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AORTIC ARCH INTERRUPTION WITHOUT DUCTUS ARTERIOSUS AND NO VENTRICULAR SEPTAL DEFECT

To the Editor:

We read the interesting case report by Cazavet and associates¹ on “an infant with a previously unrecorded type of aortic arch interruption.” However, similar types of aortic arch interruption without ductus arteriosus and without ventricular septal defect, although rare, have been previously described since 1964.²⁻⁶ The common features of these unusual patients were the longevity up to adolescence with no severe symptoms and the presence of extensive collateral circulation supplying the descending aorta.²⁻⁶ Surgical treatment was also reported in relation to the various anatomic patterns.²⁻⁶ In the field of pediatric cardiology, sometimes some old scientific papers can represent important sources of knowledge.

The interesting new aspect described by Cazavet and associates¹ is the association with del 22q11 and the relative embryogenetic implications that expand the spectrum of cardiovascular defects of this syndrome.

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RECOGNITION AND MANAGEMENT OF HEPARIN-INDUCED THROMBOCYTOPENIA IN PATIENTS AFTER CARDIAC SURGERY

To the Editor:

In a recent publication, Demma, Paciullo, and Levy¹ evaluated the recognition and treatment of heparin-induced thrombocytopenia (HIT) in patients after cardiothoracic surgery using the direct thrombin inhibitor (DTI) argatroban. We agree that the detection and management of HIT in patients who have had cardiac surgery is of clinical importance, and we have

used a similar method of HIT identification and treatment with the DTI bivalirudin.

Our familiarity with the use of bivalirudin was heightened after our participation in the CHOOSE-ON trial published in 2007, which evaluated the use of this DTI during cardiopulmonary bypass in patients having cardiac surgery.² Since then, we have standardized our method of identifying HIT patients postoperatively and treating them with bivalirudin. To detect HIT, we use a method similar to that of Demma, Paciullo, and Levy¹ in which patients who score in the moderate range on the 4-Ts schema³ are subjected to a platelet factor 4/heparin immunoassay. Occasionally, we will also confirm the diagnosis with a serotonin release assay. To then distinguish HIT from hemodilution or other causes of thrombocytopenia, we also categorize the patterns of platelet counts as outlined by Poupard and colleagues.⁴

Our standardization of detection methods did not alter the number of patients who received a diagnosis of HIT (14.5% compared with 15.1% before 2007). However, we did see an increased rate in the use of bivalirudin to treat thrombocytopenia (23.1% compared with 17.5%) (Table 1). This suggests that our standardization procedures may have resulted in earlier identification and medical management of HIT.

TABLE 1. Characteristics of patients treated with bivalirudin after cardiac surgery

	Before (2004-2006)	After (2007-2010)
Thrombocytopenia	15.1% 463/3065	14.5% 476/3288
DTI use in patients with thrombocytopenia	17.5% 81/463	23.1% 110/476
HITS (per decreased platelets)	4.3% 20/463	5.5% 26/476
HITS (per total adult patients)	0.7% 20/3065	0.8% 26/3288
HITS mortality	60.0% 12/20	23.0% 6/26

Thrombocytopenia is defined as a 50% decrease in platelets from preoperative baseline by 24 hours after cardiopulmonary bypass. Mortality is defined as death within index admission. DTI, Direct thrombin inhibitor; HITS, heparin-induced thrombocytopenia and thrombosis syndrome.

Interestingly, whereas there did not appear to be a difference in the incidence of HIT and thrombosis syndrome (HITS), we did find that mortality owing to HITS decreased from 60.0% to 23.0% after altering our HIT detection and treatment procedures (Table 1). Together, these findings suggest that the standardization of the methods for earlier identification and management of HIT with bivalirudin improved the mortality rate among patients diagnosed with HIT after cardiac surgery.

The article by Demma, Paciullo, and Levy¹ has made an important contribution by raising awareness of methods used to identify and treat HIT with argatroban in patients who have had cardiac surgery. Combined with our aforementioned findings using bivalirudin, we can conclude that the early detection and management of thrombocytopenia with DTIs in the patient who has had cardiac surgery is an important clinical consideration in improving patient outcomes.

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ASSOCIATION OF CATHETERIZATION WITH ACUTE KIDNEY INJURY IN THE CARDIAC SURGICAL PATIENT To the Editor:

In a recent publication, Andersen and colleagues¹ concluded that cardiac catheterization within 1 to 3 days before proximal aortic surgery is not associated with an increase in postoperative acute kidney injury (AKI). Although our own interest in the occurrence and duration of AKI has lain primarily with patients undergoing coronary artery bypass grafting,^{2,3} we are conscious of special circumstances in which the postoperative AKI rate does not appear to be affected, such as for same-day cardiac catheterization and aortic valve surgery.⁴ Despite our knowledge of these special circumstances, we wish to caution readers to pay close attention to the limitations of the study by Andersen and colleagues,¹ limitations that they themselves have clearly detailed in their article but have not reflected in their title.

Of particular interest are the effects of contrast media on AKI in cardiac surgical patients. In their analysis, Andersen and colleagues¹ demonstrated that patients who received contrast media within 3 days before proximal aorta surgery actually had a lower rate of postoperative AKI than did patients who were exposed between days 4 and 54 before the operation. In contrast, we have recently demonstrated that the incidence of postoperative AKI was significantly increased when cardiac catheterization was conducted within the same hospital admission as cardiac surgery.² Although we do not have direct evidence to support a role for contrast media in the occurrence of AKI in this model, a previously published study has shown that exposure to contrast media may augment necrosis and renal tubular cell apoptosis.⁵ We therefore agree with Andersen and colleagues¹ regarding the importance

of careful preoperative evaluation of the risks of contrast exposure in the cardiac surgical patient.

Fortunately, Andersen and colleagues¹ were thorough in emphasizing the limitations of their findings, in that they discussed the possibility that variability between groups could have confounded their results. Specifically, the outcomes may have been influenced by the presence of known risk factors for AKI in the long-interval catheterization group, such as age, body mass index, and congestive heart failure. In addition, Andersen and colleagues¹ discussed differences in the catheterization protocols, noting the limited use of contrast media and increased intravenous hydration in the short-interval catheterization group relative to the long-interval group. These differences could have affected the significance of their findings, and we therefore believe that additional, well-designed studies are needed for further delineation of the ideal time frame between catheterization and surgery.

Although there are strong motivations for reducing the time between cardiac catheterization and surgery, such as reducing hospital costs, patient inconvenience, and physician anxiety about a potentially life-threatening disease, the long-term implications of postoperative AKI with respect to reduced patient survival remain of the utmost concern.³ The article of Andersen and colleagues¹ provides interesting insights into the management of patients between catheterization and cardiac surgery; however, the confounding factors in their study encourage the development of further investigations to delineate the exact time needed between contrast media exposure and the initiation of cardiac surgery. Future studies of this nature may aid in determining the best methods to ensure the lowest possible risk of AKI and provide the best chance for