardial injury, which is the purpose of the test.

In the abstract by Ohman et al., both cardiac troponins T and I were helpful in stratifying patients with acute coronary syndromes by risk. In order to make further comments regarding the comparison of troponins I and T performed by these investigators, we will wait for peer review and publication of the data.