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Cardiac surgery, cardiopulmonary bypass, and preoperative renal dysfunction

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Cardiac surgery, cardiopulmonary bypass, and preoperative renal dysfunction

Editor—We read with interest the paper by Lofe and colleagues.¹ This complex study adds new information to the subject and the authors should be congratulated for their work. We wish to add some comments and ideas to the discussion. Our group published, in 1998, a study of elective coronary patients with abnormal preoperative renal function undergoing surgery with cardiopulmonary bypass (CPB),² using ‘gold standard’ techniques to measure glomerular filtration rate (GFR) and effective renal plasma flow (ERPF). Further studies by our group have been carried out in children and patients with valvular disease undergoing surgery with CPB, with similar methodology. Our study, however, was intended to focus only in the preoperative period. It is difficult to study cardiac surgery patients after operation, due to the many variables involved, such as changes in haemodynamic variables, use of vasoactive drugs, changes in haematocrit, and bleeding. Thus, the results presented¹ are speculative. It is interesting that GFR remains within normal values in patients with preoperative renal dysfunction during both studies,^{1,2} suggesting that modern techniques for CPB management are able to protect renal function. It also refutes earlier work, suggesting that CPB reduced the GFR in all patients. ERPF increased during CPB in all our earlier studies in coronary patients. In recent studies on valvular and paediatric patients, ERPF remains within baseline values throughout the study. This difference could be related to new strategies during CPB: higher perfusion pressures, vasopressor drugs, and higher haematocrit. We disagree with the authors regarding the explanation for changes found in filtration fraction (FF). We showed in our work that during CPB (hypothermia), FF decreased. FF has been used as an index of vasodilatation or vasoconstriction of the efferent arteriole. The reduction of FF most likely represents a vasodilatation state, due to hypothermia, low viscosity, or the release of vasoactive endothelial factors. A high FF in the postoperative period could represent vasoconstriction with unknown consequences for renal function, thus the significance of this finding is unclear. CPB has been blamed for many years for renal dysfunction in cardiac patients; however, in most of the recent trials, CPB has not been an independent risk factor for perioperative renal failure.³ Interestingly, studies with off-pump surgery have shown the same incidence of renal dysfunction in patients with preoperative abnormal renal function.⁴ We agree with the authors that hypothermia seems to be the best protective intervention in these

patients, mainly due to reduced metabolism. Drugs that increase blood flow non-specifically have not shown benefit so far.

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Editor—We thank Dr Lema and colleagues for their comments and ideas on the subject discussed. Their remarks concern both the absence of change in GFR and the mechanism of change in FF. With respect to GFR, unfortunately, only a limited number of small studies evaluating the effect of cardiac surgery with CPB on renal function using gold standard techniques have been reported. However, recently Witczak and colleagues⁵ studied the effect of nifedipine infusion on GFR in patients ($n=20$) with impaired renal function undergoing CPB surgery. GFR was measured as the plasma clearance of ⁵¹chromium-ethylene-diamine-tetraacetic acid before operation and 48 h after operation and creatinine clearance was measured before operation and 0–4, 20–24, and 44–48 h after operation. The authors found no statistically significant change in the GFR or in creatinine clearance over time within, or between, groups. This study supports our observation that modern techniques for CPB management are able to protect renal function in patients with preoperative renal dysfunction.^{1,2} With respect to FF, we agree that interpretation of our data as to the cause of the increased FF after CPB remains speculative. In patients, with normal renal function (plasma creatinine <1.5 mg dl⁻¹), undergoing coronary surgery, Lema and colleagues⁶ found an abnormally elevated FF before operation, and a significant decrease during bypass, which returned to abnormally elevated baseline values 1 h after operation FF. The increased FF we observed on day 7 after operation may thus represent an extension of the suggested vasoconstrictive state, with unknown consequences to renal function. However, it may also represent a structural remodelling of the vasculature, particularly in our patients with modest impairment of renal function. Until now, renal function studies in cardiac surgical patients are limited to the

hospital period and no long-term follow-up is available. Renal function measurements in cohorts of cardiac surgical patients should extend the hospital period to provide insight into the mechanism of changes in renal parameters, and the possible bearings on the long-term effects of cardiac surgery with CPB on renal function.

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Renal oxygen delivery during cardiopulmonary bypass

Editor—I read with interest the study by Loeff and colleagues,¹ on renal function in selected patients undergoing cardiac surgery with cardiopulmonary bypass (CPB). It is reassuring to discover that there was no significant evidence of deterioration in glomerular filtration rate (GFR). A deleterious patient or treatment characteristic could theoretically have caused a significantly lower GFR after operation and there was an attempt to select out such patients. I am nevertheless concerned for several reasons about the omission of data on oxygen delivery/DO₂ (e.g. haemoglobin level, pump flow) before, and at the time of CPB. First, the medullary zone of the kidneys has for some time been assumed to be prone to hypoperfusion

because it is an area of high metabolic rate ‘on the verge of hypoxia’. Recent research on rats has suggested that this may be more specifically the juxta medullary zone.² Reduced oxygen delivery (calculated by the oxygen delivery equation) therefore gives a physiological basis for potential renal damage. Low intraoperative haematocrit occurs as a result of blood loss, haemodilution, and low starting haemoglobin and directly affects oxygen delivery during cardiac surgery. Ranucci and colleagues³ demonstrated that lowered haematocrit is linked to development of renal dysfunction. Blood transfusion data have not been declared, but red cell transfusion has also been identified as a risk factor for adverse outcome after cardiac surgery.⁴ In addition, Karkouti and colleagues⁵ identified that a preoperative haemoglobin level below 9 g dl⁻¹ is an independent risk factor for adverse outcome after cardiac surgery. I would also like to comment that although pump flow is stated at 2.2 litre min⁻¹ kg⁻¹ in the paper, in our department it is customary to reduce this by approximately 5% per °C of cooling according to mixed venous oxygen estimation and I wonder if this was the case in this study. Ranucci has commented that increasing pump flow when haematocrit falls can counteract the effect of reduced O₂ carrying capacity, so if pump flow was not reduced I wonder if this has had a beneficial effect. It certainly would be of interest in the future to have a similarly conducted study which includes a group with anaemia to permit comparison of outcome, as anaemic patients do present for cardiac surgery relatively commonly.

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Editor—We thank Dr Morrice for addressing several issues that may explain the maintenance of renal function after CPB in our patient population. At our institution, pump flow is routinely maintained at a fixed level during mild hypothermia. Further, haematocrit levels during CPB are targeted at 20–25%; if necessary because of haemodilution, blood donation is instituted at the start of bypass. Because of our procedure, we agree with Dr Morrice that the fixed pump flow during CPB, providing increased DO₂, may offer an explanation for the protection of renal function. However, our study was not designed to establish the effects of oxygen delivery on perioperative renal function and the data requested are unavailable. Indeed, it would be of interest in the future to have a similar study conducted to evaluate the influence of oxygen delivery on renal function during CPB. In view of the ease of recruiting patients and the duration of the trial, it would be worthwhile to explore whether such a study would benefit from the use of sensitive markers for glomerular and tubular function.⁶