## References

1. Ereth MH, Oliver WC Jr, Beynen FMK, Mullary CJ, Orszulak TA, Santrach PJ, Ilstrup DM, Weaver AL, Williamson KR: Autologous platelet-rich plasma does not reduce transfusion of homologous blood products in patients undergoing repeat valvular surgery. ANESTHESIOLOGY 79:540–547, 1993

2. Owens M, Holme S, Heaton A, Sawyer S, Cardinali S: Post-transfusion recovery of function of 5-day stored platelet concentrates. Br J Haematol 80:539–544, 1992

3. Mohr R, Goor DA, Yellin A, Moshkovitz Y, Shinfeld A, Martinowitz LL: Fresh blood units contain large potent platelet that improve hemostasis after open heart operations. Ann Thorac Surg 53:650–654, 1992

4. Jones JW, McCoy TA, Rawitscher RE, Lindsley DA: Effects of intraoperative plasmapheresis on blood loss in cardiac surgery. Ann Thorac Surg 49:585–590, 1990

5. Giordano GF, Rivers SI, Chung GKT, Mammana RB, Marco JD, Raczkowski AR, Sabbagh A, Sanderson RG, Strug BS: Autologous platelet-rich plasma in cardiac surgery: Effect on intraoperative and postoperative transfusion requirements. Ann Thorac Surg 46:416–419, 1988

6. Boldt J, von Bormann B, Kling D, Jacobi M, Moosdorf R, Hempelmann G: Preoperative plasmapheresis in patients undergoing cardiac surgery procedures. ANESTHESIOLOGY 72:282–288, 1990

7. Davies GG, Wells DG, Mabee TM, Sadler R, Melling NJ: Platelet-leukocyte plasmapheresis attenuates the deleterious effects of cardiopulmonary bypass. Ann Thorac Surg 53:274–277, 1992

*(Accepted for publication December 3, 1993.)*

Anesthesiology
80:716–717, 1994


*In Reply:*—We agree with Davies *et al.* and Stover and Siegel that the clinical efficacy and appropriate application of autologous platelet-rich plasma in cardiac surgery is yet to be defined. We also agree that there are important differences in the techniques of platelet extraction and methodologies presented in the works by ourselves and others. We do, however, emphasize the importance of the introduction of blinding methods to the assessment of this particular blood conservation technique.

We acknowledge the concern about platelet yield and the limitations of the Haemonetics Plasma Saver (Braintree, MA). Yet, to our knowledge, the only two prospective, randomized, and blinded trials published to date have used the Haemonetics procedure in primary and repeat cardiac surgical cases and have shown no reduction in bleeding or transfusion requirements.[1,2] The variability of transfusion practice in cardiac surgery is well recognized and documented.[3] We believe that the blinding technique we introduced has enhanced experimental design in trying to limit the observational bias and subjective nature of transfusion practices.

With regard to the technical comments and calculations by Davies *et al.*, we did, in fact, alter the Haemonetics protocol to extend sampling 40 ml into the red cell layer. Our median platelet yield, as reported in our article, was 2.7 units, with a "unit" defined as $5 \times 10^{10}$ platelets according to the American Association of Blood Banks standards for platelets from whole blood.[4] Accordingly, our median yield was $1.5 \times 10^{11}$, or 42% of the $3.5 \times 10^{11}$ mean yield Davies *et al.* reported in their first article[5] to be efficacious and 50% of the American Association of Blood Banks standards for apheresis platelets. Apheresis platelets are stored for as many as 5 days, and the hypothesis that small amounts of fresh platelets might be as efficacious as twice their number of stored platelets is not unreasonable, based on the work of others.[6]

Techniques that provide higher yields of platelet-rich plasma may result in a more effective product. There may, in fact, be a critical level or volume of platelet-rich plasma that must be reached before a difference in transfusion requirements is demonstrated. However, we are unaware of any blinded trials of high-yield platelet-rich plasma procedures. We believe that the routine use of platelet-rich plasma in cardiac surgery merits further consideration. Whether it is truly clinically effecacious and cost effective in cardiac surgical procedures is not known.

In summary, as we stated in our Discussion, "We cannot conclude that other groups of patients in other clinical situations may not benefit" from the use of platelet-rich plasma. We acknowledge that high-yield platelet-rich plasma techniques may be effective, but we believe that blinded methods must be used to evaluate further this procedure before it can be declared clinically efficacious for routine use in cardiac surgery. We appreciate the comments by the above groups and look forward to reviewing or conducting a prospective, randomized, and blinded trial of high-yield platelet-rich plasma.

**Mark H. Ereth, M.D.**
**William C. Oliver, Jr., M.D.**
**Froukje M. K. Beynen, M.D.**
**Charles J. Mullany, M.B.B.S.**
**Thomas A. Orszulak, M.D.**
**Paula J. Santrach, M.D.**
**Duane M. Ilstrup, M.S.**
**Amy L. Weaver, M.S.**
**Kenneth R. Williamson, M.D.**
Departments of Anesthesiology, Surgery, and Laboratory Medicine and Pathology
Mayo Clinic
Rochester, Minnesota 55905

## References

1. Ereth MH, Oliver WC Jr, Beynen FMK, Mullany CJ, Orszulak TA, Santrach PJ, Ilstrup DM, Weaver AL, Williamson KR: Autologous platelet-rich plasma does not reduce transfusion of homologous blood products in patients undergoing repeat valvular surgery. ANESTHESIOLOGY 79:540–547, 1993

2. Tobe CE, Vocelka C, Sepulvada R, Gillis B, Nessly M, Verrier ED, Hofer BO: Infusion of autologous platelet rich plasma does not

reduce blood loss and product use after coronary artery bypass. J Thorac Cardiovasc Surg 105:1007–1014, 1993

3. Goodnough LT, Johnston MFM, Toy PTCY: The variability of transfusion practice in coronary artery bypass surgery. JAMA 265:86–90, 1991

4. Widman FE: Standards for Blood Banks and Transfusion Services. 15th edition. Bethesda, American Association of Blood Banks, 1993

5. Davies GG, Wells DG, Mabee TM, Sadler R, Melling NJ: Platelet-leukocyte plasmapheresis attenuates the deleterious effects of cardiopulmonary bypass. Ann Thorac Surg 53:274–277, 1992

6. Mohr R, Martinowitz U, Lavee J, Amroch D, Ramot B, Goor DA: The hemostatic effect of transfusing fresh whole blood versus platelet concentrates after cardiac operations. J Thorac Cardiovasc Surg 96: 530–534, 1988

*(Accepted for publication December 3, 1993.)*



Anesthesiology
80:717–718, 1994

J. B. Lippincott Company, Philadelphia

# Pacemaker Interactions with Transcutaneous Cardiac Pacing

*To the Editor:*—Kemnitz and Peters[1] describe interactions of pacemakers with transcutaneous cardiac pacing. We would like to offer alternative explanations to some of their conclusions.

The first case reported[1] involved a patient with sick sinus syndrome who had a permanent VVI pacemaker. Sinus rhythm prevailed for 20 min after induction of anesthesia, at which point the heart rate decreased progressively below the programmed rate to initiate VVI pacing. As the authors point out, this pacing mode was associated with a decrease in blood pressure due to loss of the atrial contribution to ventricular filling. To improve hemodynamics, the anesthesiologist intended to increase the heart rate using a previously applied transcutaneous pacemaker, but before this could be accomplished he noticed that low currents delivered by the transcutaneous pacemaker inhibited the permanent pacemaker and restored sinus rhythm with improved hemodynamics.

We question the very decision to use the transcutaneous cardiac pacemaker because it is a temporary VVI pacemaker and would offer no advantage to this patient with congestive heart failure, who was dependent on atrioventricular synchrony and the atrial contribution. Fortunately, incremental increases in the current were therapeutic to restore sinus rhythm by inadvertently inhibiting the permanent pacemaker. As discussed by the authors, other measures to inhibit permanent pacemakers include applied stimuli *via* a cutaneously applied nerve stimulator or perhaps by another temporary pacemaker; careful titration of isoproterenol; and overdrive transesophageal atrial pacing. In addition, temporary transvenous atrial pacing may be used in the acute setting while the option of a permanent dual chamber pacemaker is considered with the patient's cardiologist.

The second patient described by Kemnitz and Peters[1] was pacemaker-dependent after cardiac surgery. Pacing was achieved by a temporary VVI pacemaker at a rate of 90 beats/min. To test the feasibility of transcutaneous cardiac pacing, the noninvasive pacemaker was set at 95 beats/min with the threshold current progressively increased to 40 mA. The authors state that this maneuver failed to produce ventricular pacing and inhibited the temporary pacing, resulting in temporary asystole.

Here again, intentional delivery of external current transcutaneously should not be interpreted as an interference but as an expected interaction with the temporary VVI pacemaker. The failure for pacing capture with the transcutaneous pacemaker may be due to the relatively low current output used, as acknowledged by the authors, especially in a patient recovering from cardiac surgery, because of the presence of air or fluid in the chest, which may mitigate pacing

**Table 1. Rate Settings of the Noninvasive Transcutaneous Pacemaker**

| Pacing Rate Desired (pulses/min) | Actual Heart Rate (beats/min) |
|---|---|
| 60 | 55 |
| 70 | 63 |
| 80 | 71 |
| 90 | 78 |
| 100 | 86 |
| 110 | 93 |
| 120 | 100 |
| 130 | 107 |
| 140 | 114 |
| 150 | 120 |
| 160 | 126 |

Comparison of actual paced heart rate and displayed pacing rate. This assumes that every paced beat is sensed.

$$\text{Actual heart rate} = \frac{60{,}000\ (\text{ms})}{\text{pacing rate (ms)} + 100\ \text{ms}}$$

Example: desired pacing rate = 60 (pulses/min)

$$\text{Actual rate} = \frac{60{,}000\ \text{ms}}{1000\ \text{ms} + 100\ \text{ms}} = 55\ \text{beats/min}$$

Data provided as a technical communication by Zoll Medical Company (Woburn, MA).