to transiently delay return of synchronized atrioventricular conduction and increase the likelihood of ventricular fibrillation immediately after aortic unclamping. No long-term effects were noted, and these changes were avoided by delivering the cardioplegic solution with the *target* calcium concentrations in the original formulation. This is achieved by supplementing the priming fluid in the extracorporeal circuit with CaCl_2 .

Initially, we added CaCl_2 , 200 mg/L, to any acalcemic priming fluid (Plasma-Lyte-A, Isolyte, or Normosol) to furnish the same CaCl_2 in the crystalloid component as present in Ringer's lactate solution. This addition, in general, avoided systemic hypocalcemia because (1) the 2 L prime is mixed with 5 L of normocalcemic blood (the patient's blood volume) and (2) body calcium stores are mobilized promptly. Occasionally, however, borderline calcium levels (0.9 mmol/L) were observed in smaller patients (<50 kg) with less blood volume.

Our recent analysis suggests that the 2000 ml acalcemic prime used in adult patients, which contains 1800 ml acalcemic crystalloid (i.e., Plasma-Lyte) and 200 ml 25% albumin, should be supplemented with 300 mg CaCl_2 per liter of prime: (1) Plasma-Lyte solution contains gluconate, which binds ionized calcium; (2) there is further binding of calcium by albumin. The increase in CaCl_2 from 200 mg/L to 300 mg/L compensates for this and ensures a normal ionized calcium in the asanguineous component. We tested also asanguineous priming fluids containing Ringer's lactate and albumin and found that normal calcium concentrations are achieved by adding 100 mg CaCl_2 per liter of prime. The calcium binding by albumin caused the mild lowering of calcium levels.

Fig. 1, A and B shows in vitro data generated with simulation of the standard pump prime (9:1, reflecting 1800 ml Plasma-Lyte and 200 ml 25% albumin) and with Ringer's lactate and albumin, as well as when the crystalloid component is reduced (ratios < 9:1). The potential for iatrogenic systemic hypocalcemia and subsequent inadvertent excessive lowering of ionized calcium concentrations in the cardioplegic solution would, of course, be increased if (1) a standard prime were used in patients with a smaller blood volume (i.e., <50 kg patient) or in children, in whom proportionately more albumin is used. The minor modification of adding CaCl_2 to the priming fluid will leave nothing to chance and will ensure delivery of the target concentrations in the induction, maintenance, and reperfusion solutions. Periodic measurement of ionized calcium in blood from the extracorporeal circuit will allow detection and correction of inadvertent hypocalcemia.

We hope this information is helpful and that the illustration provides useful information where our formulations are used in priming fluids with higher proportions of albumin to acalcemic fluids (e.g., Plasma-Lyte, Isolyte, and Normosol).

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Origins of warm heart surgery

To the Editor:

During the past few years, our group has been interested in the experimental and clinical use of continuous normothermic blood cardioplegia. As a coauthor of the "original" report on warm heart surgery, I am eager to inform you of a remarkable paper from Lillehei's group in 1957 by Gott and coworkers.¹ In this report, Gott and coworkers first described the conceptual framework for what is now called "warm heart surgery." Their pioneering experimental study combined normothermic blood perfusion with potassium-induced arrest (with Melrose's solution of potassium citrate).

Even during this early period of cardiac surgery, Lillehei's group was interested in the clinical use of this technique for coronary artery surgery and valve surgery. It is truly unfortunate that Melrose's solution fell into disfavor (largely because of the high concentration of the potassium citrate) and that the cardiac surgical community moved away from this early technique of continuous normothermic blood cardioplegia.

We are once again reminded of the enormous contributions of Lillehei's group to the development of cardiac surgery. I am sorry that we were not previously aware of this landmark paper. We should therefore now give appropriate credit to these early workers as the originators of the technique. It should make us humble and remind us to avoid extravagant claims of priority. Many times in the history of medicine, researchers have "reinvented the wheel." It appears that we have done this once again.

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Vasoplegic syndrome: A new dilemma

To the Editor:

The postperfusion syndrome is a well-known entity and its consequences are fully documented. Among them are variable degrees of lung and kidney dysfunction, hemorrhagic diathesis, increased tendency to infection, swelling, leukocytosis, fever, vasoconstriction, and hemolysis.¹

Recently, a new form of postperfusion syndrome has been

Fig. 1. Relation of norepinephrine (NOR) administration and systemic vascular resistance index (SVRI) in a patient with a vasoplegic syndrome in the immediate postoperative (PO) period.

worrying us. We have observed a vasoplegic syndrome² in the early postoperative period with severe hypotension and normal or elevated cardiac output, decreased filling pressures, and low systemic vascular resistance. Physical examination reveals normal capillary filling at the extremities, normal oxygen saturation, but oliguria and hypotension.

Our comments are based on six cases in which thermodilution monitoring was done in the immediate postoperative period. The hemodynamic data showed low wedge and right atrial pressures (wedge pressure from 4 to 11 mm Hg), mean arterial pressure in the range of 40 to 65 mm Hg, cardiac index from 2.97 to 3.82 L · min⁻¹ · m⁻², and systemic vascular resistance index (SVRI) from 700 to 1200 dyne · sec · cm⁻⁵ · m² with heart rates in the range 120 to 130 beats/min. Restoration of volume was not sufficient to restore normal hemodynamic parameters. These patients needed a high dosage of vasoconstrictor drugs (norepinephrine) for hours or even days until symptoms were completely reversed. A chart relating sequential values of norepinephrine and SVRI in one of our patients demonstrates an initial response of SVRI to norepinephrine and then the need for further increases of norepinephrine until SVRI stabilizes and increases (Fig. 1). In this patient norepinephrine was withdrawn after a little more than 24 hours; in some other patients norepinephrine was continued for more than 72 hours. Interestingly, even high dosages of norepinephrine in these patients did not produce the classic situation of cool extremities and faint peripheral pulses. The morbidity and mortality in this patient group is high, however.

No isolated or predictive risks could be incriminated in the development of this syndrome. Several aspects may be related to the emergence of this complication. Plasticizers—contaminants of tubes and oxygenators—affect cardiovascular dynamics,³ and leukocyte activation with cytokines and release of tumor necrosis factor,⁴ as occur in endotoxic shock, could produce similar clinical features.

Curiously, this syndrome was observed in our service after blood hypothermic cardioplegia had been introduced as a method of myocardial protection.

Despite its infrequent appearance (the prevalence in our series is 1/120 cases), the deleterious effects of the vasoplegic syndrome, as well as its mortality, have been worrisome. Efforts

should be directed to determine the cause of this syndrome and to prevent its onset. Once it has developed, treatment with intravenous vasoconstrictors is usually necessary.

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Atrioventricular septal defect with native subaortic stenosis: Correction by extended valvular detachment

To the Editor:

When associated with atrioventricular septal defect (AVSD), treatment of native subaortic stenosis remains a surgical challenge. Four different techniques¹ can be used for surgical cor-